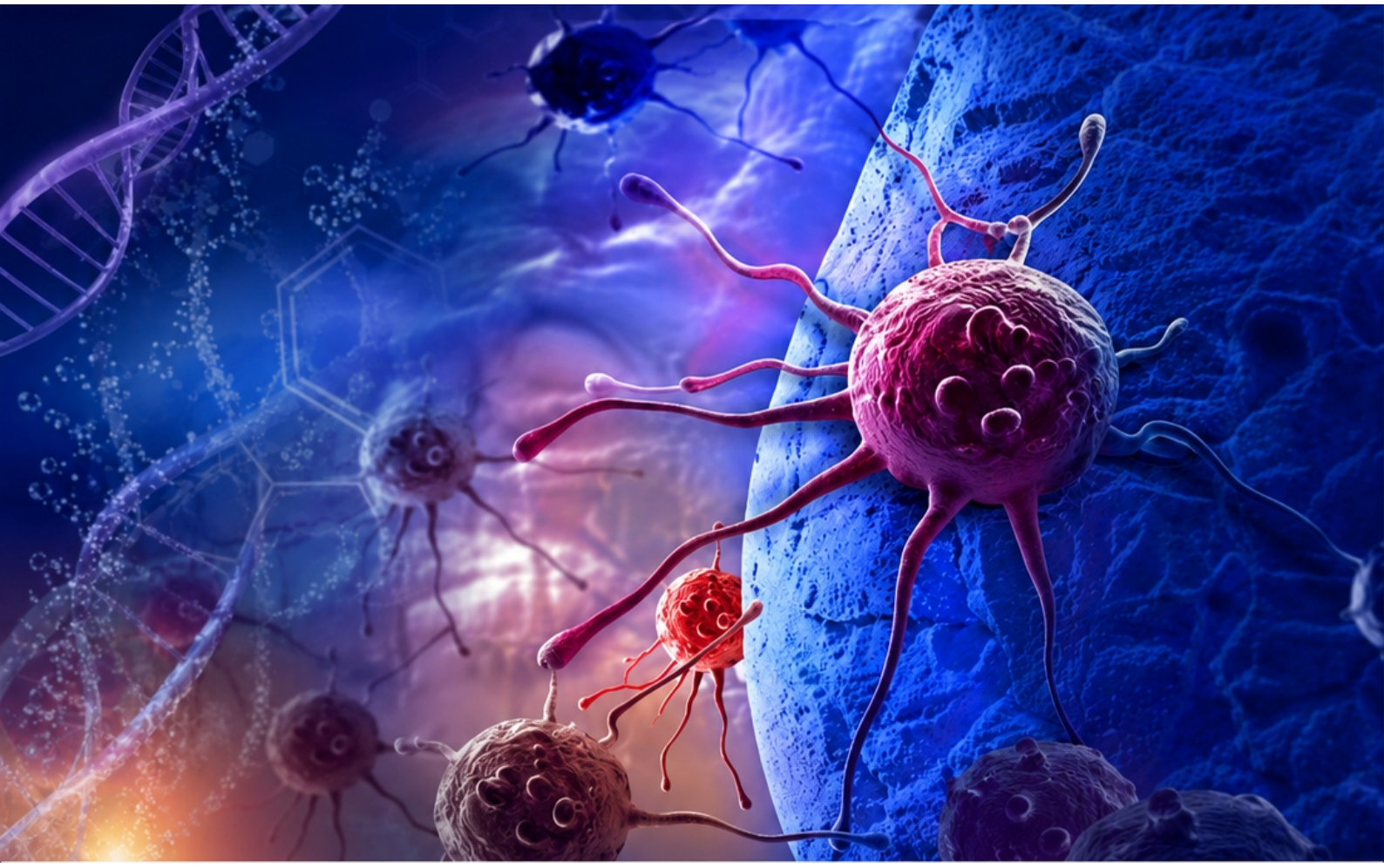
BioGenesis

THE JOURNAL OF BIOLOGY AND MEDICINE



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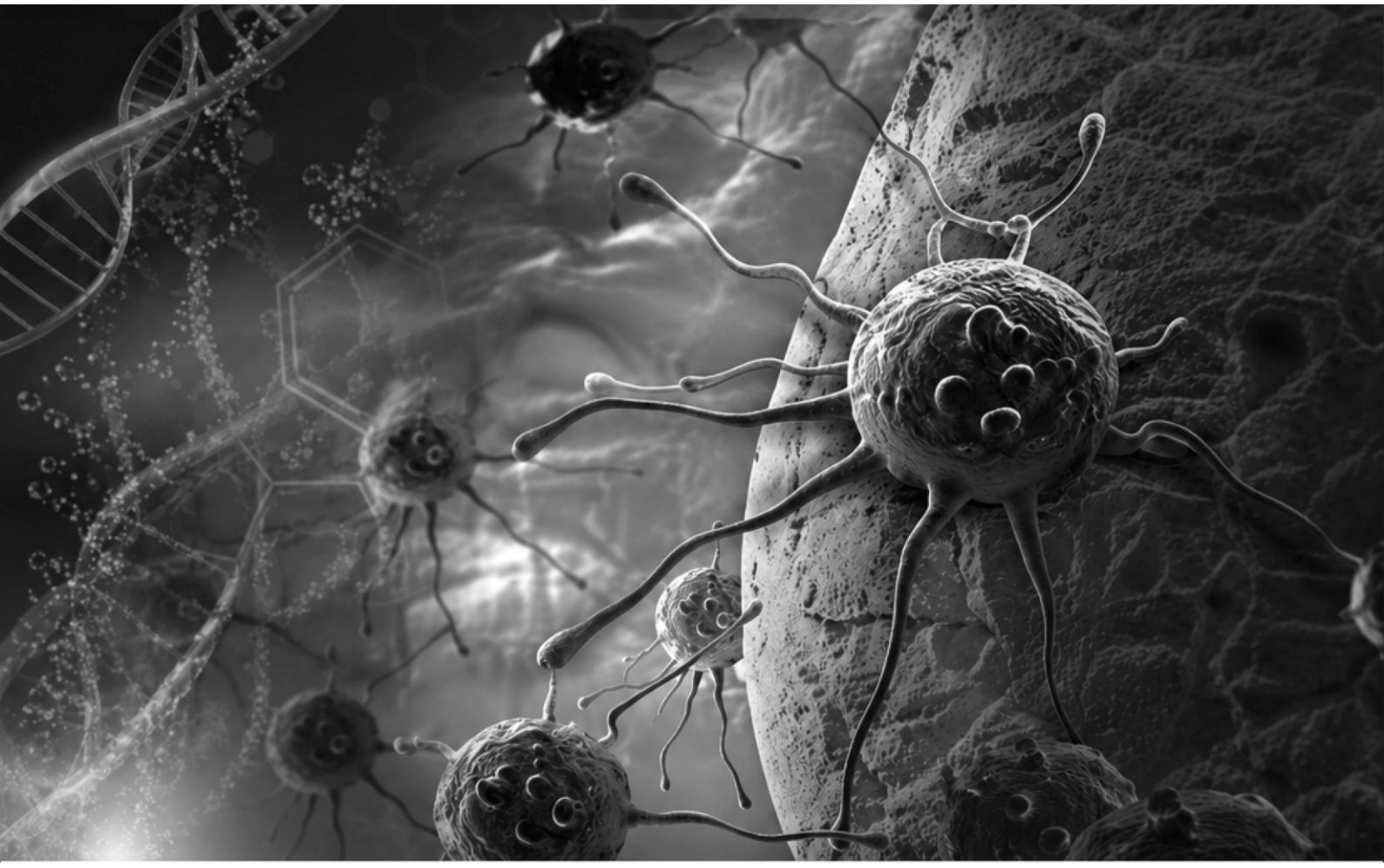
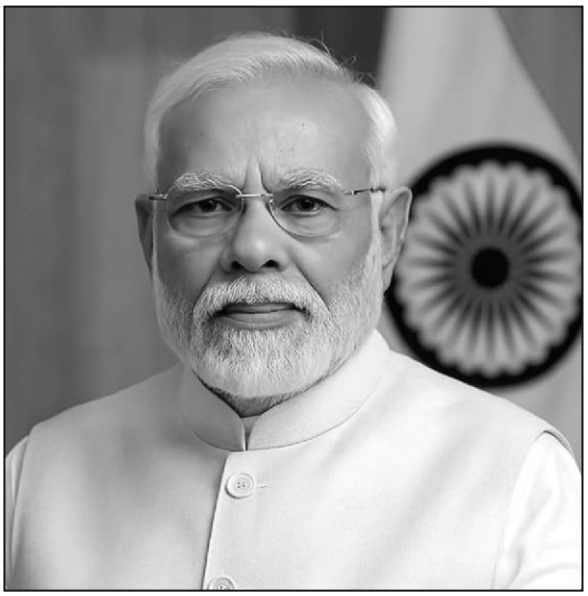
7th GLOBAL CANCER SUMMIT™ 2026

International Collaborative Conference

23rd August, 2026

Banaras Hindu University (BHU), Swatantrata Bhawan Auditorium,
Varanasi, Uttar Pradesh, India



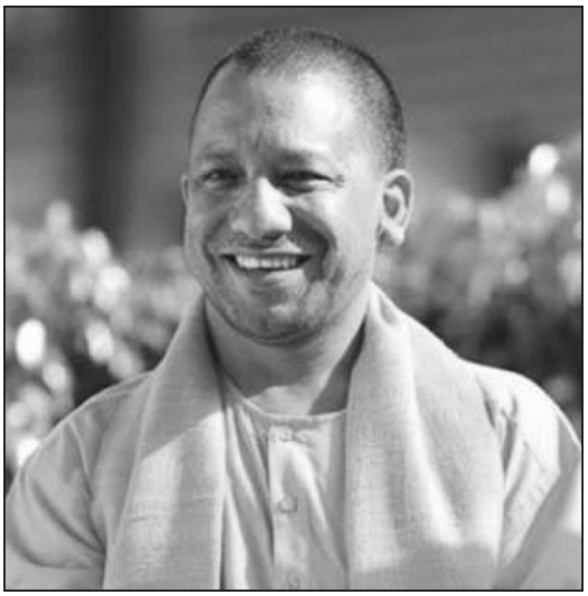


“Inspired by the National Vision for Cancer Care

India has made remarkable progress in strengthening healthcare through a strong emphasis on preventive health, early diagnosis, affordable treatment, innovation, and research. The national commitment to expanding cancer screening, promoting indigenous technologies, and ensuring equitable access to quality healthcare continues to inspire the medical and scientific community.”

- Hon'ble, Shri. Narendra Modi

Prime Minister of India



“The Vision of Hon'ble Chief Minister Shri Yogi Adityanath

The Government of Uttar Pradesh envisions a healthier and developed state under the 'Viksit Uttar Pradesh @2047' mission. With a strong emphasis on cancer prevention, early detection, timely diagnosis, affordable treatment, research, and equitable access to quality healthcare, the State is expanding cancer treatment facilities across districts while strengthening specialist services and modern medical infrastructure. This vision inspires all stakeholders to work together towards substantially reducing the burden of cancer and improving the quality of life for every citizen of Uttar Pradesh”

- Hon'ble, Shri. Yogi Adityanath

Chief Minister of Uttar Pradesh

“Vision to transform Uttar Pradesh into India's leading healthcare state by ensuring equitable, affordable, accessible, and technology-driven healthcare for every citizen through world-class medical infrastructure, skilled healthcare professionals, innovation, and compassionate patient care.”

- Hon'ble, Shri. Brajesh Pathak

Deputy Chief Minister
Uttar Pradesh

"Banaras Hindu University has long stood as a beacon of excellence in higher education, medical sciences, research, and innovation. Under the visionary leadership of its Vice-Chancellor, the University continues to strengthen academic standards, promote cutting-edge biomedical research, and nurture the next generation of healthcare professionals.

The vision is to create an ecosystem where young minds are empowered through quality education, ethical medical practice, scientific inquiry, and interdisciplinary collaboration. By encouraging innovation, compassion, and leadership, BHU aims to prepare future doctors, researchers, nurses, and public health professionals who will address emerging healthcare challenges and contribute to the well-being of society."

Prof. Ajit Kumar Chaturvedi

Vice-Chancellor of Banaras Hindu University (BHU), Varanasi.

Vision on Healthcare and Medical Education in the State of Uttar Pradesh

Vision Statement

"To transform Uttar Pradesh into a global leader in equitable, affordable, technology-driven, and research-oriented healthcare by ensuring universal access to quality medical services and creating a world-class ecosystem for medical education, innovation, and scientific excellence."

Strategic Vision

Uttar Pradesh, the most populous state in India, has the potential to become a national and international model for healthcare delivery and medical education. The state's future lies in building an integrated healthcare system that combines preventive, promotive, curative, rehabilitative, and palliative care with excellence in medical education and biomedical research.

The vision is to ensure that every citizen—whether living in a metropolitan city or a remote village—has access to affordable, high-quality healthcare supported by skilled healthcare professionals, modern hospitals, digital technologies, and advanced research institutions.

Dr. Amit Kumar Ghosh, IAS
Additional Chief Secretary, Medical Health, Family Welfare &
Medical Education Department, Government of UP

Cancer: A Global Healthcare Challenge

"Cancer remains one of the most complex and formidable challenges in modern health sciences. Despite remarkable advances in understanding its biology, prevention, diagnosis, and treatment, it continues to be a leading cause of illness and mortality worldwide, affecting millions of individuals and families each year.

Over the past few decades, significant progress has been achieved through innovations in molecular biology, genomics, precision medicine, immunotherapy, targeted therapies, artificial intelligence, robotic surgery, advanced imaging, and personalized patient care. These scientific and technological breakthroughs have substantially improved early detection, treatment outcomes, survival rates, and quality of life for many patients.

However, the global fight against cancer is far from complete. Many forms of cancer remain difficult to diagnose at an early stage, while disparities in healthcare infrastructure, access to quality treatment, affordability, and public awareness continue to pose major challenges, particularly in low- and middle-income countries.

Achieving a cancer-free world requires sustained investment in research, innovation, multidisciplinary collaboration, public health initiatives, and equitable access to modern healthcare. Governments, healthcare professionals, researchers, industry partners, and civil society must work together to accelerate scientific discoveries, promote preventive strategies, strengthen screening programmes, and ensure that every patient receives timely, affordable, and compassionate care.

The vision of a world where cancer is no longer a life-threatening disease is an ambitious but achievable goal. Through continued scientific excellence, technological innovation, international collaboration, and unwavering commitment to patient-centred care, the global healthcare community can move closer to transforming cancer from a major public health burden into a preventable, manageable, and ultimately curable disease for future generations."

Dr. G. K. Rath, Head

National Cancer Institute (India)

Science, Innovation, and the Global Fight Against Cancer

"Science represents humanity's collective pursuit of knowledge and discovery. Throughout history, scientists have dedicated their lives to understanding diseases, uncovering their causes, and developing innovative solutions that improve health and save lives. Every major medical breakthrough is the result of years of rigorous research, collaboration, and an unwavering commitment to humanity.

Cancer remains one of the greatest scientific and public health challenges of the twenty-first century. Thousands of scientists, clinicians, and healthcare professionals across the world are working tirelessly in research laboratories, universities, hospitals, and biotechnology industries to understand the complex biology of cancer and to develop safer, more effective methods for its prevention, early detection, diagnosis, and treatment.

Among women, breast and cervical cancers continue to be major contributors to the global cancer burden. These diseases account for a significant proportion of cancer cases and deaths, particularly in low- and middle-income countries where access to screening, vaccination, early diagnosis, and quality treatment remains limited. While HIV/AIDS is not a cancer, it increases the risk of certain cancers by weakening the immune system and therefore requires integrated public health strategies.

International organizations, including the United Nations and its health partners, are working with governments and global institutions to reduce the burden of cancer through awareness, prevention, capacity building, and improved access to healthcare services. Their efforts aim to strengthen health systems, promote equity in cancer care, and protect vulnerable populations around the world.

India has made significant progress in advancing cancer care through precision medicine, molecular diagnostics, immunotherapy, digital health technologies, expanded cancer screening programmes, and the establishment of comprehensive cancer centres. National initiatives are increasingly focused on early detection, affordable treatment, public awareness, and strengthening research and innovation to reduce the burden of cancer across the country.

The journey towards a world with fewer cancer-related deaths requires sustained scientific research, international collaboration, strong public health policies, and equitable access to quality healthcare. By bringing together researchers, clinicians, policymakers, industry leaders, and patient advocates, we can accelerate the translation of scientific discoveries into practical solutions that improve survival, enhance quality of life, and reduce the global burden of cancer.

The vision of a future where preventable cancers are eliminated, treatable cancers are effectively managed, and every individual has access to timely, compassionate, and high-quality care remains a shared global responsibility. Through science, innovation, and collective commitment, we can move steadily towards a healthier and more hopeful future for all."

- Dr. Ajit Kumar Saxena, Professor & Head
Department of Pathology/Lab Medicine,
AIIMS, Patna.
Scientific Advisor, BioGenesis Health Cluster

It is with immense pleasure and a profound sense of responsibility that I welcome you to the *7th Global Cancer Summit 2026, organized by **Biogenesis Health Cluster*, a not-for-profit scientific organization dedicated to advancing biomedical research, innovation, and public health. The Summit brings together distinguished scientists, clinicians, oncologists, surgeons, researchers, academicians, policymakers, healthcare professionals, industry leaders, students, and patient advocates from India and around the world to share knowledge and strengthen global collaboration against one of humanity's greatest health challenges.

Cancer remains one of the leading causes of death worldwide. Every year, millions of people are diagnosed with various forms of cancer, and millions of families experience its physical, emotional, and economic consequences. Although significant progress has been achieved in diagnosis, surgery, radiation therapy, chemotherapy, immunotherapy, precision medicine, molecular diagnostics, and targeted therapies, cancer continues to place an enormous burden on individuals, healthcare systems, and national economies.

Cancer is not a single disease but a complex group of disorders characterized by the uncontrolled growth and spread of abnormal cells. Under normal conditions, cells grow, divide, repair themselves, and die in an orderly manner.

Cancer develops when genetic mutations disrupt these natural processes, allowing abnormal cells to multiply uncontrollably, invade surrounding tissues, and spread to distant organs through the bloodstream or lymphatic system. This process, known as metastasis, remains the principal cause of cancer-related mortality.

Today, more than two hundred distinct forms of cancer have been identified, affecting nearly every organ of the human body. The causes of cancer are multifactorial and include inherited genetic factors, tobacco use, alcohol consumption, unhealthy diets, obesity, environmental pollution, occupational exposures, chronic infections, ultraviolet radiation, lifestyle changes, and population ageing. Many cancers are preventable, while others can be detected early through screening programmes and treated successfully when diagnosed at an early stage.

The global scientific community has entered an exciting era of cancer research. Revolutionary advances in genomics, artificial intelligence, molecular biology, bioinformatics, precision oncology, liquid biopsy, robotic surgery, nanotechnology, immunotherapy, CAR-T cell therapy, cancer vaccines, and digital health are transforming the way cancer is detected and treated. These innovations are making medicine increasingly personalized, enabling clinicians to select therapies tailored to the molecular characteristics of individual patients while improving outcomes and quality of life.

Despite these remarkable advances, major challenges remain. Large disparities persist in access to early diagnosis, specialized treatment, essential medicines, advanced technologies, palliative care, and survivorship services, particularly in low- and middle-income countries. Closing these gaps requires stronger healthcare systems, sustained investment in scientific research, international collaboration, and equitable access to innovation.

The *7th Global Cancer Summit 2026* has been designed as a multidisciplinary platform where experts from medicine, life sciences, engineering, public health, biotechnology, pharmaceuticals, digital health, policy, and industry can exchange ideas and translate

scientific discoveries into practical solutions. Our objective is to encourage dialogue across disciplines, stimulate collaborative research, promote innovation, and accelerate the implementation of evidence-based cancer care.

The Summit will highlight emerging trends in cancer biology, prevention, screening, molecular diagnostics, imaging technologies, surgical oncology, radiation oncology, medical oncology, pathology, translational research, immunotherapy, precision medicine, rehabilitation, survivorship, palliative care, digital health technologies, and artificial intelligence. Special emphasis will be placed on strengthening research capacity, promoting young investigators, and encouraging innovation among students and early-career scientists.

One of the greatest strengths of this Summit is its commitment to nurturing the next generation of healthcare professionals and researchers. The future of cancer care depends upon talented young minds equipped with scientific knowledge, ethical values, compassion, and innovative thinking. By providing opportunities for students and young investigators to interact with world-renowned experts, we hope to inspire future leaders who will transform cancer care over the coming decades.

Scientific progress alone is not sufficient to eliminate the burden of cancer. Public awareness, healthy lifestyles, tobacco control, vaccination programmes, environmental protection, regular screening, equitable healthcare delivery, patient-centred care, and strong public policy are equally important. Governments, academic institutions, hospitals, industry, professional societies, non-governmental organizations, and civil society must work together to create sustainable solutions that improve health outcomes for all.

The publication of this *International Abstract Book* reflects the collective intellectual contributions of researchers, clinicians, academicians, and students from diverse institutions and countries.

Each abstract represents a commitment to scientific excellence and a determination to improve prevention, diagnosis, treatment, and survivorship through research and innovation. We hope this volume will serve as a valuable scientific resource for universities, research institutes, medical colleges, hospitals, libraries, policymakers, and healthcare professionals worldwide.

We express our sincere gratitude to Banaras Hindu University for hosting this prestigious scientific gathering and to all distinguished speakers, authors, reviewers, organizing committee members, scientific advisors, sponsors, exhibitors, volunteers, and delegates whose dedication has made this Summit possible. We also acknowledge the invaluable contributions of national and international collaborators who continue to strengthen global scientific partnerships.

As we gather in the historic city of Varanasi—one of the world's oldest centres of learning—we reaffirm our collective commitment to advancing science for the benefit of humanity. Let this Summit strengthen international cooperation, inspire scientific discovery, encourage innovation, and translate research into better patient care.

Together, through knowledge, compassion, innovation, and global collaboration, we can move steadily toward a future in which cancer is detected earlier, treated more effectively, prevented whenever possible, and ultimately becomes a disease with a greatly reduced impact on humanity. While eliminating cancer entirely remains a long-term scientific challenge, our shared efforts today will bring us closer to a world where fewer lives are lost, more patients survive, and every individual has access to high-quality cancer care.

On behalf of the Organizing Committee, I extend my heartfelt welcome to every participant and thank you for contributing to this global movement against cancer.

Dr. V. P. Rao
7th Global Cancer Summit 2026
BioGenesis Health Cluster.
Hyderabad, India.

About Conference

About the 7th Global Cancer Summit 2026

The 7th Global Cancer Summit 2026 (GCS-2026), themed "Living with Cancer in the 21st Century", with a special focus on the global burden of cancer, will be held on 23 August 2026 at the Swatantrata Bhawan Auditorium, Banaras Hindu University (BHU), Varanasi, Uttar Pradesh, India.

The Summit is being jointly organized by BioGenesis Health Cluster, Global Cancer Foundation, and Banaras Hindu University. It will bring together leading oncologists, clinicians, scientists, researchers, public health experts, policymakers, industry leaders, and healthcare professionals to discuss the latest advances in cancer prevention, diagnosis, treatment, translational research, and public health.

Objectives of the Summit

The conference aims to:

Promote scientific collaboration between Indian and international cancer researchers.

Showcase the latest innovations in cancer prevention, early detection, diagnosis, treatment, and survivorship.

Encourage translational research to accelerate the adoption of laboratory discoveries into clinical practice.

Strengthen partnerships between academia, healthcare institutions, industry, and government organizations.

Facilitate knowledge exchange on policies, technologies, and best practices for reducing the global cancer burden.

Support young researchers and clinicians by providing opportunities for scientific presentations, networking, and mentorship.

Major Scientific Themes

The scientific programme will focus on:

Cancer Stem Cells

Basic and Applied Cancer Research

Anti-Cancer Drug Discovery and Development

Clinical Oncology

Precision and Personalized Cancer Medicine

Immunotherapy and Immune Checkpoint Research

Cancer Metabolism

Women's Cancers

Artificial Intelligence and Digital Technologies in Oncology

Cancer Prevention, Screening, and Early Detection

Palliative Care and Cancer Survivorship

Benefits of Attending

Participation in the 7th Global Cancer Summit will provide delegates with opportunities to:

Learn from internationally recognized experts presenting the latest scientific and clinical developments.

Exchange ideas and collaborate with leading researchers and healthcare professionals from across the world.

Develop national and international research partnerships and multicentre collaborations.

Gain exposure to emerging technologies, innovative therapies, and precision oncology.

Interact with policymakers, funding agencies, and industry representatives.

Present original research through keynote lectures, oral presentations, and poster sessions.

Explore opportunities for academic exchange, joint publications, and international cooperation.

Contribute to recommendations that can strengthen cancer control strategies in India and globally.

Edward Kennedy Memorial Award

The Summit will host the prestigious Edward Kennedy Memorial Award, recognizing outstanding contributions in:

Experimental Cancer Research

Translational Cancer Research

Clinical Cancer Research

The award honours excellence and innovation in advancing global cancer research and patient care.

Legacy of the Global Cancer Summit

Previous editions of the Global Cancer Summit, including Bengaluru (2015) and Kuala Lumpur (2017), attracted over 1,500 delegates.

Participants represented universities, research institutions, hospitals, government organizations, industry, cancer care providers, patient advocacy groups, and cancer survivors, creating a truly multidisciplinary platform for scientific exchange.

Delegate Feedback

Independent delegate evaluations from previous Global Cancer Summits demonstrated exceptional satisfaction, with 98% of participants recommending the conference to their colleagues. Delegates particularly appreciated:

High-quality scientific sessions

Distinguished international faculty

Excellent networking opportunities

Productive academic collaborations

Interactive discussions and multidisciplinary learning

Strong industry-academia-government engagement

The 7th Global Cancer Summit 2026 aims to further strengthen India's leadership in global cancer research by promoting scientific excellence, international collaboration, innovation, and policy dialogue to improve cancer prevention, treatment, and patient outcomes worldwide.

7th GLOBAL CANCER SUMMIT® – 2026

International Collaborative Conference

SPEAKERS

Clinical Proteomic and Glycoproteomic Integration Reveals LGALS3BP as a Promising Biomarker for Glioma Progression

Rashmi Rana
Senior Scientist, Department of Biotechnology and Research,
Professor, GRIPMER, Sir Ganga Ram Hospital, New Delhi, India

Background: Brain Complexity contributes to the lacking in understanding pathogenesis behind several Brain Disorders and CNS Tumor. Thus, this aspect restricts the diagnosis and treatment strategies. Several Research groups working on finding out the possible factors involve in tumor progression, metastasis and proliferation. Similarly, we have found out the galectin 3 binding protein (LGALS3BP) in different grade of gliomas in a progressing manner. Following on this, our study aimed to explore LGALS3BP expression across all glioma grades (I-IV) to identify any associations with clinicopathological factors that might influence patient survival.

Material and Method: Tissue samples from 80 glioma patients were analyzed using IHC, flow cytometry, ICC, and Western blot to assess LGALS3BP levels and expression. Clinicopathological markers (ATRX, IDH-1, p53, Ki-67) were evaluated alongside LGALS3BP, with overall survival analyzed via Kaplan-Meier curves and correlations determined using the Pearson correlation coefficient.

Results: Our study demonstrates a clear correlation between LGALS3BP expression and glioma grades (I-IV), with the highest levels observed in Grade IV (GBM) and progressively lower levels in grades III, II, and I, with minimal detection in controls. IHC, flow cytometry, WB, and ICC analysis confirmed this trend, showing a significant increase in LGALS3BP from lower to higher-grade gliomas. Survival analysis revealed that IDH-mutant Grade IV patients had longer survival than IDH-wildtype, and combinations of LGALS3BP with p53mut/ATRXmut/IDHmut were associated with improved outcomes. However, Pearson correlation analysis found no direct association between LGALS3BP and clinicopathological markers.

Conclusion: This study highlights a significantly elevated level of LGALS3BP across grades of glioma, much more pronounced in high-grade glioma. These findings suggested that the LGALS3BP is a potential diagnostic biomarker and improves their survival outcomes by solving clinical problems associated with proliferative nature, recurrence, and metastasis. This could provide us targeted approach for therapeutic interventions

Biography: Dr. Rashmi Rana, an Associate Professor at the Department of Biotechnology & Research, Sir Ganga Ram Hospital (SGRH), Delhi, and an adjunct faculty and Ph.D. coordinator at MAHE, Manipal and Course Coordinator, PG Diploma in Clinical Research and Regulation at SGRH.

Dr. Rana is a leading researcher in the fields of brain tumor (Glioma and Meningioma) diagnostics, dengue, sepsis, and chronic kidney disease (CKD). Since joining SGRH in 2017, Dr. Rana's research has focused on early diagnosis and therapeutic strategies for gliomas, a type of aggressive brain tumor. Her work has led to the identification of LGALS3BP as a promising biomarker for early glioma detection, enabling better prognosis and monitoring of tumor progression across glioma grades (I-IV).

Dr. Rana is now pursuing innovative strategies for glioma treatment, utilizing a three-dimensional approach. She aims to silence LGALS3BP to inhibit tumor growth and progression. Additionally, Dr. Rana is employing CRISPR gene-editing technology to correct mutated genes that drive glioma. Her research also includes developing specialized machinery to cross the blood-brain barrier, delivering chemotherapy drugs directly to tumor sites for improved treatment efficacy while minimizing side effects.

Alongside her glioma work, Dr. Rana is actively involved in research on dengue, sepsis, and CKD, expanding her expertise in diverse areas of biomedical science. Her ground breaking contributions have earned her numerous prestigious awards, including the ESMO, HUPO, and EMBO fellowships, the Bill & Melinda Fellowship in 2023, the HCFI Dr. K. K. Aggarwal Research Innovation Fund Award, DST Young Scientist Award, NAMS Fellow, VIHA Young Scientist Award, Part of EMBO leadership programme, NAMS task force member on vector borne diseases, Convenor in Proteomic Society of India, ASSOCHAM research excellence award and the FCR Award from Harvard. She also serves as an editor for several journals and publishing houses. She awarded S. Nundy Best Published Research Article, 2025. She has published more than 67 articles in International and National journals.

Recently, Recently She established a Multi-Omics Core Facility in SGRH, Delhi.

Currently, Dr. Rana is leading five ongoing research projects and completed three project successfully, while mentoring team of scientists, doctoral, and graduate students. Her work continues to make significant strides in the fields of Brain Tumor Research and therapeutic innovations.

SPEAKERS

The miR-127/136 Cluster as a Regulator of Tumor Behavior and Cancer Stemness in Cervical Cancer

Dr. Samatha Bhat

Assistant Professor, Department of Biotherapeutics Research, Manipal Academy of Higher Education, Manipal, India

Cervical cancer (CC) is the fourth most common cancer among women in India. Despite multiple screening and early detection strategies, disease incidence and mortality remain high, with one woman dying from cervical cancer every eight minutes. Persistent infection with high-risk human papillomavirus (HPV) is the primary etiological cause of CC; however, disease progression, metastasis, therapeutic resistance, and recurrence are driven by a subpopulation of cancer stem-like cells (CSCs). These cells exhibit enhanced self-renewal and plasticity regulated by microRNAs (miRNAs), key post-transcriptional regulators of gene expression. Despite its emerging importance, the mechanistic role of the miR-127/136 cluster in regulating cervical cancer stemness remains largely unexplored.

In this study, we investigated the functional role of the miR-127/136 cluster in cervical cancer. The miR-127/136 cluster was stably overexpressed in three cervical cancer cell lines using a lentiviral delivery system, and its overexpression was confirmed by RT–qPCR. Functional assays demonstrated that miR-127/136 overexpression significantly altered tumor behavior, as assessed by colony formation, cell proliferation, migration and invasion, apoptosis, and cell cycle progression. Western blot analysis revealed altered expressions of key stemness-associated and epithelial-mesenchymal transition (EMT)-related proteins, indicating a role for this cluster in CSC regulation. Bioinformatic target prediction and luciferase reporter assays revealed kinesin family member 11 (KIF11), a known oncogene, as a direct target of the miR-127/136 cluster, whose downregulation was validated in miR-127/136-overexpressing cells. Ongoing studies include tumorsphere-based CSC enrichment, in vivo xenotransplantation models, and whole-transcriptome profiling to define downstream signaling pathways regulated by this cluster.

Collectively, the results of this study reveal that the miR-127/136 cluster is a critical regulator of cervical cancer stemness, highlighting its potential as a novel RNAi-based therapeutic target and biomarker for early diagnosis and improved disease management.

Biography: Dr. Samatha Bhat is an Assistant Professor in the Department of Biotherapeutics Research at Manipal Academy of Higher Education (MAHE), Manipal, India. She holds a Ph.D. in Cancer Epigenetics from MAHE, Manipal, where her research focused on DNA methylation-regulated genes and their role in cervical and oral cancers. She has over 15 years of research experience spanning cancer biology, stem cell biology, and regenerative medicine.

Prior to her academic role, Dr. Bhat served as in charge of the Production Department at Stempeutics Research Pvt. Ltd., where she was instrumental in the large-scale GMP-compliant manufacturing of mesenchymal stromal cells for clinical and commercial applications. Dr. Bhat has made significant contributions to understanding epigenetic deregulation in cancer, particularly in identifying methylation-based biomarkers for early diagnosis. She has authored more than 25 research articles, three book chapters, and several conference proceedings, and has been the recipient of multiple awards, including the Dr. T.M.A. Pai Gold Medal for outstanding research, the Dr. Vidya Ravi Memorial Award as a Top Women Scientist in Stem Cell Therapy and the Invention award, awarded by Intellectual Ventures India, Bangalore. She has successfully secured extramural research grants and has filed several Indian patents. Her current research interests include investigating the role of miRNAs in cancer stemness, developing novel biotherapeutic products from stem cell secretome, and translating epigenetic biomarkers for clinical applications.

SPEAKERS

Palliative Care Management of a Advance Head and Neck Cancer Patient in a Resource Poor Area: A Case Report

Dr. Samujjhal Bharadwaj

Physician Assistant, Department of Palliative Medicine, Dr B Borooah Cancer Institute, Guwahati, Assam

Background: Managing advanced head and neck cancers in remote geographic areas presents significant clinical and socioeconomic challenges, often necessitating flexible, home-based palliative interventions.

Case Presentation: A 53-year-old male residing 67 kilometers from a tertiary treatment facility presented to the OPD of the Deptt. Of Head and Neck Oncology with a tracheostomy tube in situ. He was diagnosed with Squamous Cell Carcinoma (SCC) of the right Aryepiglottic Fold and Pyriform Sinus. Initial oncological intervention with curative intent was delayed due to concomitant tuberculosis, which required a course of Anti-Tubercular Therapy (ATT).

Management and Challenges: Following ATT clearance, the patient received Neoadjuvant Chemotherapy (Docetaxel and Cisplatin); however, the disease showed no clinical response. Although a total laryngectomy was planned, surgical fitness was repeatedly denied due to hypertension. The multidisciplinary group subsequently altered the plan to Palliative Radiotherapy, administering 30 Gy in 10 fractions.

The patient's clinical course was severely impeded by repeated treatment defaults caused by financial constraints, regional flooding, and poor road connectivity in hilly terrains. To ensure continuity of care, the patient was enrolled in a home care program.

A total of 7 home visits have been conducted till date, providing critical interventions including right neck ulcer dressings (3 instances) , T-Tube changes (2 instances) , antifibrinolytic injection deliveries (4 instances) alongside analgesic optimization.

Additionally, the caregivers of the patient were also motivated for holistic care of the patient with an informal gathering. The medical social workers successfully intervened and provided Rs. 48,400 through Government financial aid scheme.

Conclusion: This case highlights the necessity of integrating robust, home-based palliative care programs and social support systems to manage advanced cancer patients facing geographical and financial barriers.

Biography:

Experience: Oral and Maxillofacial Consultant

Nalbari Multispecialty Hospital, Nalbari, Assam, India

From 08-05-2019 to present day

Doctor

Integrated Hospital based Continuity of Care, Department of Palliative Medicine,

Dr B Borooah Cancer Institute, Guwahati, Assam, India

From 28-7-2021 to present day

Current

Skills: - USG GuidedParacentesis/ Pleurocentesis

- Managing Patients of Palliative Care in OPD, IPD, Casualty/ Emergency and Home

Care with all round services

- Breaking Bad News/ Communication/ Counselling

- Using Hospital EMR etc.

SPEAKERS

Application Of Ayurvedic Bhasmsa (ancient Indian Nanomedicine) For The Treatment Of Cancer

Dr. Sathyanarayana Badekila,M.D. Ayu

Principal and Medical Superintendent, Muniyal Institute of Ayurveda Medical Sciences, 34/C, Shivalli Industrial Area, Manipal-576104

Bhasma, an ancient Indian herbomineral formulation, is increasingly recognized as a form of traditional nanomedicine due to its unique preparation process that yields particles in the nanometer range (1–100 nm). In cancer research, Bhasmas are studied for their ability to target cancer cells, induce cell death, and act as nanocarriers.

Swarna Bhasma (Gold), one of the most researched, it contains gold nanoparticles that exhibit significant anticancer, immunomodulatory, and anti-inflammatory activities. Clinical trials have shown benefits in treating solid malignancies, including rectal and breast cancers. Abhraka Bhasma (Mica) contains mica nanoparticles and is traditionally used for tumors ("Arbuda"). Studies on breast cancer cell lines (MCF-7) demonstrate dose-dependent cytotoxicity and potential for reducing genotoxicity. Manikya Bhasma (Ruby), characterized as a nanomedicine (~70 nm), induces mitochondrial-dependent apoptosis (programmed cell death) in various cancer cell lines, including HeLa and HCT-116. Research on Yashada Bhasma (Zinc) indicates it can act as a cytostatic drug, specifically showing promise in inducing cell growth arrest in human pancreatic cancer cells. Heerak Bhasma (Nano dimond product) has demonstrated immunostimulatory and tumor-killing properties.

Anticancer activity of these Bhasmas is due to various mechanisms such as, induction of Apoptosis, cell Cycle Arrest (like Yashada Bhasma), immunomodulation and also nano carrier potential. Despite promising results, the use of Bhasma as a primary cancer treatment faces several hurdles such as lack of standardization, safety concerns, need of controlled clinical trials.

Key words: Bhasma, nanomedicine, cancer, Ayurveda

Biography:

- Lecturer in the subjects - Rasashastra and Bhaishajya Kalpana for both undergraduate and post graduate students since last 25years.
- PG an PhD guide
- Working as an R&D executive, MR for ISO in an Ayurvedic pharmacy (Muniyal Ayurveda Research Center) and actively involved in,
- Design and development of Ayurvedic formulations
- Quality control of Ayurvedic medicines
- Research on single herbs and herbomineral preparations
- Clinical Research in Muniyal Ayurveda Hospital under various Chikitsa Kalpa

SPEAKERS

Artificial Intelligence, Machine Learning, Computer Vision, Natural language processing

Dr. Sripana Saha
Indian Institute of Technology Patna, Bihta Patna District, India

Breast cancer is a concerning disease due to its high incidence and mortality. In recent decades, the incidence and mortality have continuously increased. Its early-stage detection and the correct prognosis is the only effective way to eradicate fatalities caused. The prognosis and diagnosis of cancer primarily rely on clinical, genomic, and histopathological tissue image modalities. To have a better understanding of the situation and proceed with the correct treatment, it is required to explore all the possible modalities. The complex and heterogeneous nature of these modalities along with the variations of clinical outcomes in this disease poses a serious challenge in the prognosis. So, the situation demands some artificial-intelligence automated system for more accurate and reliable prognosis prediction of cancer patients.

In recent years, we have exploited gene expression, DNA methylation, copy number variation/alteration, and microRNA expression with regard to genomics, clinical profiles for previous conditions, history, lifestyle, and severity of the disease, and histopathological tissue image for cell-level visualization of cancer patients. We further utilized this information to design multi-modal machine-learning architectures for the prognostication of breast cancer. Our extensive experiments and comparative analysis support the superiority of proposed architectures over many other state-of-the-art methods. My talk will discuss the architectures and algorithms that we have developed for multimodal breast cancer prognosis prediction.

The corresponding publications are listed below:

1. Nikhilanand Arya and Sriparna Saha. Deviation-Support based Fuzzy Ensemble of Multimodal Deep Learning classifiers for Breast Cancer Prognosis Prediction. Scientific Reports. Nature Publishing Group, 2023.(Impact Factor:4.99, h5-index: 206).

2. Nikhilanand Arya, Sriparna Saha, Archana Mathur and Snehanshu Saha. Improving the Robustness and Stability of a Machine Learning Model for Breast Cancer Prognosis through the use of Multi-Modal Classifiers. Scientific Reports, volume 13, pages 4079. Nature Publishing Group, 2023.(Impact Factor: 4.99, h5-index: 206).

3. Nikhilanand Arya, Sriparna Saha, Archana Mathur, and Snehanshu Saha. Proposal of SVM Utility Kernel for Breast Cancer Survival Estimation. IEEE/ACM Transactions on Computational Biology and Bioinformatics, 2022.(Impact Factor: 3.702).

4. Nikhilanand Arya and Sriparna Saha. Multi-modal advanced deep learning architectures for breast cancer survival prediction. Knowledge-Based Systems, volume 221, pages 106965. Elsevier, 2021.(Impact Factor: 8.139).

5. Nikhilanand Arya and Sriparna Saha. Generative incomplete multi-view prognosis predictor for breast cancer: GIMPP. IEEE/ACM Transactions on Computational Biology and Bioinformatics, 2021.(Impact Factor: 3.702).

6. Nikhilanand Arya and Sriparna Saha. Multi-Modal Classification for Human Breast Cancer Prognosis Prediction: Proposal of Deep-Learning Based Stacked Ensemble Model. IEEE/ACM Transactions on Computational Biology and Bioinformatics, 2020.(Impact Factor:3.702).

Biography: Dr. Sriparna Saha is currently an Associate Professor in the Department of Computer Science and Engineering, Indian Institute of Technology Patna, India. She has authored or coauthored more than 450 papers. Her current research interests include natural language processing, machine learning, deep learning, multiobjective optimization and biomedical information extraction. Her h-index is 55 and total citation count of her papers is 16408 (according to Google scholar). She is also a senior member of IEEE, ACM, fellow of IET, UK; IETE. She is the recipient of Lt Rashi Roy Memorial Gold Medal from the Indian Statistical Institute for outstanding performance in MTech (computer science). She is the recipient of the Google India Women in Engineering Award, 2008, INSA Young Associate 2024, INSA Distinguished Lecture Fellow 2025, NASI YOUNG SCIENTIST PLATINUM JUBILEE AWARD 2016, BIRD Award 2016, IEI Young Engineers' Award 2016, SERB WOMEN IN EXCELLENCE AWARD 2018, Pattern Recognition Letters Associate Editor Award 2023, SERB Early Career Research Award 2018, Young Faculty Research Fellowship" under Visvesvaraya PhD Scheme for

Electronics & IT, Jan, 2019–Jan, 2024. She is the recipient of Fulbright-Nehru Academic and Professional Excellence Fellowship 2025, Humboldt Research Fellowship, Indo-U.S. Fellowship for Women in STEMM (WISTEMM)Women Overseas Fellowship program 2018 and CNRS fellowship.

For more information, please visit https://www.iitp.ac.in/~sriparna/.

SPEAKERS

A novel ECM associated protein modulates matrix stiffness to impede glioma progression

Varinder Singh1,2*, Devansh Swadia1, Pritam Saha1, Debasish Nath3, Nilesh Deokate1 and Sabyasachi Rakshit1#

1. Department of Chemical Science, Indian Institute of Science Education and Research Mohali, Punjab-140306

*2. *Current Affiliation: Department of Experimental Medicine and Biotechnology, Postgraduate Institute of Medical Education and Research, Chandigarh -160012*

3. Institute of Nanoscience and Technology, Mohali, Punjab-140306

 # Corresponding author

Background: Extracellular matrix (ECM) stiffening is a hallmark of glioma malignancy that promotes tumor invasion and therapeutic resistance, yet the molecular regulators governing tumor matrix mechanics remain poorly defined. We identified a novel ECM-associated protein, enriched in low-grade gliomas, as a candidate regulator of tumor matrix mechanics and glioma progression.

Methods: Transcriptomic profiling of low-grade glioma (LGG) and glioblastoma (GBM) TCGA cohorts ($log_2FC>1$; $q<0.01$ was performed using GEPIA2 webserver. Isoform-specific expression and alternative splicing were analysed using TCGASpliceSeq (PSI values). IDH1 mutation-associated regulation was assessed via cBioPortal, and validated by RT-qPCR in U87MG cells. Copy Number Variation (CNV) association with gene expression was analysed from cBioportal. Functional assays included cell proliferation assay, 3D spheroid invasion assays in Matrigel/Collagen I matrices supplemented with recombinant protein, and temozolomide (TMZ) cytotoxicity quantified by Annexin V/PI flow cytometry. Matrix mechanics were quantified by custom-built magnetic tweezers (stiffness measurements).

Results: The identified protein was significantly enriched in LGG ($log_2FC = 2.48$; $p < 0.0001$) and reduced in GBM ($log_2FC = -2.01$; $p < 0.0001$), with high expression predicting improved overall survival (HR=0.28; $p<0.0001$). Its elevated expression in LGG was associated with an IDH1-mutation linked reduction in gene methylation, while progressive copy-number loss drove stage-dependent expression reduction in GBM. Functionally, overexpression of the protein suppressed U87MG proliferation, reduced spheroid invasion area by ~50 μm^2 at 120 h ($p=0.0132$). Incorporation of the recombinant protein enhanced TMZ-induced cell death by up to 17% ($p<0.001$). Mechanistically, the protein reduced bulk ECM stiffness in a concentration-dependent manner (magnetic tweezers), generating a mechanically compliant matrix.

Conclusion: The study identifies a novel ECM-associated protein that reduces tumor matrix stiffness, suppressing glioma invasion and sensitizing tumors to temozolomide. These findings highlight ECM mechanical normalization as a promising therapeutic strategy in glioblastoma.

Biography: Technical Skills - Cell & tissue culture techniques: Extensive experience in handling mammalian cell cultures both adherent and suspension, generation and maintenance of stable cell lines; lentiviral transduction; recombinant protein expression in mammalian cells; 3D spheroid culture systems; isolation of exosomes and extracellular vesicles.

Flow cytometry & imaging: Proficient in flow cytometric analysis of cells, spheroids, exosomes, and extracellular vesicles; confocal microscopy for cellular and subcellular imaging.

Molecular & cellular biology: Skilled in PCR, qRT-PCR, gene cloning, isolation of DNA, RNA, miRNA and proteins from cultured cells, exosomes, and clinical specimens; experienced in western blotting and thermal shift assays.

Clinical & Microbiological techniques: Trained in venepuncture for human blood collection for research purposes; experienced in microbial culture, maintenance of bacterial strains, and recombinant protein expression and purification from bacterial systems.

Computational & analytical methods: Experience in virtual screening, molecular docking, and thermal shift assay-based validation for ligand identification; worked with with PyRx and AutoDock for in silico identification of ligands for proteins.

Softwares: GraphPad and Adobe Illustrator for statistical analysis and figures preparation.

Work Experience

1. CSIR-Junior Research Fellow from July 2011 to July 2012, in the lab of Prof. Neena Capalash at Deptt. of Biotechnology, Panjab

University, Chandigarh.

2. Postdoctoral Fellow from May 2019 to November 2021 in the lab of Dr. Sabyasachi Rakshit at Deptt. Of Chemical Sciences, Indian

Institute of Science Education and Research (IISER), Mohali.3. Postdoctoral Fellow from April 2022 to March 2023 in the lab of Dr. Sharmistha Sinha at Deptt. Of Chemical Biology, Institute of Nano

Science and Technology (INST), Mohali.

4. Senior Demonstrator, Deptt. of Experimental Medicine & Biotechnology, PGIMER, Chandigarh,

--- from September 2023 to December 2023; &

--- March 2024 -- ongoing

SPEAKERS

Cancer Prevention and the Role of Diet and Lifestyle in Cancer Awareness

Dr. Gaurang Joshi,
International Ayurveda Physician and Ayurveda Cancer Expert,
Director-Atharva Multi-Specialty Ayurveda Hospital, Cancer Research Center, Rajkot.

Cancer represents a multifactorial, non-communicable disease driven by complex interactions among genetic susceptibility, metabolic dysregulation, chronic inflammation, environmental exposures, and lifestyle factors. Epidemiological evidence suggests that a substantial proportion of cancers are preventable through modification of diet, physical activity, stress, and daily behavioral patterns. This abstract, presented for the Global Cancer Summit, examines cancer prevention and awareness through an Ayurvedic framework, with a focus on diet and lifestyle, as articulated by International Ayurveda Physician Dr. Gaurang Joshi, and contextualized within contemporary biomedical understanding.

Ayurveda describes carcinogenesis as a chronic, multistage process associated with impaired metabolic regulation (Agni Mandya), systemic inflammation, accumulation of toxic metabolic byproducts (Ama), immune dysfunction (Ojas Kshaya), and progressive tissue pathology (Dhatu Dushti). These concepts parallel modern scientific models involving oxidative stress, mitochondrial dysfunction, immune evasion, dysregulated cell signaling, and altered metabolism. Ayurvedic dietary principles (Ahara) emphasize whole, minimally processed, plant-forward nutrition; regulation of glycemic load; gut health optimization; and use of bioactive phytochemicals from herbs and spices with demonstrated antioxidant, anti-inflammatory, immunomodulatory, and chemopreventive properties. Lifestyle interventions (Vihara) focus on circadian rhythm alignment, physical activity, stress reduction, sleep optimization, and behavioral modification, aligning with current evidence linking these factors to reduced cancer risk via hormonal balance, epigenetic regulation, and immune surveillance.

Dr. Gaurang Joshi highlights that cancer awareness through Ayurveda is grounded in primary prevention, risk stratification based on individual constitution (Prakriti), and long-term health optimization rather than disease-centric intervention alone. Integrating Ayurvedic preventive strategies with evidence-based oncology and public health frameworks may enhance cancer awareness, reduce modifiable risk factors, and support sustainable, population-level cancer prevention. This integrative model offers a scientifically relevant and systems-oriented approach to global cancer prevention efforts.

Biography: B.A.M.S.

➢ Consulting International Ayurveda Physician

➢ Expert In All Chronic Diseases

➢ Ayurveda Skin Specialist

➢ Expert In Ayurveda Oncology (Cancer)

➢ President-International Psoriasis Foundation

➢ Director-Atharva Multispecialty Ayurveda Hospital, Cancer Research Centre, Panchakarma and Skin Care Hospital, Rajkot, Gujarat, India.

➢ Fouder Director-Atharva Global Academy of Ayurveda Accredited by Ayurveda Training Accreditation Board, Rashtriya Ayurveda Vidyapith,Ministry of Ayush, Govt. of India.

➢ Director-Green Tree Botanicals Pvt. Ltd.

➢ Chairman-Atharva Life Science Academic and Research Foundation (ALSAAR Foundation) Director and Professor-International School of Ayurveda, Colombia

➢ Organizer – World Ayurveda Conference, Ahmedabad, WACA 2019, Scheduled on 15th to 17th February

➢ Chairman- Global Ayurveda Research Foundation

➢ International Ambassador for India of World Spa Organization

➢ Ex. Director- Professional European Ayurvedic Center of Excellence (PEACE), Slovakia.

➢ President- National Integrated Medical Association (NIMA), Rajkot

➢ Ex. Vice President-Vishwa Ayurveda Parishad, Gujarat State

➢ Ex. Organizing Secretary -International Conference On Ayurved and CAM-RAJAYUCON 2013, Feb 23-24th2013, Rajkot

➢ Vice President-International Cancer Foundation

➢ Faculty for International Academy of Ayurved for Overseas

➢ Editor-Journal of Cancer Prevention and Current Research, MedCrave Group, Oklahoma, USA.

➢ Editorial Board member- International Journal of Dermatology,USA.

➢ Editor- Madridge Journal of Dermatology & Research,USA.

➢ Editor- Juniper Online Journal of Case Studies,USA

➢ Ex. hair Person –Clinical Board : Europe Ayurveda Academy

➢ Ex. Chairman-Skill Development and Personality Development, Vishwa Ayurved Parishad, Gujarat.

➢ Life member- Vijananbharti…

➢ Life Member- Shri Rakot Vaidyasabha, Rajkot

SPEAKERS

Intervention In Stenson Duct

Dr. Manoj Meena BDS, MDS

Prof and Head) Oral Medicine & Radiology Rajasthan Dental College and Hospital Jaipur MANA, MESC

OBJECT- Parotid gland tumor also known as pleomorphic adenoma which is one of the commonest forms of tumor of salivary gland in fourth to sixth decade of life. The present modality of treatment is superficial parotidectomy or complete removal.

METHOD - A minimal invasive procedure will conducted through a guide wire in the stenson duct. The procedure will be in day care in which patient vitals would be measure and regular blood investigation would be done. The complete procedure of invasion would be done under tropical anesthesia followed by local anesthesia. The procedure will be done with compete preservation of facial nerve followed by parotid gland.

OBSERVATION- since the above method is a study model

RESULT – The procedure will be done under the guidance of oral medicine and radiology expert only. This procedure will open a new aspect in dental surgery

Biography:

Professor & Head, Department of Oral Medicine & Radiology with 12+ years of academic and clinical experience. Expertise in oral diagnosis, radiology, TMJ disorders, oral cancer, and advanced imaging (CBCT). Active researcher, speaker, and innovator with multiple patents and publications.

Education

- MDS (Oral Medicine & Radiology) – Goa Dental College, 2013
- BDS – Surendra Dental College, Rajasthan, 2010

Current Position

- Professor & Head, Oral Medicine & Radiology

Rajasthan Dental College & Hospital, Jaipur

Key Skills

- Oral cancer & precancer diagnosis
- TMJ & orofacial pain management
- Dentomaxillofacial radiology (CBCT)
- Salivary gland & mucosal disease management
- Interventional & minimally invasive procedures
- Tobacco cessation counseling

Research & Innovations

- 5 Patents (Dental devices & imaging technologies)
- Multiple Books (2026) on oral cancer, imaging, and interdisciplinary innovations
- Thesis: Dermatoglyphics in oral precancer & cancer

Publications

- 40+ research papers in national & international journals
- Key areas: CBCT imaging, oral cancer, TMJ disorders, salivary diagnostics, bioinformatics

Conferences & Recognition

- National & international speaker (IAFOCON, Indo-Pacific Forensic Odontology, etc.)
- Chaired scientific sessions at multiple national conferences
- Best Paper Award – IAOMR Conference, 2012

Professional Membership

- Life Member – IAOMR

Workshops & Training

- CBCT, oral imaging, oncology (Tata Memorial Hospital)
- Multiple CME/CDE programs in oral medicine, radiology, and research

Presentations

- Multiple paper & poster presentations in oral medicine, radiology, and TMJ disorders

SPEAKERS

Computational Multi-Target Profiling of Tadalafil and Skimmianine: Promising Therapeutic Strategies for Glioblastoma Management

Neha

Department of Forensic Science, School of Basic and Applied Sciences, SGT University, India; neha_sbas@sgtuniversity.org

Glioblastoma (GBM) is the most aggressive primary brain tumor, characterized by rapid progression, therapeutic resistance, and poor prognosis, necessitating the development of multi-target therapeutic strategies. This study employed an integrated computational approach to investigate the anti-glioblastoma potential of Tadalafil, a repurposed phosphodiesterase-5 inhibitor, and Skimmianine, a natural furoquinoline alkaloid. Network pharmacology identified 28 common targets for Tadalafil associated with GBM and its neuropsychiatric comorbidities, while 72 overlapping targets were identified for Skimmianine against GBM. Protein–protein interaction and pathway enrichment analyses revealed key hub genes involved in tumor proliferation, angiogenesis, inflammation, apoptosis, immune regulation, and therapeutic resistance. Molecular docking demonstrated strong binding affinities of Tadalafil toward TOP2A, AXL, MMP2, MMP9, and NFKB1, whereas Skimmianine exhibited favorable interactions with ERK1, AKT1, JAK2, EGFR, mTOR, PI3K, STAT3, VEGFR2, and PD-L1, supporting their multi-target therapeutic potential. Furthermore, in vivo evaluation of Tadalafil in an ENU-induced glioblastoma rat model demonstrated significant improvement in cognitive function and preservation of hippocampal architecture, confirming its neuroprotective effects. Collectively, these findings highlight the complementary potential of drug repurposing and natural product-based therapeutics for GBM management and provide a strong rationale for further preclinical and translational investigations.

Keywords: Glioblastoma, Tadalafil, Skimmianine, Network Pharmacology, Molecular Docking, Drug Repurposing, Multi-Target Therapy, Neuroprotection, Computational Drug Discovery, Natural Products.

Biography: Neha Received a memento during an academic visit to Jamia Hamdard on 5th December 2024.

• Secured 2nd place in the poster presentation and received a certificate at the symposium organized by the 3600 Club, Department of Forensic Science, FABS, from 16th to 20th December 2024.

• Received Research excellence award, Research & Development Cell, SGT University, 6th February 2026.

• Received Faculty Research Coordinator Award, Research & Development Cell, SGT University, 6th February, 2026.

• ISN-APSN-YIC for the

• APSN-FAONS-HKSN 2026 Neuroscience Conference (August 23-26, 2026) in

Hong Kong has been accepted. As a YIC awardee, will receive USD750 as a subsidy to your cost

SPEAKERS

Precision Theranostics: Integrating Logic-Gated Nanoplatforms with ctDNA-Guided MRD Assessment for Optimized Oncology Care

Prof. Rekha Kumari,
Former Director Higher Education. Bihar, Professor& Head, University Department of Zoology, Patliputra University, Patna, Bihar

Introduction: Despite advancements in systemic therapy, the clinical management of solid malignancies remains hindered by two persistent challenges: sub-clinical relapse due to undetected molecular residual disease (MRD) and systemic toxicity arising from off-target therapeutic delivery. This research proposes a novel, integrated framework that combines high-sensitivity liquid biopsy monitoring with stimuli-responsive nanomedicine to transition oncology toward a truly "precision-active" paradigm.

Methods: We investigate the clinical utility of longitudinal circulating tumor DNA (ctDNA) profiling to identify patients with MRD post-adjuvant intervention. Building upon this diagnostic precision, we introduce a logic-gated Nanoplatforms engineered to respond exclusively to the tumor microenvironment's (TME) unique biochemical signatures—specifically, simultaneous pH-sensing and enzyme-triggered release. This "AND-gate" mechanism ensures therapeutic activation only at the site of residual disease, minimizing systemic exposure.

Results: Preliminary data indicates that the integration of ctDNA-directed treatment modulation enables the early detection of molecular relapse up to 6 months prior to radiological evidence. Furthermore, the application of logic-gated nanocarriers demonstrated a significant reduction in off-target toxicity in preclinical models, maintaining high therapeutic efficacy even in immune-cold, treatment-resistant phenotypes. By bypassing traditional systemic dosing, this approach allows for sustained, localized tumor modulation.

Conclusion: The synergy between real-time molecular diagnostics and "smart" nanomedicine represents a critical evolution in the 21st-century oncology toolkit. By shifting the focus from generalized cytotoxic delivery to site-specific, TME-responsive intervention, we can effectively mitigate the burden of treatment-related morbidity. This framework offers a scalable, data-driven pathway for achieving more durable, personalized outcomes for cancer patients, aligning with the 2026 goals for sustainable, precision-centered oncology care.

Keywords: logic –gated nano-platform, logic- gated nanocarriers, Molecular residual disease (MRD)

Longitudinal circulating Tumor (ctDNA)

Biography: Prof. (Dr.) Rekha Kumari is a distinguished academician, researcher, and higher education administrator with more than 29 years of teaching experience and over 33 years of research in the fields of Zoology, Immunology, Endocrinology, Fish Biology, and Nanobiotechnology. She is currently serving as University Professor and Head, Department of Zoology, Patliputra University, Patna, Bihar, India.

Prof. Kumari has the unique distinction of serving two consecutive terms as Director of Higher Education, Government of Bihar, where she played a pivotal role in policy formulation, academic reforms, and the modernization of higher education. Her research contributions include 39 publications in national and international journals, four books, several book chapters, and the successful supervision of numerous doctoral scholars.

Her recent research focuses on nanobiotechnology, cancer biology, immunology, and the biomedical applications of eco-friendly nanomaterials. She has led UGC-funded research projects, delivered invited lectures at prestigious national and international conferences, and received several honors, including the Lifetime Achievement Award and the Innovative Scientist Award.

As a speaker at the 7th Global Cancer Summit 2026, Prof. Kumari brings extensive expertise in translational research, nanotechnology, and biomedical sciences, fostering interdisciplinary collaborations for advancing cancer research and healthcare.

SPEAKERS

The Convergence of Artificial intelligence (AI) Biopharma and Genomics

Dr. T S Rao

Former Senior Adviser, Department of Biotechnology, Ministry of Science and Technology, Govt. of India.

The convergence of Artificial Intelligence (AI), Biopharma, and Genomics represents a seismic shift toward "precision at scale"—the ability to deliver highly personalized healthcare to large populations rather than niche groups. By integrating massive, multi-omic datasets with machine learning, this intersection is accelerating drug discovery, optimizing therapeutic design, and tailoring medical treatments to individual genetic, environmental, and lifestyle factors.

Key Intersections and Technologies

The meeting of these three fields is characterized by several key advancements:

• AI-Powered Genomic Analysis: AI accelerates the interpretation of vast genomic datasets, enabling the identification of complex disease-associated variants and patterns that classical, manual methods would miss.

• Predictive Modeling in Drug Development: AI transforms drug discovery by predicting drug-target interactions, optimizing molecular structures, and forecasting toxicity, reducing the drug discovery timeline by 1–2 years.

• Multimodal AI for Biomaterials & Therapy: AI models analyze genetic profiles to predict immune responses to treatments (such as implants or cell therapies), optimizing materials for individual patient compatibility.

• Digital Twins and Clinical Trial Optimization: AI enables the creation of "digital twins"—virtual patient models—allowing researchers to simulate responses to drugs, thereby streamlining clinical trials and improving patient selection.

Impact on Biopharma and Healthcare

• Accelerating Drug Discovery: AI algorithms are now capable of generating novel drug molecules and improving the success rate of Phase 1 trials to 80-90% compared to the industry average of ~40-65%.

• Precision Oncology & Rare Diseases: AI and genomics are enabling faster diagnosis and targeted treatments for cancer and rare diseases, focusing on identifying actionable mutations.

• Personalized mRNA Therapeutics: AI is revolutionizing RNA medicine, including the optimization of coding sequences, untranslated regions, and lipid nanoparticle design, accelerating the development of personalized mRNA therapies.

Market and Future Trends

• Rapid Market Growth: The AI in genomics market is projected to grow from 1.26 billion in 2025 to 18.82 billion by 2033, growing at a CAGR of 40.30%, with drug discovery and development holding the largest market share.

• Shift to Agentic AI: The next 24 months are expected to see a rise in "agentic AI"—AI agents capable of automating complex, iterative design-validation cycles.

• Key Drivers: The main drivers for this revolution are falling costs of genomic sequencing, increasing computational power, and the integration of real-world evidence from electronic health records (EHRs).

Challenges to Scaling

Despite the potential, the field faces significant hurdles:

• Data Silos and Quality: Accessing high-quality, structured, and diverse data for training algorithms is difficult.

• Regulatory & Ethical Hurdles: Ensuring patient privacy and establishing intellectual property protections for AI-discovered compounds.

• Interpretability: Understanding the rationale behind AI-driven predictions is critical for clinical adoption.

Biography: Dr. TS Rao is a former Senior Adviser (Additional Secretary) in the Department of Biotechnology (DBT), Ministry of Science and Technology, Govt. of India. Dr. Rao has been associated with the DBT for the past 34 years (since its inception) and has coordinated programs related to Medical Biotechnology, Vaccines, Human Genetics, Medical technologies, Translational research etc. Dr. Rao played a significant role in the Polio Eradication programme of the Govt. of India from 1987-88, and India became a polio-free Nation in 2014.

Coordinated the development and commercialization of the first indigenously developed ROTAVAC and ROTASIL vaccines, which were introduced in the National Immunization Program in all states. Involved in developing Covaxin, including intranasal vaccine for Covid-19. Established BIBCOL, IVCOL, NBRC, THSTI, InStem and Niche centres, viz, VIDRC, PBC, CBD, CDSA, CAMP, CCBT, CNS etc. Establish several biotech science clusters including NCR and B-Life Bengaluru. Developed DPR for IIT Delhi to establish a Health Technology Campus (HTC) with NCI- AIIMS Jhajjar. Associated with development of immunotherapy for lung and ovarian cancers (cancer vaccine). Dr Rao is a member of several expert committees of national and international organizations. Dr. Rao has about 23 scientific publications and popular articles to his credit in National and International Peer Reviewed Journals in the area of Vaccines, Genetics, immunology etc. He has also published book chapters in the field of biomedical research in both languages (Hindi and English).

Awarded Prestigious Distinguished Service Award of USIBS at Atlanta, USA and Pratibha Puraskar of Government of AP in 2015. Dr. Rao received HonorisCausa, Honorary D.Sc. Degree from NITTE University (Medical) in Nov 2022 for his significant contribution to public health, especially in the polio eradication program, development of indigenous ROTA and Covid vaccines. He was also awarded the “Bharat ke ANMOL” National Award on 31st July 2023 for his significant contribution to health sciences over the past three decades. Dr. Rao was involved in G-21 Indo-African initiative in vaccines, pharma and medical technologies. Dr. Rao is a part of scientific advisory committee of Govt of AP. Dr. Rao received Globax international award on 5th September, 2024 for significant contribution in biomedical research for developing cost-effective vaccine and affordable medical technologies. He has also published book chapters in the field of biomedical research in both languages (Hindi and English).

SPEAKERS

Updated Guidance on Transvaginal Ultrasonography for Postmenopausal Bleeding

Prof Uma Pandey,
MD (BHU), FRCOG (London)

Biopsy may be omitted ONLY if ALL of the following apply:

➢ Single episode of postmenopausal bleeding

➢ Fully visualized endometrium ≤ 4 mm

No high-risk factors for endometrial cancer

Prompt access to gynecologic follow-up

The American College of Obstetricians and Gynecologists (ACOG) released updated clinical guidance for evaluating postmenopausal bleeding, recommending that most patients undergo both transvaginal ultrasonography (TVUS) and endometrial tissue sampling (biopsy) during initial evaluation

The updated ACOG Clinical Practice Update shifts away from the previous reliance on TVUS alone, aiming to catch the steady rise in endometrial cancer cases and prevent missed malignancies.

Cancer of the endometrium is the most common type of gynecologic cancer in the United States. Vaginal bleeding is the presenting sign in more than 90% of postmenopausal women with endometrial carcinoma. Clinical risk factors for endometrial cancer, including but not limited to age, obesity, use of unopposed estrogen, specific medical comorbidities (eg, polycystic ovary syndrome, type 2 diabetes mellitus, atypical glandular cells on screening cervical cytology), and family history of gynecologic malignancy also should be considered when evaluating postmenopausal bleeding.

Transvaginal ultrasonography usually is sufficient for an initial evaluation of postmenopausal bleeding if the ultrasound images reveal a thin endometrial echo (less than or equal to 4 mm), given that an endometrial thickness of 4 mm or less has a greater than 99% negative predictive value for endometrial cancer. Transvaginal ultrasonography is a reasonable alternative to endometrial sampling as a first approach in evaluating a postmenopausal woman with an initial episode of bleeding. If blind sampling does not reveal endometrial hyperplasia or malignancy, further testing, such as hysteroscopy with dilation and curettage, is warranted in the evaluation of women with persistent or recurrent bleeding. An endometrial measurement greater than 4 mm that is incidentally discovered in a postmenopausal patient without bleeding need not routinely trigger evaluation, although an individualized assessment based on patient characteristics and risk factors is appropriate. Transvaginal ultrasonography is not an appropriate screening tool for endometrial cancer in postmenopausal women without bleeding.

Biography: Professor and Former Head of the Department of Obstetrics and Gynaecology, Institute of Medical Sciences, Banaras Hindu University, Varanasi.

Examiner for MRCOG Examinations.

Co-opted Representative, RCOG North Zone 2023-2025.

CHAMPION Trial: Principal investigator @ BHU, Published in New England Journal of Medicine; and the trial findings were incorporated in WHO PPH recommendations.

PI @ BHU for REACH trial, WHO.

SPEAKERS

What are the numbers needed to diagnose one cancer case? Outcome of a multi-cancer detection screening program in a community setting

Gunjan Shrivastav

Consultant, Department of Medical Oncology, Cancer Hospital & Research Institute, Gwalior, MP

INTRODUCTION: Early diagnosis remains pivotal in improving oncological outcomes. While cancer screening has been integrated in state policy in some countries, it is limited to only cervical cancer(CC) screening in our country. In addition to CC, breast cancer(BC) and head and neck cancer(HNC) are prevalent in India and accessible for screening.

MATERIAL & METHODS: Cancer Hospital & Research Institute(CHRI) has been conducting cancer screening camps in community setting. These camps include Papsmear, FNAC, Biopsy facility with added mammography, screening by infra red device for oral cancers and Nirmaya cancer screening in some camps. We audited the camp conducted between October 2023 and 2025 to calculate the numbers of investigations needed to diagnose(NND).

RESULTS: Over a period of 2 years, 80 community screening camps were conducted. 7000 people attended the camp and 3189 (65% males and 35% females) underwent doctor's consultation. 1650(51.7%) had at least one investigation that included pap smear(737), FNAC(9), Biopsy(37), Nirmaya breast screening(35), oral infra red screening(45) and mammographies(747). Total 30 malignant and one premalignant lesions were diagnosed. Malignancy consisted of HNC(22), breast(1), cervix(2), lymphoma(4) and melanoma(1). Yield of investigation was 1.9%. NND for one malignant case was 55 and one malignant plus premalignant was 53. No case was diagnosed by mammography.

CONCLUSION: Despite awareness sessions before camps, general population is reluctant to undergo cancer screening. Targeting single cancer decreases the cost effectiveness and we advocate a multi-cancer detection community camp preceded by awareness session. Utility of mammography in community program is still to be validated. In our study NND was 53 as majority patients to undergo investigation are symptomatic. We need a more comprehensive and intensive community program to diagnose cases at pre-malignant stage.

Biography:

ACADEMIA

DrNB Medical Oncology from Max Super Speciality Hospital, Saket, Delhi (2021).

MD General Medicine from Sri Aurobindo Medical College & PG Institute, Indore, MP (2015).

MBBS from Peoples College of Medical Science, Bhopal, MP (2005).

COURSES & TRAINING

Executive Education Course by Harvard Medical School on "Advanced Management of Oncological Diseases" (2024).

The Lung Cancer Preceptorship by Princess Margaret Cancer Centre, 2025.

Attended the workshop, "Synapse 2025" on Artificial Intelligence and molecular oncology conducted at IIT Guwahati, 14- 15 Nov 2025.

EXPERIENCE

Senior Resident, Department of Medicine, Tata Memorial Hospital, Parel, Mumbai (Oct 2016 - Feb 2017).

Senior Resident, Department of Medical Oncology, Hinduja Hospital, Mahim, Mumbai (Oct 2017 - Nov 2017).

Consultant, Department of Medical Oncology, Cancer Hospital & Research Institute, Gwalior, MP (Sep 2021 - Present).

o Started the Pediatric Oncology Department at Cancer Hospital & Research Institute, Gwalior.

CONFERENCES

Organized the National CME on Paediatric and Haematological Malignancies at Gwalior, 30.05.2026.

Secretary, 53rd Indian Cooperative Oncology Network Conference, Gwalior (2025).

Secretary, National Conference on "Hospital Infection Control Conference, HICON, Gwalior 2024 & 2025".

Co-Secretary, MP GYNEC-ONCON 2024, Gwalior (14-15 Sep 2024). Invited as panellist at the 9th LION conference, Bhopal, April 2026.

Invited ad faculty at the 7th Annual Meeting of Supporitve Care in Cancer hosted by IASCC in collaboration with MASCC at Bomaby on 29th nov'25.

Invited as panellist at the 52nd Indian Co-operative Oncology Network Conference, Delhi (2025).

Invited as faculty at the 6th Annual Asia-Pacific Cholangiocarcinoma Conference, Tata Memorial Hospital, Mumbai (2023).

Invited as panellist at the BALCO Cancer Conclave, Raipur (2024).

Invited as chairperson at the PICSEP Workshop organized by GOGS & FOGSI (2024).

Invited as faculty at the Max Cancer Congress (2023).

Invited as faculty at the MPCCON 2023, Indore.

Invited as faculty at the Bhopal Oncology Update-1 (2023).

SPEAKERS

Safety Profile of Alpelisib Plus Fulvestrant in PIK3CA-Mutated HR-Positive, HER2-Negative Advanced or Metastatic Breast Cancer: A Phase IV Multicenter Study in Indian Patients

Kaushal Patel, MBBS,MD, DM (Medical Oncology)

A 702, CapitalLife, Opp. Poddar Residency, Canal Road, Vesu, Surat – 395007

This Phase IV, prospective, multicentre, open-label, non-comparative interventional study in India evaluated the safety of planned treatment for 6 months of alpelisib 300 mg once daily plus fulvestrant 500 mg in patients with PIK3CA-mutated, HR-positive, HER2-negative advanced or metastatic breast cancer whose disease had progressed on or after prior endocrine therapy. The primary endpoint was the proportion of patients experiencing at least one treatment-emergent adverse event (TEAE), defined as any event occurring or worsening from the first dose until 30 days after the last dose.while the secondary endpoints were proportion of patients with TEAE related by investigator and severe adverse reaction (SAE). A total of 40 patients was enrolled, all of whom were postmenopausal women, with a mean age of 54.9 years (ranging between 38-91). 87.5% patients had stage IV disease and 10% had Stage IIIA disease. 75% had invasive ductal carcinoma, 10% had metaplastic disease and 7.5% had Ductal-carcinoma-in-situ. The median (IQR) treatment duration was 5.5 (2.65) months. All patients experienced at least one TEAE, with 311 TEAEs reported overall. 24% of all AEs were Grade 3 or higher TEAEs and occurred in 77.5% of patients. The most common TEAEs were hyperglycaemia (90.0%), stomatitis (40.0%), and diarrhoea (37.5%). Grade 3 hyperglycaemia occurred in 55.0% of patients. However, at baseline, 33.0% of patients had prediabetes and 11.0% had diabetes. The median (IQR) time to onset of hyperglycaemia adverse event was 8 (1.5) days. Most TEAEs resolved completely (81.0%), and recovery or resolution was documented in 92.5% of patients, allowing treatment continuation in most cases. Four patients were discontinued due to adverse events. Dose reductions were required in 60.0% and 27.5% of patients to 250 mg and 200 mg, respectively. Overall, the safety profile was consistent with the pivotal SOLAR-1 trial.

Keywords:

alpelisib, fulvestrant, PIK3CA mutation, HR-positive breast cancer, HER2-negative, advanced breast cancer, metastatic breast cancer, safety, hyperglycemia, India, post-menopausal women

Biography:

CURRENT AFFILIATIONS:

• Senior Consultant Medical Oncologist and Hematologist & Director of Department at following Institute:

1. Elite Hemat Onco Care Centre, Nanpura, Surat (Own Clinic)
2. Bharat Cancer Hospital, Saroli, Surat
3. Mahavir Hospital, Surat
4. Sunshine Global Hospital, Surat
5. Metas Adventis Hospital, Surat
6. Nirali Cancer Hospital, Navsari
7. Visiting All Surat Hospitals, Surat

SPEAKERS

Relevance of Early Diagnosis in Breast Cancer Patients

Dr Ajit Kumar Saxena
Director, R S Memorial Cancer Research Centre, Patna Bihar India

Breast cancer is a frontier area for women's diseases that increases the burden every year for morbidity and mortality says World Health Organization and becomes a challenge for clinicians and scientists. The management of breast cancer is not as easy what people think because of dual origin- hereditary or sporadic in nature. No doubt, the origin of cancer is the activation of oncogenes due to reciprocal translocation of chromosomes or complex chromosome rearrangements (CCRs) by epigenetic factors. Only classical devices are known for the diagnosis of breast cancer patients based on histopathological or immunological investigations that required time consuming and their efficiency is also doubtful, even with the advancement of technology like RTPCR using biomarkers. Still there is no device is available for early diagnosis of cancer patients. Recently, circulating tumor cells (CTC), the FDA (USA) approved the application for early diagnosis of primary tumours to metastatic stage using epithelial-mesenchymal markers- Sox4, EpCAM, cytokeratin 19 and Vimentin using monoclonal antibodies with the help of flowcytometeric analysis. The study of CTCs population in cancer patients is very difficult phenomenon because of its very less population i.e 3 or 4 in number per 7.5 ml of blood. Classical methodology is based on 15 to 20 ml of blood is required for CTCs using Ficoll's gradient method. The phenotypic expression of cancer patients is aggressive in nature because of discordance in the frequency of expression of early transcription factor (Sox4), CK19, and EpCAM. Vimentin, a glycoprotein, is abundant in cytoplasm becomes relevant EMT marker for clinical diagnosis to confirm metastasis in tumor patients. The transition of EMT markers from the primary tumor site to the metastatic stage during onset of tumorigenesis is still unknown. Present study has been designed with the aim to reduce sample size (1.0ml blood), and, with help of lymphocyte cultures for 72 hrs to increase the CTC population. Ficoll gradient procedure was adapted for CTCs isolation and after DNA/RNA isolation, EMT marker expression were observed by RT PCR using specific primers. Agarose gel (1.5%) was used for the characterization of bands after using fluorescence dye- ethidium bromide staining and DNA intensity of individual bands was characterized by software inbuilt inside Gel Doc system.(USA) for copy number variations to evaluate the genetic susceptibility of the individual cancer patient. The sensitivity of CTCs was further evaluated by two anticancer drugs used by clinicians- 5 Azacytidine and cyclophosphamide using in vitro for therapeutics and examine the same EMT markers to explore the etiopathology between the drugs and breast cancer patients. Findings reveal discordance in the percentage frequency of EMTs markers, but significant differences were observed at 72-hour exposure of drugs, suggesting longer exposure of these drugs makes efficiency and may be helpful for better management of cancer patients.

Secondly, vimentin – a metastatic marker showing isoform, reporting first time in CTCs might have increased the efficiency of the drug to bind a subpopulation of proliferating tumor cells. Simultaneously, the data of the DNA copy number variations becomes relevant to evaluate individual genetic susceptibility that support the above findings of the CTCs population with different age groups of the patients. CTCs population is significantly higher in the young age group, followed by a decrease as age increases. Several questions arise for clinicians: how to differentiate management for cancer patients between familial and non-familial cases? To answer of this question further study is required to reverse the E→T to T→E phenomenon by developing new molecules and save the life of cancer patients otherwise not possible

Keywords: Breast Cancer; Circulating Tumour Cells, Flowcytometer, RT-PCR

Biography: Dr Ajit K Saxena, Gold Medalist, Academician, Philosopher and Writer. He obtained his PhD (Anatomy-Cytogenetics) from Institute of Medical Sciences, BHU, Varanasi, and D.Sc in Clinical Genetic). He did Post Doctorate from NIOH, USA. Dr Saxena possess more than thirty years of Teaching, Training, Clinical Diagnosis of Suspected Genetically Abnormal Patients to Confirm the Diagnosis with Research Experience in India and Abroad. Presently his research work published in more than 110 peer reviewed index journals comprises Cytogenetics, Molecular Genetics and Stem Cell Biology associated risk factor in Birth Defects, Infertility and Cancer. He also present his research work of about more than 100 National and International Conferences, Dr Saxena is recipient of more than thirty awards including Life Time Achievement awards , Excellence award from Times Groups, Excellence Health Care award from Indian Medical Association, Federation of Indian Chamber of Commerce for outstanding contribution in Clinical and Basic Research etc. Dr Saxena is very humble and amicable in nature among the students and colleagues. He is author of books including -1. Genes and Male Infertility in Human Scientific Media (UK) 2019, 2. FISH Analysis for Drugs and Chemicals Mediated Cellular Toxicity Springer, , Nature Group, UK 2020.3. Novel Corona Virus 19 – In-silico Vaccine Design and Drug Discovery in Springer Nature Group, UK, 2020. 4. Basic and Clinical Consequences of Genetic Disorders in Human Cambridge Scholars, UK, 2021. Dr Ajit K Saxena patented four his original research work on decoding of gene and protein structure and new technology for circulating tumor cells. Dr Saxena also possess the ability as Head of the Department in India and abroad, Examiner of National Board of Examination., New Delhi, and Advisor of the Selection Committee of Union Public Service Commission. New Delhi. Dr Saxena is a fellow and member of varies academic societies in India and USA. He believes that work is worship

SPEAKERS

LncRNA YIYA Promotes Metabolic Adaptation in Pancreatic Cancer through KRASDriven Glycolysis and GOT1-Mediated Glutamine Metabolism under Hyperglycemia

Dr. Rajdeep Chowdhury
Professor and Head, Department of Biological Sciences
Birla Institute of Technology and Science, Pilani, Pilani Campus, Pilani 333 031, Rajasthan, India

Pancreatic ductal adenocarcinoma (PDAC) is characterised by a strong association with diabetes-induced hyperglycemia. However, the regulatory mechanisms linking hyperglycemia to the metabolic adaptations in PDACs that promote aggressiveness are poorly defined. In the present study, we identify the novel long non-coding RNA (lncRNA) YIYA as a glucose sensor that regulates hyperglycemia-driven metabolic reprogramming in PDAC. Functional analyses revealed that YIYA promotes cancer cell proliferation and the Warburg effect by simultaneously regulating glucose- and glutamine-related pathways essential for tumour growth. Mechanistically, YIYA promoted KRAS-driven metabolic reprogramming by interacting with KRAS, protecting itself from autophagic degradation, and upregulating the glycolytic enzyme PKM2, thereby reinforcing the Warburg phenotype. Beyond its effects on glycolysis, YIYA was also found to act as a competing endogenous RNA (ceRNA) in glutamine metabolism. Experimental validation demonstrated that a hyperglycemic state elevates glutamine uptake and YIYA directly interacts with miR-9-5p, functioning as a molecular sponge. As a result, repression of the key glutamine metabolic enzyme, glutamate oxaloacetate transaminase 1 (GOT1), by miR-9-5p is disabled, leading to elevated GOT1 expression. YIYA-mediated GOT1 upregulation promotes glutamine utilisation and supports maintaining cellular redox balance. Silencing of YIYA by siRNA or CRISPR led to decreased GOT1 expression, impaired glutamine metabolism, disrupted redox homeostasis, and reduced proliferative potential, highlighting the significance of these axes. Collectively, our findings establish YIYA as a central metabolic regulator that coordinates two major metabolic pathways in PDAC: KRAS-associated glycolytic reprogramming and glutamine-dependent redox maintenance. These results provide new insights into the molecular basis of metabolic adaptation in PDAC and identify the YIYA as a potential therapeutic vulnerability for targeting hyperglycemia-associated PDAC.

Biography:

Awards and Honours:

2008: Post-Doctoral Fellowship from the Department of Biotechnology (DBT)- Post

Doctoral Program in Biotechnology & Life Sciences.

2008: Selected for post of Application Scientist and Faculty at Imperial

Bio-services (ILS), Gurgaon, India.

2004: Qualified West Bengal School Service Commission for Assistant

Teacher of Zoology at Shaktigarh High School, West Bengal.

2003: Doctoral Fellowship from Council of Scientific & Industrial

Research (CSIR) in National Eligibility Test (NET).

2003: Doctoral Fellowship from Zoological Survey of India, Govt of India.

SPEAKERS

Formulation and evaluation of Andrographolide loaded emulgels: Bench to Bedside: Comprehensive In Vitro/ In Vivo Anticancer Potential

*N V L Sirisha Mulukuri*1, Pankaj Kumar2, Sujeet Kumar3, N.V. Satheesh Madhav4*

1. Department of Pharmacognosy, Nitte College of Pharmaceutical Sciences (NCOPS), Nitte (Deemed to be University), Bangalore, India.

2. Department of Pharmaceutical chemistry, NGSM Institute of Pharmaceutical Sciences (NGSMIPS), Nitte (Deemed to be University), Mangalore, India

3. Department of Pharmaceutical chemistry, Nitte College of Pharmaceutical Sciences (NCOPS), Nitte (Deemed to be University), Bangalore, India.

4. Vital Therapeutics and Formulations Pvt. Ltd., Abhinav Colony, Padmarao Nagar-500025, Hyderabad, Telangana, India

Objectives: The aim of the current study is to develop and evaluate Nano sized Andrographolide loaded Glycerogels for their potential against skin cancer with improved permeation.

Methods: Nano sized Andrographolide was done by sonicaiton followed by solvent evaporation technique. The best formulation was subjected for various physicochemical evaluations, release kinetics studies, particle size analysis, ex-vivo permeation studies, biocompatibility tests, cytotoxicity assays against A431 cell lines, cell cycle analysis, skin irritability tests, and in-vivo cellular uptake studies.

Results: The optimized nano-emulgel has exhibited the In-vitro drug release of 92.5 ± 0.5%, following Higuchi kinetics. Ex-vivo permeation studies showed approximately 90% drug release within 24 hours, with 10% retention in the epidermal layer, shows the superior skin permeation and retention properties of the optimized emulgel.

Conclusion: The enhanced permeability observed with the nano glycerogel formulations of Andrographolide indicates a promising novel phytotherapeutic approach for the treatment of skin cancer.

Biography:

Worked as Asst. Professor at Sri Vishnu College of Pharmacy, Bhimavaram, Andhra Pradesh, 2010-11.

* Worked as Asst. Professor at Aditya Bangalore Institute of Pharmaceutical Education and Research, Bangalore, Karnataka, 2011-12

* Worked as Asst. Professor at Karnataka College of Pharmacy, Bangalore, Karnataka, 2012-19

* Working as HOD & Associate Professor at Nitte College of Pharmaceutical Sciences, Bangalore from August 2019 to till date.

Total Teaching experience: 16 years

AWARDS AND RECOGNITION

* GPAT Rank 1566

* NIPER Rank 346

* Received 2nd position in South zone, DRPI-2021 under young researcher category.

* Received Best Poster presentation award during the paper presentation, conducted by Department of Pharmaceutical Chemistry, T. John College of Pharmacy, Bangalore on March 26, 2022.

* Received Best Oral Presentation award at Society of Pharmaceutical education and researchInternational conference, Mangalore on Jan 2025.

* Received Best PhD thesis award from RIPER R& D Centre, Ananthpur on Feb 2025

SPEAKERS

Autophagy as a Quality Control Mechanism Driving Glioma Stem Cell Maintenance and Chemoresistance

Dr. Meenakshi Tiwari, Ph.D.
Professor,
Department of Center for Advance Research, King George's Medical University, Lucknow

Glioblastoma (GBM) is the most aggressive primary brain tumor and remains largely incurable because of rapid recurrence and resistance to current therapies. A major challenge in GBM treatment is the persistence of glioma stem cells (GSCs), also known as glioma-initiating cells (GICs), which possess the ability to self-renew, regenerate tumors, and survive conventional therapy. Understanding the mechanisms that enable these cells to withstand therapeutic stress is essential for developing more effective treatments.

Our research focuses on the cellular quality control mechanisms that support GIC survival. We previously demonstrated that autophagy, a major intracellular recycling pathway, is not only required for the maintenance of GICs but is also activated by temozolomide (TMZ), the standard chemotherapy for GBM. Importantly, TMZ-induced autophagy promotes enrichment of the therapy-resistant GIC population, whereas inhibition of autophagy prevents this expansion, suggesting that autophagy is a key adaptive response underlying treatment resistance.

Building on these findings, the present work investigates whether mitochondrial quality control (MQC) acts as a central mechanism that coordinates the survival and metabolic adaptation of GICs. Using paired GBM and peri-tumor tissues, established GBM cell lines, and patient-derived spheroid models, we show that GIC-enriched tumors exhibit enhanced mitochondrial biogenesis, increased DRP1-mediated mitochondrial fission, and elevated autophagy/mitophagy. These mitochondrial adaptations are associated with higher mitochondrial membrane potential, increased ATP production, and controlled reactive oxygen species (ROS), enabling GICs to maintain mitochondrial fitness and survive under therapeutic stress.

We further explored the influence of metabolic reprogramming on GIC maintenance by studying gliomas carrying IDH1/2 mutations. Consistent with the favorable clinical outcome associated with these tumors, our findings demonstrate a significantly reduced GIC population in IDH-mutant gliomas compared with IDH-wild-type tumors, supporting the concept that altered cellular metabolism influences stem cell maintenance.

Overall, our findings support a unifying model in which mitochondrial quality control integrates mitochondrial dynamics, mitophagy, and cellular metabolism to preserve GIC function and promote therapeutic resistance. These results identify MQC as a potential therapeutic vulnerability and provide a conceptual framework for developing strategies that selectively target therapy-resistant GICs to improve GBM treatment.

Together, these findings open new therapeutic avenues: by targeting autophagy and exploiting metabolic vulnerabilities such as IDH1/2 mutations, it may be possible to deplete the GSC reservoir, overcome chemoresistance, and improve treatment outcomes for patients with GBM.

Keywords: Cancer stem cells, Glioblastoma, Autophagy, Chemotherapeutics

Biography:

Dr. Meenakshi Tiwari did post-graduation in Biochemistry from Jiwaji University and was awarded Gold Medal for securing first position in merit. She also received prestigious NET/LS/CSIR JRF and ICMR fellowship for pursuing Ph.D. After masters, Dr. Tiwari joined Ph.D. program at Sanjay Gandhi Post Graduate Institute of Medical Sciences, India, as CSIR JRF. During her Ph.D. she studied molecular mechanism of chemotherapeutic agents in glioblastoma cells and was awarded Gold Medal for best paper and excellence in research. Later, she joined as Postdoctoral fellow at UTHSCSA, San Antonio, Texas, USA (2007-2013). She joined All India Institute of Medical Sciences Patna in 2013 where held progressive faculty positions (Assistant Professor till Additional Professor) in Dept. of Biochemistry / Lab Medicine (2013–2024). She has joined as Professor in the Molecular Biology Lab in the Department of the Center for Advance Research at King George's Medical University. She is involved in various research projects with intramural and extramural funding from ICMR, DST, and DBT.

Research Interest: Genetics, Molecular Biomarkers, Therapeutics, Aging and associated disorders Cancer, Neurodegeneration.

SPEAKERS

Mitochondrial Quality Control in Triple-Negative Breast Cancer: From Pathogenesis to Therapeutic Opportunities

Dr. Lokendra Sharma
Additional Professor
Department of Molecular Medicine and Biotechnology
Sanjay Gandhi Post Graduate Institute of Medical Sciences (SGPGIMS) Rae Bareli Road Lucknow, (U.P.) 226014, India

Emerging evidence highlights mitochondrial reprogramming as a critical adaptive mechanism that enables cancer cells to withstand metabolic and therapeutic stress. Triple-negative breast cancer (TNBC), an aggressive and therapeutically refractory subtype, exhibits a disproportionately higher incidence in the Indian population. In this study, we examine the contribution of mitochondrial quality control processes—including biogenesis, fission–fusion dynamics, and mitophagy—to TNBC progression and therapy resistance.

Using patient-derived tumor samples, three-dimensional culture systems, and an in vivo TNBC mouse model, our results demonstrate that TNBC cells exhibit elevated mitochondrial turnover. Pharmacological or genetic inhibition of the mitochondrial fission regulator DRP1, as well as disruption of mitophagy, resulted in significant attenuation of tumor cell proliferation, invasion, and angiogenesis, accompanied by enhanced apoptotic cell death. Notably, combinatorial targeting of mitochondrial fission and autophagy pathways in vivo produced a marked reduction in tumor burden and metastatic dissemination.

Collectively, our findings establish mitochondrial dynamics as a central driver of TNBC aggressiveness. Targeting the functional interplay between mitochondrial fission and mitophagy may offer novel therapeutic opportunities and facilitate the identification of mitochondria-associated biomarkers for precision treatment of aggressive breast cancers.

Biography:

Dr. Lokendra Kumar Sharma is an Additional Professor in the Department of Molecular Medicine & Biotechnology at Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow, India. He has more than 15 years of academic and research experience, including advanced training in mitochondrial biology and translational cancer research. His work focuses on the role of mitochondrial alterations, oxidative stress, and quality control in cancer, mitochondrial disorders, maternal and reproductive health. He leads actively funded, interdisciplinary research programs supported by extramural and intramural grants, and maintains strong multi-institutional collaborations. In addition to his research, Dr. Sharma is involved in teaching and training postgraduate and PhD students, and contributes to clinical services through molecular and biochemical diagnostics, including HPV genotyping for cervical cancer screening.

SPEAKERS

Targeting Autophagy and PI3K-Mediated Metabolic Reprogramming in Breast Cancer Stem Cell-Enriched 3D Spheroids Using Alpha-Lipoic Acid

Dr. Bandana Chakravarti
Associate Professor
Stem cell/cell culture lab, Centre For Advance Research, King George's Medical University, Lucknow, U.P., India.

Breast cancer stem cells (BCSCs) constitute a highly tumorigenic subpopulation that contributes to therapeutic resistance, tumor progression, metastasis, and disease recurrence. The maintenance of BCSC stemness is driven by metabolic plasticity and aberrant activation of signaling pathways, including PI3K/Akt/mTOR and autophagy. Increasing evidence suggests that autophagy supports metabolic adaptation and survival of BCSCs under therapeutic stress, making it an attractive target for overcoming drug resistance. Alpha-lipoic acid (LA), a naturally occurring antioxidant and metabolic modulator, has emerged as a promising anticancer agent; however, its effects on autophagy-mediated metabolic reprogramming in BCSCs remain poorly understood. Breast cancer stem cell-enriched three-dimensional spheroids were generated from MCF-7 and MDA-MB-231 cell lines. The interaction of LA with phosphoinositide 3-kinase (PI3K) was investigated using molecular docking. Untargeted metabolomic profiling was performed to characterize LA-induced metabolic alterations. The effects of LA alone and in combination with doxorubicin (Dox) on spheroid growth were evaluated, while modulation of autophagy and PI3K/Akt/mTOR signaling was assessed using molecular analyses. Molecular docking demonstrated a favorable interaction between LA and PI3K, supporting its potential to inhibit PI3K-mediated signaling. Metabolomic analysis revealed profound metabolic reprogramming following LA treatment.

In MCF-7 spheroids, LA increased the abundance of 15 metabolites and reduced 5 metabolites, whereas MDA-MB-231 spheroids exhibited increased levels of 3 metabolites and decreased levels of 16 metabolites, indicating subtype-specific metabolic responses. LA significantly enhanced the antiproliferative activity of doxorubicin, demonstrating a synergistic effect on breast cancer spheroid growth. Mechanistically, LA suppressed PI3K/Akt/mTOR signaling and inhibited protective autophagy, resulting in impaired metabolic adaptation, reduced proliferation, and diminished BCSC survival. Alpha-lipoic acid effectively disrupts autophagy-dependent metabolic reprogramming in breast cancer stem cell-enriched 3D spheroids by targeting the PI3K/Akt/mTOR pathway. Its ability to enhance doxorubicin efficacy highlights its potential as a metabolic adjuvant for combination therapy aimed at overcoming breast cancer stem cell-mediated therapeutic resistance and improving treatment outcomes.

Biography:

Area of Research: Autophagy and Energy metabolism, Breast Cancer Stem Cells, Cardiovascular diseases/development and Metabolic diseases

SPEAKERS

Cancer has become an integral part of life with the future to live together

Arvind Kumar

School of Biotechnology, Institute of Science, BHU, Varanasi Uttar Pradesh, India-221005

The global cancer burden reported is approximately 20.6 million new cases and nearly 10 million deaths annually as the world's second leading death. Asia accounts half of the global cases. In Bihar incidence rate of cancer has been estimated to be the lowest in the country, it translates to a huge annual incidence is 110,448 new cases in 2022, due to Bihar known for 3rd most populous state in the country, with projected increase to more than 135,000 new cases annually by 2026. Lung and mouth cancer is more in men due to excessive use of tobacco. In women breast and cervical cancers are dominant due to lack of awareness. In most developed country such as United States, the American Cancer Society projected roughly 21 Lakhs new cases with 6 Lakhs deaths in 2026 with decrease in mortality rates due to universal health coverage, robust monitoring systems. Abnormality in the normal proliferation of cells due to mutation in anti-cancerous genes and proto- oncogenes leads to oncogenesis and development of cancer by the creation of micro-environment by the production of inflammatory cytokines, such as IL-1ß, IL-6, TNF-a, and novel molecule HMGB1. It is responsible for carcinomas (epithelial cells), sarcomas which is tumors of connective tissues, muscle, bone, cartilage and fibrous tissues and Leukaemia's/Lymphomas. Cancer leads to failure of normal signal transduction pathway leading to disruption in cell cycles that leads to uncontrolled cell division, cell proliferation, angiogenesis, invasion and spreading to normal tissues, normal organs becoming benign to malignant and later on metastatic. However, only malignant cancers are referred as cancer that has an ability to invade and metastasize after angiogenesis to make cancer dangerous increasing the level of fatality. So far too many genes responsible for the cancer development that have been reported but only surgical removal of benign tumors could be cured but once it's malignant, develops resistance against cancer drugs. Resistance towards drugs might be due to activation of MAP-kinase signal transduction pathway either mutation in downstream molecule constitutively activating different kinases. Currently we are looking for the role of HMGB1 in the activation of MAP-kinase signal transduction pathway leading to the genesis and progression of cancer and even reoccurrence of cancer after cure.

Key words: Cancer, mortality, health coverage, medication

Biography:

TEACHING RESPONSIBILITIES:

1. Taking classes in the M.Sc. previous and final years

2. Guiding M.Sc. final year students one year experimental project course

3. Guiding Ph.D. students and teaching their Ph.D. course work

4. Taught minor elective courses of other department M.Sc. students within the Institute of Science.

5. Took classes of B.Sc. in Faculty of Science, in Ancillary courses

AWARDS, FELLOWSHIPS AND DISTINCTIONS:

* Received National merit Scholarship (during Intermediate 11th & 12th & B.Sc. (H))

* UGC-Junior/Senior Research Fellowship award during Ph.D. in School of Biotechnology, Faculty of Science, BHU, Varanasi

* Post-doctoral Fellowship in NIH project at Baylor College of Medicine, Dept. of Neurosurgery Center for Cell and Gene Therapy & University of Pittsburgh

* Rashtriya Gaurav Award, New Delhi, 2012

* Received certificate of appreciation for helping in organizing 20th national children's science congress of DST the focal theme ENERGY: Explore, Harness and Conserve, held at BHU during 27-31 Dec, 2012.

RESEARCH & TEACHING EXPERIENCE:

* Research & Teaching EXPERIENCE including Ph.D. 31 Years

* Research & Teaching EXPERIENCE excluding Ph.D. & including Post. Doc. * 25 Years.

* Teaching EXPERIENCE in M.Sc. as Reader/Associate Professor/ Professor * 20 Years

SPEAKERS

Strategic Healthcare Vision of the Government of Uttar Pradesh: Towards a Disease-Free Uttar Pradesh by 2047

Dr. Amit Kumar Ghosh, IAS
Additional Chief Secretary, Medical Health, Family Welfare & Medical Education Department
Government of UP

The keynote address may focus on the Government of Uttar Pradesh's long-term healthcare strategy and its roadmap towards achieving the vision of Viksit Bharat 2047. The address may highlight the State's initiatives to strengthen public health systems, expand medical education, improve access to affordable and quality healthcare, and ensure equitable healthcare services for all citizens.

Key areas of discussion may include:

• The Government of Uttar Pradesh's strategic vision for strengthening healthcare infrastructure and medical education.

• Universal access to affordable, quality, and patient-centered healthcare services.

• Prevention, early detection, and effective management of communicable and non-communicable diseases, including cancer.

• Expansion of cancer screening, diagnosis, treatment facilities, and palliative care across the State.

• Leveraging digital health technologies, artificial intelligence, genomics, and telemedicine to improve healthcare delivery.

• Strengthening primary healthcare, maternal and child health services, and preventive healthcare.

• Capacity building of healthcare professionals through advanced medical education and research.

• Public-private partnerships to accelerate innovation and improve healthcare accessibility.

• Community participation, health awareness, and lifestyle interventions to reduce the burden of disease.

• A roadmap towards achieving a Disease-Free Uttar Pradesh by 2047, aligned with the national vision of Viksit Bharat 2047, through coordinated efforts in prevention, innovation, research, and universal healthcare.

This keynote will provide an opportunity to present the transformative healthcare initiatives undertaken by the Government of Uttar Pradesh and outline the future roadmap for creating a healthier, more resilient, and disease-free State by 2047.

Bridging the Gap between Clinicians and Researchers: Advancing Precision Cancer Care through Molecular Diagnostics

Dr. V. P. Singh
Savera Cancer Hospital, Department of Surgical Oncology, Kankarbagh Patna, Bihar

Cancer care is undergoing a paradigm shift from conventional anatomy- and stage-based treatment to precision oncology driven by molecular biology. The integration of genomic profiling, next-generation sequencing (NGS), liquid biopsy, and companion diagnostics has revolutionized cancer diagnosis, prognostication, and therapeutic decision-making. Biomarkers such as EGFR, HER2, KRAS, BRCA, MSI, and PD-L1 enable personalized treatment strategies, allowing clinicians to select targeted therapies and immunotherapy that maximize clinical benefit while minimizing unnecessary toxicity. The successful implementation of precision medicine relies on close collaboration between clinicians, pathologists, molecular scientists, and researchers, ensuring that laboratory discoveries are translated into improved patient outcomes. Emerging technologies including artificial intelligence, spatial genomics, and circulating tumor DNA (ctDNA) are further expanding the scope of individualized cancer care. This presentation highlights the evolving role of molecular diagnostics in modern oncology and emphasizes the importance of strengthening clinician–researcher partnerships to deliver evidence-based, patient-centered cancer care in the era of precision medicine.

Keyword: Precision oncology; Molecular diagnostics; Next-generation sequencing (NGS); Liquid biopsy; Biomarkers; Targeted therapy; Immunotherapy; Precision cancer care.

Biography: Dr. V. P. Singh is a Senior Surgical Oncologist & Managing Director at Savera Cancer Hospital, Patna, with over three decades of experience in the field of Surgical Oncology. He has been instrumental in advancing comprehensive cancer care in Bihar and is recognized for his contributions to Cancer Surgery and precision medicine. His academic interests include molecular diagnostics, hereditary cancer syndromes, translational oncology, and multidisciplinary cancer care. He has delivered numerous invited lectures at national scientific forums and has actively contributed to clinical research, medical education, and the promotion of personalized oncology practice. He remains committed to bridging the gap between clinical oncology and translational research to improve outcomes for cancer patients.

SPEAKERS

O DEJARDIN

Despite the frequency and lethality of oral cancers in France, there are no detailed general population data regarding the characteristics of these patients to fuel the public health authorities' reflections about early detection policies. Thus, the objective of this study was to determine, in the general population, the characteristics of both patients and tumours at the time of the diagnosis. A high-resolution, population-based study using 13 French registries was conducted on 1089 tumours diagnosed in 2010. Men accounted for 75% of cases. The most frequent sites were tonsil (28.4%) and oral tongue (21.1%). The median age varied from 56.7 years for floor of mouth to 66.4 years for gum.

The lesions were mainly diagnosed on pain and those diagnosed after routine clinical examination were scarce (2.6%). There were 65.5% stage III and IV at diagnosis. Oral tongue, floor of mouth and palate presented tumours less than 2cm only in 34 to 40% of cases. Advanced stage was associated with the presence of comorbidities, and tonsil or base of tongue topography. Stage was not associated with Département, deprivation index or gender. This study provided a picture of the characteristics of oral cancer patients and their tumours and showed that diagnoses are often made late, even for those tumours most easily accessible to direct visual and tactile examination. Nevertheless, it remains to define the target population of an early detection and to evaluate the benefit of such detection on the mortality rate.

Care Of Palliative Care In India

Dr Geeta Joshi, MD, DA, PGDHHM, THE PRESIDENT IAPC
Former, Chief Executive Officer
Community Oncology Center, Gujarat Cancer Society, Ahmedabad
Former Dy Director, Prof & Head,
Dept of Palliative Medicine, Gujarat Cancer & Research Institute, A'Bad

Despite its limited coverage, palliative care has been present in India for about 20 years. Obstacles in the growth of palliative care in India are too many and not only include factors like population density, poverty, geographical diversity, restrictive policies regarding opioid prescription, workforce development at base level, but also limited national palliative care policy and lack of institutional interest in palliative care. Nonetheless we have reasons to be proud in that we have overcome several hurdles and last two decades have seen palpable changes in the mindset of health care providers and policy makers with respect to need of palliative care in India. Systematic and continuous education for medical staff is mandatory, and a major break-through for achieving this purpose would be to increase the number of courses and faculties in palliative medicine at most universities.

Biography:

Publications & Presentations

She is National faculty for Pain and Palliative Care trainings & conferences since > 25 Years

60 research papers which includes Three book chapters & Co-author for a Book on Cancer

Posts held in various organisations

Director, International Institute of Distance Learning, NAPCAIM & President, Guj Chapter

The President 2025-28, Indian Association Palliative Care

Past Secretary & Past President, Indian Society for The Study of Pain

Past President, South Asian Regional Pain Society

Trustee & founder member of a NGO "UNNAT"

Awards & Achievements

She started Department of Palliative Medicine at Gujarat Cancer & Research Institute,

First in Gujarat, with Post graduation in the subject

Excellence Award 2016 : For Best talent to start New subject by Gujarat University

Received SAARC Country award for Excellence & leadership in Palliative Care

given by Cancer Aid Society for the year 2017-2018

* GCRI-GCS Luminary award for in Feb 2023

* Life Time Achievement Award from Indian Society for the Study of Pain in Feb 2025

SPEAKERS

Mohammad Rahbari

Steatotic liver diseases (SLDs), including metabolic dysfunction-associated steatotic liver disease (MASLD; formerly known as non-alcoholic fatty liver disease) and alcoholic liver disease, are the leading causes of chronic hepatitis, liver dysfunction, cirrhosis and liver cancer development. Severe manifestations of MASLD and alcoholic liver disease include metabolic dysfunction-associated steatohepatitis (MASH; formerly known as non-alcoholic steatohepatitis) and alcoholic steatohepatitis (ASH) or a combination thereof, termed metabolic dysfunction and alcohol-associated liver disease.

While MASH-associated and ASH-associated liver cancers display common histopathological features, the underlying cellular and molecular pathophysiological mechanisms of each are distinct. Recent studies indicate that SLDs encompass previously unrecognized spectra of heterogeneous, metabolic, immunological and genetic diseases that, in combination with lifestyle measures and individual patient comorbidities, affect disease pathogenesis and therapy response. Here, we review the current knowledge of the molecular, genetic and cellular mechanisms underlying the transition of MASH or ASH to liver cancer as well as novel developments in liver cancer risk assessment in SLDs. We further discuss possible obstacles of differential diagnosis and outline current developments in the therapeutic management of both MASH-related and ASH-related liver cancer.

Gunnar Myrdal

Objective: Sweden has one of the largest population-based cancer registers in the world that provides an opportunity to examine the trend of lung cancer incidence during a 35-year period. The primary aim of the present study was to estimate the effects of birth cohort, year of diagnosis (period), and age on the time trends of lung cancer incidence rates, and to analyze the gender-specific incidence of different histopathological types of lung cancer.

Results: Among men the age-standardized incidence rate increased steadily up to 1982, when a peak of 49 cases per 100,000 person-years was reached. Among women the incidence rate was lower and showed a monotonic increase throughout the observation period. The fastest rate of increase was noted among the youngest women.

In women, but not in men, there was a steady increase in risk with each successive birth cohort. For both sexes there were large changes in the histopathological distributions of cases. The most notable was a major increase in adenocarcinomas.

Conclusions: The overall age-adjusted incidence rate of lung cancer in Sweden has stabilized in men during the past two decades while rates are still increasing in women. In view of the continued high prevalence of smoking among young women, a future definite increase in the overall number of lung cancer cases in women can be expected.

Professor Carlos Caldas, FMedSci

Professor of Cancer Medicine
University of Cambridge, United Kingdom

Professor Carlos Caldas is a world-renowned British oncologist, physician-scientist, and internationally recognized leader in breast cancer research. As Professor of Cancer Medicine at the University of Cambridge and a senior investigator at the Cancer Research UK Cambridge Institute, he has made pioneering contributions to the understanding of breast cancer genomics and precision oncology.

His groundbreaking research has transformed the molecular classification of breast cancer, enabling more personalized diagnostic and treatment strategies that have influenced clinical practice worldwide. Professor Caldas has authored hundreds of peer-reviewed scientific publications in leading medical journals and has received numerous international awards for his outstanding contributions to cancer research.

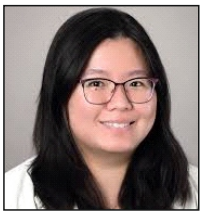
At the 7th Global Cancer Summit, Professor Caldas will present a scientific abstract highlighting the latest advances in breast cancer research, precision medicine, and translational oncology. His presentation will provide valuable insights into emerging technologies, innovative therapeutic approaches, and the future of personalized cancer care.

His participation reflects the summit's commitment to bringing together globally acclaimed experts to advance cancer research, foster international collaboration, and improve patient outcomes.

Areas of Expertise
Breast Cancer Genomics
Precision Oncology
Molecular Classification of Breast Cancer
Translational Cancer Research
Biomarker Discovery
Personalized Cancer Therapy

SPEAKERS

Szu-Ting Yang



A significant decline in both incidence and prevalence of cervical cancers after widespread-introducing cervical screening strategy by Papanicolau test (Pap test) has been found in the world, but cervical cancer is still one of the most common female cancers, reporting the fourth prevalence and also one of the leading causes to result in main women-associated morbidity and mortality, particularly for those women living in low- and middle-income countries. Cervical cancer is one of the most important health concerns directly destroying the global health-care system, partly because of not only increasing the disability either secondary to diseases themselves of victims or mediated by treatment-related adverse events to the survivors but also acting as a leading cause of death of diseased patients worldwide, alarming the urgent need to do something to minimize the catastrophic diseases-related heavy socioeconomic burden.

It is fortunate that cervical cancer is a preventable disease, based on its strong association with human papillomavirus (HPV) infection (more than 95%), particularly for those high-risk HPV (HR-HPV) and its high possibility by detecting HPV infection before the development of cervical cancer as well as an effective prevention by HPV vaccination. That is why WHO (World Health Organization) considers cervical cancer as a public problem and attempts to accelerate the elimination of cervical cancer program by three-pillar approach (90:70:90% targets), including (1) 90% of girls are fully vaccinated with HPV vaccine by 15 years of age; (2) 70% of women are screened with a high-performance test by 35 and 45 years of age and precancerous lesions are treated early; and (3) 90% of women identified with cervical diseases receive appropriate and adequate treatment. Herein, this review focuses on the HPV vaccination as Part I, including global recommendations and Taiwan government's policy for HPV vaccination.

Bettia Celestin

Hypoxic pulmonary hypertension (HPH) is associated with pulmonary artery (PA) remodeling and right ventricular (RV) overload. We have previously uncovered collagen-mediated mechanisms of proximal PA stiffening in early HPH by manipulating collagen degradation and cross-linking using a transgenic mouse strain and a potent collagen cross-link inhibitor, β-aminopropionitrile (BAPN). However, the roles of collagen in distal PA remodeling, overall RV afterload, and RV hypertrophy in HPH remain unknown. Here, we used the same experimental strategy to investigate the effect of pulmonary vascular collagen content and cross-linking on steady and pulsatile RV afterload and on RV hypertrophy in early HPH. Collagenase-resistant mice (Col1a1R/R) and their littermate controls (Col1a1+/+) were exposed to normobaric hypoxia for 10 days with or without BAPN treatment. In vivo pulmonary vascular impedance, a comprehensive measure of RV afterload, was measured via simultaneous RV catheterization and echocardiography. Morphology and collagen accumulation were examined using histological techniques and ELISA in lungs and RVs. In both mouse strains, BAPN did not limit increases in pulmonary arterial pressure or pulmonary vascular resistance, indicating a negligible effect of either collagen content or cross-linking on steady RV afterload. However, BAPN prevented the increase in pulse pressure and RV hypertrophy in Col1a1+/+ mice and these effects were absent in Col1a1R/R mice, suggesting a role for PA collagen content, not cross-linking, in the pulsatile RV afterload. Moreover, we found a significant correlation between pulse pressure and RV hypertrophy, indicating an important role for pulsatile RV afterload in RV overload in early HPH.

Biography:

Bettia Celestin, MD, MSc, is a French board-certified cardiologist and postdoctoral scholar in the Department of Pathology at Stanford University School of Medicine. Her research focuses on echocardiographic imaging, right heart assessment, pulmonary hypertension, and women's cardiovascular health, with growing interest in sex-specific echocardiographic phenotyping, adverse pregnancy outcomes and cardiovascular risk across the menopausal transition, and AI-enhanced cardiovascular imaging.

Dr. Celestin serves as part of the co-investigator team in the clinical core on a NIH P01 on immune aging, where she leads cardiovascular phenotyping for a deeply characterized immune aging cohort. Her work on AI-based echocardiography implementation in pulmonary arterial hypertension was published in CHEST (2025) and featured on the CHEST Journal Podcast. She has multiple peer-reviewed publications in journals including CHEST, Pulmonary Circulation, Circulation: Cardiovascular Imaging, JAHA, and JACC: Heart Failure, and has presented at national and international conferences including ISHLT and AHA.

Dr. Celestin holds an MD from Sorbonne Paris University and an MSc in Biostatistics from Paris-Saclay University. She completed her cardiology training in France with over 10 years of clinical experience and an expertise in echocardiography. She is an active member of the AHA.

SPEAKERS

DIKSHA RAWAT

Business Development, Sales & Marketing Specialist | CRO, Lifesciences, Bengaluru, India

Background : Cervical Cancer (CC) is a vaccine-preventable disease, and it is treatable if diagnosed early and managed properly. However, it is the fourth most common cancer in women worldwide with about 604,127 cases and 341,831 deaths in 2020. In Italy, it represents the fifth most common cancer in women under 50 years of age with about 2,400 new cases in 2020. The CC elimination is today a global public health goal published by the World Health Organization (WHO) in 2020 and a commitment of the European Union that has included it in Europe's Beating Cancer Plan. Therefore, urgent action is needed, at international and national level, to implement value-based interventions regarding vaccination, screening and timely management of the disease. Our study aims to describe the state of the art of Human Papilloma Virus (HPV) prevention in Italy and to get a consensus on indicators for monitoring the progress toward CC elimination at national level.

Biography:

SKILLS

• Lead Generation & Qualification Client Engagement & Retention

• Market Research

Competitor Landscaping Negotiation

RFP/RFI/Feasibility Mgmt Campaign Management CRM Management

Cross-functional Collaboration Outbound Marketing (Email, Linkedin)

TRAININGS & CERTIFICATION

NIH Clinical Research

• Linkedin Marketing

• HubSpot Inbound Marketing

Dr. Ryan Edbert Husni, MD, PhD

Regional Medical Director, Oncology — East Asia & Global South
Eisai Co., Ltd., Japan

Dr. Ryan Edbert Husni is a clinician-scientist and senior medical affairs leader with extensive experience in oncology drug development, medical strategy, and cross-regional healthcare systems across Asia.

He currently serves as Regional Medical Director for Oncology covering East Asia and the Global South at Eisai Co., Ltd. In this role, Dr. Husni provides strategic medical oversight across multiple markets, drives evidence generation initiatives, and leads scientific engagement with key stakeholders across diverse healthcare landscapes.

Dr. Husni holds a Doctor of Medicine degree and a PhD in Medical Sciences from the University of Tsukuba, Japan. His research and clinical interests focus on epigenetics of lung cancer.

Dr. Amit Nandan Dhar Dwivedi

Md (radiodiagnosis & Imaging)
Professor: Dept Of Radiodiagnosis & Imaging. Banaras Hindu University

Interventional vascular radiology is a rapidly expanding field, but can broadly be split into embolization and stent-grafting techniques. Frequently these can be performed percutaneously with local anaesthetic infiltration and do not require the involvement of an anaesthetist. Some procedures, particularly endovascular aneurysm repair (EVAR) require surgical access to the vasculature, necessitating anaesthesia. Solid organ embolization can be painful and patients for haemorrhage control may also require our input to manage ongoing resuscitation and stabilization. Each procedure is different and communication and team working is essential to understand the planned procedure and the requirements for anaesthesia and operating conditions.

This article will discuss the different procedures performed (excluding neurological and cardiological interventions) with a focus on EVAR, and their anaesthetic implications.

SPEAKERS

Harnessing Microbial Metabolites For Multi-target Cancer Therapy: From Replication Arrest To Apoptosis Induction

Dr. J. Angayarkanni
Professor Cancer therapeutics laboratory
Department of Microbial Biotechnology Bharathiar University Coimbatore 641046

Cancer remains a multifactorial disease characterized by uncontrolled proliferation, angiogenesis, metastasis, and evasion of apoptosis. Conventional chemotherapeutics often suffer from non-specific cytotoxicity and resistance, necessitating exploration of novel, biologically derived alternatives. This study reviews the therapeutic potential of microbial metabolites and proteins as multi-targeted anticancer agents. Replication inhibition was achieved through nucleotide pool depletion using pterin deaminase and Topoisomerase II inhibition via GidA protein coupled with ruthenium complexes. Anti-angiogenic activity was explored through microbial peptide-mediated inhibition of Angiotensin Converting Enzyme, thereby curbing tumor vascularization. Anti-metastatic potential was demonstrated by matrix metalloprotease inhibition using lupeol and Nm23 protein, restricting invasion and secondary tumor establishment. To eliminate cellular immortality, microbial metabolites were employed to inhibit telomerase activity, a hallmark enzyme sustaining unlimited replicative potential in cancer cells. Apoptotic induction was achieved using pterin derivatives, fungal-derived lovastatin analogues, and koninginin, alongside modulation of stress-response proteins such as heme oxygenase, which exhibits dual pro- and anti-apoptotic roles depending on cellular context, the latter counteracted using synthetic imidazole compounds. Additionally, bacterioferritin was highlighted as a promising theranostic biomarker enabling targeted diagnostic and therapeutic delivery. Palliative applications were also considered, with GABA-producing probiotics proposed for psychobiotic intervention to enhance patient quality of life. Collectively, these findings underscore the pharmacological versatility of microbial-derived compounds in disrupting multiple oncogenic hallmarks simultaneously. This integrative, microbiome-based approach offers a promising, low-toxicity adjunct or alternative to conventional oncotherapy, warranting further preclinical and clinical validation.

Biography: Dr. J. Angayarkanni with her UG degree in Biochemistry has started her career as Technical Officer in the Department of Biotechnology, Bharathiar University, Coimbatore in the year 1990. She continued her PG degree in Environmental Sciences and completed her Ph.D. in Biotechnology in the year 2002. Her topic of research for her doctoral degree is fungal enzymes,viz. xylanase, cellulose and pectinase for industrial application. She entered the teaching profession in the year 2005 as Assistant Professor in the same department. Since the inception of new department of Microbial Biotechnology in 2008, she is working in Department of Microbial Biotechnology.

Dr. Vijay Kumar C.

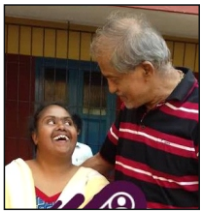
MBBS, MS, DNB, FELLOWSHIP UROGYNECOLOGY

Urogynaecology is a subspecialty that manages urinary incontinence and pelvic floor disorders in women. Management options vary, and are often dependent on the patient's choice of treatment and the outcome desired. Minimal access surgery is becoming increasingly popular in the field of urogynaecology for patients opting for surgical treatment.

Laparoscopic surgery offers the benefit of shorter hospital stays, reduction in post-operative pain, and similar results to those achieved through the open approach. Despite this, laparoscopic surgery presents its own challenges and complications specific to this surgical approach. This review article contains a summary of the relevant surgical anatomy for laparoscopic urogynaecology, theatre set-up and patient selection, the surgical treatment options for common pelvic floor disorders, and challenges in training and service provision.

Dr Phillip Mascarenhas

Divine Medical Center Goa



The goal of palliative care is to relieve the suffering of patients and their families by the comprehensive assessment and treatment of physical, psychosocial, and spiritual symptoms experienced by patients. As death approaches, a patient's symptoms may require more aggressive palliation. As comfort measures intensify, so should the support provided to the dying patient's family. After the patient's death, palliative care focuses primarily on bereavement and support of the family.

Biography: Dr. Phillip Mascarenhas is a highly qualified and experienced medical examiner with an M.B.B.S degree, dedicated to providing exceptional healthcare services. Operating from Divine Medical Centre, located at Door No. S1, S2, S5, & S7, 2nd Floor, Kamat Medical, Martires Dias Road, Near Hari Mandir, Salcete, Margao, South Goa, Goa- 403601, the clinic is a trusted medical facility for seafarers.

SPEAKERS

ASFAR AZMI, PHD

Department of Oncology

IMPORTANCE

Pancreatic ductal adenocarcinoma (PDAC) is a relatively uncommon cancer, with approximately 60 430 new diagnoses expected in 2021 in the US. The incidence of PDAC is increasing by 0.5% to 1.0% per year, and it is projected to become the second-leading cause of cancer-related mortality by 2030.

OBSERVATIONS

Effective screening is not available for PDAC, and most patients present with locally advanced (30%–35%) or metastatic (50%–55%) disease at diagnosis. A multidisciplinary management approach is recommended. Localized pancreas cancer includes resectable, borderline resectable (localized and involving major vascular structures), and locally advanced (unresectable) disease based on the degree of arterial and venous involvement by tumor, typically of the superior mesenteric vessels. For patients with resectable disease at presentation (10%–15%), surgery followed by adjuvant chemotherapy with FOLFIRINOX (fluorouracil, irinotecan, leucovorin, oxaliplatin) represents a standard therapeutic approach with an anticipated median overall survival of 54.4 months, compared with 35 months for single-agent gemcitabine (stratified hazard ratio for death, 0.64 [95% CI, 0.48–0.86]; P = .003). Neoadjuvant systemic therapy with or without radiation followed by evaluation for surgery is an accepted treatment approach for resectable and borderline resectable disease. For patients with locally advanced and unresectable disease due to extensive vascular involvement, systemic therapy followed by radiation is an option for definitive locoregional disease control. For patients with advanced (locally advanced and metastatic) PDAC, multiagent chemotherapy regimens, including FOLFIRINOX, gemcitabine/nab-paclitaxel, and nanoliposomal irinotecan/fluorouracil, all have a survival benefit of 2 to 6 months compared with a single-agent gemcitabine. For the 5% to 7% of patients with a BRCA pathogenic germline variant and metastatic PDAC, olaparib, a poly (adenosine diphosphate [ADB]-ribose) polymerase inhibitor, is a maintenance option that improves progression-free survival following initial platinum-based therapy.

CONCLUSIONS AND RELEVANCE

Approximately 60 000 new cases of PDAC are diagnosed per year, and approximately 50% of patients have advanced disease at diagnosis. The incidence of PDAC is increasing. Currently available cytotoxic therapies for advanced disease are modestly effective. For all patients, multidisciplinary management, comprehensive germline testing, and integrated supportive care are recommended.

Biography:

Dr. Asfar Azmi is a Professor of Oncology at Wayne State University School of Medicine. He is the Director of Pancreas Cancer Research at the NCI designated Comprehensive Cancer Center, Karmanos Cancer Institute in Detroit Michigan. He served Leader of Molecular Therapeutics and Co-Leader of Tumor Biology and Microenvironment Research Programs. He has a record of accomplishment in the area of drug discovery research. Dr. Azmi made fundamental discoveries on the role of aberrant nuclear protein transport in pancreatic cancer development and drug resistance. He has made significant contributions on pre-clinical and early phase development of new drugs for pancreatic cancer and pancreatic neuroendocrine tumors particularly, nuclear export inhibitor selinexor and KRAS pathway targeted therapies. Work done by his team led to the FDA approval of selinexor in several tumor indications. His lab uses high throughput technologies such as RNA-seq, sn-RNA-seq, spatial transcriptomics in patient derived tumors and genetically engineered mouse models to identify epigenetic and regulatory mechanisms of XPO1 and design novel drug combinations. Dr. Azmi's group is also developing novel drugs against mutant KRAS pathway proteins. His team has discovered the role of Rho GTPase effector protein p21-activated kinase 4 (PAK4) and nicotinamide adenine dinucleotide (NAD) biosynthesis pathway rate limiting enzyme nicotinamide phosphoribosyltransferase (NAMPT) in promoting tumor growth, desmoplasia and drug resistance in cellular and patient derived tumor models. His ongoing projects are focused on developing ways to favorably alter the pancreatic tumor microenvironment, making them more accessible to chemotherapeutic drugs. His lab is also exploring how targeting KRAS pathway proteins and NAD signaling can enhance immunotherapy strategies in therapy resistant cancers. Dr. Azmi has published more than 150 peer reviewed articles in prestigious journals such as Gastroenterology, Oncogene, Clinical Cancer Research, Journal of Biological Chemistry, Molecular Cancer Therapeutics, Nature Reviews Clinical Oncology and Seminars in Cancer Biology. He is the editor of several books on drug discovery topics. Dr. Azmi is the recipient of NIH MERIT award, and young investigator awards from American Pancreatic Association, American Association for Cancer Research (AACR) and American Society of Hematology (ASH). He is the recipient of Academy of Scholars Award from Wayne State University, Kales Endowed Faculty Award for Innovative Cancer Research and Heroes of Cancer Award from Karmanos Cancer Institute. He has mentored a number of students, residents, fellows and junior faculty. He serves as a mentor on training grants that his students have secured from DOD or NIH. His lab is continuously funded by NIH and pharma industry.

SPEAKERS

Knowledge, Attitudes and Clinical Practices Towards Sexual Health Among Oncology Specialists: A National Cross-Sectional Survey from India

Aman Chaudhary[1], Raja Pramanik[2]

[1] Department of Medical Oncology, Bharati Hospital and Research Centre, Bharati Vidyapeeth Deemed University Medical College, Pune, Maharashtra, India

[2] Department of Medical Oncology Dr B.R.A. IRCH AIIMS, New Delhi India

Background: Sexual dysfunction is a common yet under-recognized consequence of cancer and its treatment that significantly impairs patients' quality of life. National data from India regarding oncology specialists' knowledge, attitudes and clinical practices towards sexual health are scarce.

Methods: A national cross-sectional survey was conducted between January 2021 and June 2022 using a 32-item questionnaire adapted, with permission, from a previously published international survey. The electronic questionnaire (Google Forms) was distributed to oncology specialists across India, including medical oncologists, radiation oncologists, surgical oncologists, gynaecologic oncologists, urologists and oncology trainees. Statistical analyses were performed using STATA version 13, with $p<0.05$ considered statistically significant.

Results: Of approximately 1,200 oncology specialists approached, 337 completed the survey (response rate 28%). The median age was 35 years (range 24–69 years). Participants included medical oncologists (66%), radiation oncologists (16%), surgical oncologists (7%), oncology trainees (9.5%), gynaecologic oncologists (1%) and urologists (0.5%). Although 243/337 (72.1%) considered discussing sexual health their professional responsibility, 44% discussed sexual function with fewer than half of their patients and 29% rarely or never addressed the topic. The most common barriers were perceived referral of responsibility to another healthcare professional (73.4%), discomfort initiating discussions (68.2%), age difference between specialist and patient (65.7%), opposite-gender interactions (53.2%) and embarrassment (52.6%). Most respondents desired additional education (313/337, 92.9%), while 308/337 (91.4%) believed current oncology training inadequately addresses sexual health counselling. Specialists with ≤5 years of practice ($p=0.015$) and those specializing in breast cancer ($p=0.039$) discussed sexual health more frequently with new patients.

Conclusion: Despite recognizing the importance of sexual health, oncology specialists infrequently discuss treatment-related sexual dysfunction with patients. Incorporating structured education and communication training into oncology curricula may improve comprehensive cancer survivorship care.

Biography:

• Senior Resident (Non-academic, Department of Hematology/Medical Oncology, All India Institute of Medical Sciences (AIIMS), Rishikesh (July 2018 – July 2019).

• Senior Resident (Non-academic , Department of Medical Oncology , All India Institute of Medical Sciences (AIIMS), New Delhi (August 2019 – July 2020)

• I am currently working as Assistant Professor in the Department of Medical Oncology at Bharati Hospital and Research Center Pune, Maharashtra (Joined in August 2023).

Awards:

Gold medal in Gynecology and Obstetrics MBBS final year.

Best library user award for academic year 2021-22 AIIMS New Delhi.

DR Roshni Syam

Associate Professor at Regional Cancer Centre Thiruvananthapuram

The higher prevalence and incidents of chronic and life-threatening illnesses and the aging population living with morbidity and life-limiting conditions necessitate the need for care provisions like palliative care globally. Palliative care strives for the quality of life of patients with terminal illnesses and serious health conditions and their families. However, globally, access to palliative care remains very limited. The situation is similar in India, except for Kerala, where palliative care access to the needy population is almost universal. Kerala's community palliative care model is globally acclaimed for its operation, which includes community participation and sustainability. The palliative care policy of Kerala, which was the first one in Asia, was a significant milestone in the palliative care movement of Kerala, as it mandated the interventions from government measures and further strengthened the community-managed palliative care interventions. The palliative care efforts in Kerala have significantly influenced the health scenario. However, discussion on the role of palliative care in achieving sustainable development goals (SDGs) is minimal.

In this context, this article explores the policy and practices of palliative care in Kerala and its contribution to SDG-3, health and well-being. We have surrounded the discussion on the context of palliative care interventions and Sustainable Development Goal 3 through the unique features of the Kerala model of palliative care and its contribution to the healthcare scenario of the state. Through available literature and from the researchers' first-hand experience, this article explores the reciprocity of palliative care interventions, policy, healthcare programs, and SDG-3. Documenting the potential of Kerala's community-based palliative care for SDG-3 has implications for replications of this model in similar contexts.

Biography: Dr. Roshni Shyam is an medical professional and academic serving as faculty at the Regional Cancer Centre (RCC) (likely in Thiruvananthapuram, Kerala) and Convenor of the Health Knowledge Lab, contributing to public health education, cancer awareness, and community medical outreach programs

SPEAKERS

Dr. G K Rath

Head, National Cancer Institute (India), Chief Radiologist & Professor & Head: Department of Radiation Oncology
Dr BRA Institute Rotary Cancer Hospital, AIIMS, New Delhi

In recent years remarkable progress has been made towards the understanding of proposed hallmarks of cancer development and treatment. However with its increasing incidence, the clinical management of cancer continues to be a challenge for the 21st century. Treatment modalities comprise of radiation therapy, surgery, chemotherapy, immunotherapy and hormonal therapy. Radiation therapy remains an important component of cancer treatment with approximately 50% of all cancer patients receiving radiation therapy during their course of illness; it contributes towards 40% of curative treatment for cancer. The main goal of radiation therapy is to deprive cancer cells of their multiplication (cell division) potential. Celebrating a century of advances since Marie Curie won her second Nobel Prize for her research into radium, 2011 has been designated the Year of Radiation therapy in the UK. Over the last 100 years, ongoing advances in the techniques of radiation treatment and progress made in understanding the biology of cancer cell responses to radiation will endeavor to increase the survival and reduce treatment side effects for cancer patients. In this review, principles, application and advances in radiation therapy with their biological end points are discussed.

Keywords: Cancer, Radiation therapy, Linear energy transfer, Cell death.

Introduction

Cancer remains leading cause of death globally. The International Agency for Research on Cancer (IARC) recently estimated that 7.6 million deaths worldwide were due to cancer with 12.7 million new cases per year being reported worldwide. A significant proportion of this burden is borne by developing countries; 63% of cancer deaths are reported to be from developing countries 1, 2, 3. Cancer is a multigenic and multicellular disease that can arise from all cell types and organs with a multi-factorial etiology. Hanahan and Weinberg 4 have identified six cancer cell phenotypes or hallmarks of cancer: cells with unlimited proliferative potential, environmental independence for growth, evasion of apoptosis, angiogenesis, invasion and metastasis to different parts of body. If uncontrolled cell growth or metastatic spread occurs it will result in death of the individual 5. The past decade has witnessed a considerable progress towards the treatment and understanding of the earlier proposed hallmarks of cancer 6 and together with advances in early detection and in the various treatment modalities, many cancers have become curable 7.

After the discovery of X-rays in 1895, by Wilhelm Conrad Röntgen from Germany its clinical usefulness, as a means of cancer treatment was first appreciated. It is also one hundred years ago that Marie Curie won a second Nobel Prize for her research into radium, establishing her position as a pioneer in the field of radiation therapy. To mark this, 2011 has been designated the Year of Radiation therapy in the UK, celebrating a century of advances. Since that time, radiation therapy has developed into a recognized medical specialty with Radiation Oncology being a discipline in which various health and science professionals from numerous disciplines work together. Along with surgery and chemotherapy, radiation therapy or radiotherapy remains an important modality used in cancer treatment being a highly cost effective single modality treatment accounting about only 5% of the total cost of cancer care 8. Furthermore, approximately 50% of all cancer patients will receive radiation therapy during their course of illness 9, 10 with an estimation that radiation therapy contributes to around 40% towards curative treatment 11. Rapid progress in this field continues to be boosted by advances in imaging techniques, computerized treatment planning systems, radiation treatment machines (with improved X-ray production and treatment delivery) as well as improved understanding of the radiobiology of radiation therapy 12.

Principles of radiation therapy

Radiation is a physical agent, which is used to destroy cancer cells. The radiation used is called ionizing radiation because it forms ions (electrically charged particles) and deposits energy in the cells of the tissues it passes through. This deposited energy can kill cancer cells or cause genetic changes resulting in cancer cell death.

High-energy radiation damages genetic material (deoxyribonucleic acid, DNA) of cells and thus blocking their ability to divide and proliferate further 13. Although radiation damages both normal cells as well as cancer cells, the goal of radiation therapy is to maximize the radiation dose to abnormal cancer cells while minimizing exposure to normal cells, which is adjacent to cancer cells or in the path of radiation. Normal cells usually can repair themselves at a faster rate and retain its normal function status than the cancer cells. Cancer cells in general are not as efficient as normal cells in repairing the damage caused by radiation treatment resulting in differential cancer cell killing 10.

Radiation can be given with the intent of cure as well as being used as a very effective modality of palliative treatment to relieve patients from symptoms caused by the cancer. Further indications of radiation therapy include combination strategies with other treatment modalities such as surgery, chemotherapy or immunotherapy. If used before surgery (neoadjuvant therapy), radiation will aim to shrink the tumor. If used after surgery (adjuvant therapy), radiation will destroy microscopic tumor cells that may have been left behind. It is well known that tumors differ in their sensitivity to radiation treatment. Table 1 shows a list of common cancers treated with radiation therapy.

Biography:

Dr. G. K. Rath is a renowned cancer specialist and one of India's leading experts in radiation oncology. He serves as the Head of the National Cancer Institute (India) and is the Chief Radiologist, Professor, and Head of the Department of Radiation Oncology at the Dr. B. R. A. Institute Rotary Cancer Hospital, All India Institute of Medical Sciences (AIIMS), New Delhi.

With a distinguished career spanning several decades, Dr. Rath has contributed significantly to advancing cancer treatment, radiotherapy technologies, and comprehensive cancer care in India. He has been instrumental in establishing national cancer programs, improving access to advanced therapies, and driving innovations in clinical practices.

Dr. Rath is widely respected for his academic leadership and has trained numerous oncologists across the country. His extensive research, publications, and international collaborations have strengthened India's position in global oncology. He continues to champion patient-centered care, public health initiatives, and the expansion of high-quality cancer services nationwide.

SPEAKERS

Prof (Dr) C M Singh

Former Professor & Head (Community & Family Medicine)

Community participation is one of the founding pillars of primary health care. However, due to various reasons, we are yet to achieve complete integration of this component into the health system functioning in India. The objective of our study was to do a formative assessment of community participation in a rural healthcare setting by adopting participatory learning action (PLA). technique. The study participants included frontline health workers and members from local governing institutions of rural areas. The study design is qualitative in nature with a participatory approach. A number of three PLA techniques have been used as a part of this study to recognize available resources for community participation, address its barriers and facilitators, and finally devise a time-line-based action plan. Based on the this, a conceptual framework for community participation pertaining to the rural healthcare system has been developed.

This study highlights the importance of understanding the psychosocial aspects of community participation among various stakeholders involved in rural health care. Lessons learned from this PLA study will be helpful in the integration of community-based participatory approach within grassroot level healthcare planning and service delivery.

Biography:

• Current Role: Director, Dr. Ram Manohar Lohia Institute of Medical Sciences, Lucknow, Uttar Pradesh.

• Past Role: Professor & Head, Department of Community & Family Medicine / Medical Superintendent, All India Institute of Medical Sciences (AIIMS), Patna.

• Specialization: Community Medicine, Public Health, Preventive Medicine, and Hospital Administration.

Safety of Capmatinib in MET Exon 14 Skipping Mutation–Positive metastatic NSCLC: Findings from a Prospective Multicenter Study in India

Dr Akhil Kapoor

This phase IV prospective, multicenter, open-label, single-arm study evaluated Capmatinib safety in 50 adult Indian patients with MET exon 14 skipping mutation-positive metastatic non-small cell Lung carcinoma. Capmatinib 400 mg twice daily was planned for 8 cycles of 21 days each over 24 weeks. Primary endpoints were safety, measured by incidence of adverse events (AEs/treatment-emergent AEs), including serious adverse events (SAEs), laboratory values, vital signs, and ECGs, and tolerability, measured by dose discontinuation, interruption, and reduction with reasons. The primary analysis included all patients receiving at least one dose during the on-treatment period, from first dose to 30 days after the last dose.

Mean age was 65.7 years (SD 8.96); 33 patients (66.0%) were males. Histology showed adenocarcinoma in 45 (90.0%) and squamous-cell carcinoma in 5 (10.0%). Median (IQR) drug exposure was 175 (53) days. TEAEs and SAEs occurred in 44 (88.0%) and 14 (28.0%) patients respectively. Grade ≤2 events occurred in 33 (66.0%) and Grade ≥3 in 17 (34.0%). There were 336 events, including 7 fatal events. Most frequent TEAEs were peripheral oedema (21, 42.0%), vomiting (13, 26.0%), hypo-albuminemia (9, 18.0%), and hypo-calcaemia (9, 18.0%). Vital signs and ECG remained stable. Study-drug-related AEs occurred in 30 (60.0%) patients, with 91 events, nonfatal. Overall, 14 (28.0%) patients had dose modification; interruptions and discontinuation occurred in 6 (12.0%) and 9 (18.0%), most commonly due to adverse events in 7 patients (14.0%).

This study provides supportive evidence for capmatinib's safety in Indian patients with metastatic NSCLC, consistent with global data from the GEOMETRY mono-1 study. These results support its use with appropriate monitoring in routine clinical practice.

Keywords:

Capmatinib; MET exon 14 skipping; NSCLC; safety; tolerability.

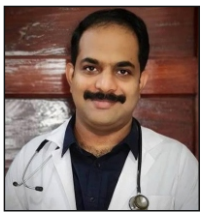
Biography:

Mahamana Pandit Madan Mohan Malviya Cancer Centre, Varanasi[1], Regional Cancer Centre, Thiruvananthapuram[2], MNJ Institute of Oncology & Regional Cancer Centre, Hyderabad[3], AIIMS, New Delhi, Delhi[4], Tata Memorial Hospital, Mumbai[5], Malabar Cancer Centre, Thalassery[6], MOC Cancer Care & Research Centre (Unit of Cellcure Cancer Centre Pvt. Ltd.), Mumbai[7], IPGME & R and SSKM Hospital, Kolkata[8], National Cancer Institute (NCI), Nagpur[9], Father Muller Medical College Hospital, Mangalore10, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry11, Jawaharlal Nehru Cancer Hospital And Research Centre, Bhopal12, Sparsh Hospitals & Critical Care (P) Ltd., Bhubaneswar13, CIMET's Inamdar Multispeciality Hospital, Pune14, Chittaranjan National Cancer Institute, Kolkata[1]5, VMMC & Safdarjung Hospital, Delhi[1]6, Aadhar Health Institute, Haryana[1]7, Bhagwan Mahaveer Cancer Hospital & Research Centre, Jaipur[1]8, All India Institute of Medical Sciences

SPEAKERS

Lijeesh.A.L

Associate Professor and oncological specialist in the Department of Radiation Oncology



In recent years remarkable progress has been made towards the understanding of proposed hallmarks of cancer development and treatment. However with its increasing incidence, the clinical management of cancer continues to be a challenge for the 21st century. Treatment modalities comprise of radiation therapy, surgery, chemotherapy, immunotherapy and hormonal therapy. Radiation therapy remains an important component of cancer treatment with approximately 50% of all cancer patients receiving radiation therapy during their course of illness; it contributes towards 40% of curative treatment for cancer.

The main goal of radiation therapy is to deprive cancer cells of their multiplication (cell division) potential. Celebrating a century of advances since Marie Curie won her second Nobel Prize for her research into radium, 2011 has been designated the Year of Radiation therapy in the UK. Over the last 100 years, ongoing advances in the techniques of radiation treatment and progress made in understanding the biology of cancer cell responses to radiation will endeavor to increase the survival and reduce treatment side effects for cancer patients. In this review, principles, application and advances in radiation therapy with their biological end points are discussed.

Dr. J K Singh

Padmashree Awardee
Oncologist, Patna

Medical Science is fastly growing Science, constantly developing Science & rapidly changing science. What is today tomorrow it is going to be obsolete.We have to remain on own toes otherwise we will be called outdated.

We are moving from cellular to Molecular era. In the path of research there are several milestones, each and every milestone gives us some new techniques, new ideas for the better management of Cancer & which contributes everytime something new in the scientific world.

Last few decades have seen tremendous technological explosion in the cancer management. In the molecular era many things have been changing. There have been many refinements in cancer surgery. With a better understanding of tumor biology & it its behaviors including the development of enhance technology it is now possible to treat even deep seated tumor even in the vital areas with lesser toxicities to surrounding tissues, which was not possible in earlier days. Since the 1st entrance of chemotherapeutic drugs, in late eighties, a broad base of experience in chemotherapy has also been developed. The dramatic entrance of targeted therapy.

Immunotherapy, Personalized treatment, CART cell therapy & Vaccine therapy has made the entire treatment very much different with very effectiveness even in the advanced stage of the disease. The past few decades have been constant progress in our knowledge of the disease, the effectiveness of the treatment & the proper choice of treatment for individual Cancer patient.

But In spite of tremendous advancement in the field of Oncology there are still some Grey areas which needs to be concurred, there are still some problems which needs to be solved and there are still some issues, which needs to be addressed. Now the problem of Cancer patients of developing countries are different from are different from developed countries. Here not only the incidence is very high but majority of patient turn up to the hospital in very advanced stage. Apart from the acceptance of the advance treatment. The Awareness Education, Prevention & Early Detection are the most important things in changing the scenario in Cancer Control Programme and I am sure if we focus on these areas it will certainly change the Cancer scenario in our country.

I congratulate Dr.Ajit Saxena all entire team for bring such a wonderful seminar, which is the need of the country right now .

SPEAKERS

Breast Cancer: Current Clinical Practice, Future Directions, and Unmet Needs

Dr Anand Mishra

Professor and Head Department of Endocrine Surgery, King George's Medical University, Lucknow
Thyroid and Breast Cancer Specialist Surgeon

Breast cancer is the most common malignancy among women worldwide and represents a biologically heterogeneous disease. Advances in molecular biology and genetics have transformed its diagnosis, prognosis, and treatment by enabling personalized medicine. Clinical management is now guided not only by traditional histopathological features but also by molecular and genetic characteristics that predict disease behavior and response to therapy. Historically, treatment decisions were based on tumor size, nodal status, grade, and histological subtype, with surgery, chemotherapy, and radiotherapy constituting the principal therapeutic modalities. The identification of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor-2 (HER2) fundamentally transformed clinical management by enabling endocrine and HER2-targeted therapies, resulting in substantial improvements in disease-free and overall survival. Contemporary management further incorporates germline BRCA1/2 testing to identify candidates for PARP inhibitors, PIK3CA mutation analysis for PI3K-targeted therapy, and PD-L1 assessment for immunotherapy in selected patients with triple-negative breast cancer. Multigene expression assays such as Oncotype DX, MammaPrint, and PAM50 have refined prognostication and optimized chemotherapy selection in early-stage disease, exemplifying the integration of molecular diagnostics into routine clinical practice.

Future advances in breast cancer management are expected to rely on dynamic molecular monitoring and comprehensive genomic profiling. Circulating tumor DNA (ctDNA), circulating tumor cells (CTCs), and other liquid biopsy technologies may enable early diagnosis, detection of minimal residual disease, real-time monitoring of therapeutic response, and identification of resistance mechanisms before clinical progression. Emerging molecular targets, including PALB2, CHEK2, ATM, ESR1, AKT1, and FGFR alterations, together with single-cell sequencing, spatial transcriptomics, epigenomic profiling, proteomics, and multi-omics integration, are anticipated to further personalize treatment strategies. Artificial intelligence-assisted molecular pathology and patient-derived organoids also hold promise for individualized therapeutic prediction and optimization.

Despite these advances, important challenges remain. Triple-negative breast cancer continues to exhibit marked biological heterogeneity, and resistance to endocrine therapy, HER2-directed therapy, CDK4/6 inhibitors, and PARP inhibitors limits long-term treatment efficacy. Standardization and prospective validation of liquid biopsy assays, identification of robust predictive biomarkers, interpretation of rare genomic variants, and integration of multi-omics data into routine clinical workflows remain major unmet needs. In addition, the high cost and limited accessibility of advanced molecular diagnostics continue to impede equitable implementation of precision oncology worldwide. Ongoing translational research and biomarker-driven clinical trials are expected to bridge these knowledge gaps, facilitating more precise, adaptive, and individualized management of breast cancer.

Dr. Girish Kumar Singh

Founder Director Aiims Patna

Critical care medicine in the 21st century has witnessed remarkable advancements that have significantly improved patient outcomes in intensive care units (ICUs). This abstract provides a concise summary of the latest developments in critical care, highlighting key areas of innovation. Recent advancements in critical care include Precision Medicine: Tailoring treatments based on individual patient characteristics, genomics, and biomarkers to enhance the effectiveness of therapies. The objective is to describe the recent advancements in Critical Care Medicine. Telemedicine: The integration of telehealth technologies for remote patient monitoring and consultation, facilitating timely interventions. Artificial intelligence (AI): AI-driven tools for early disease detection, predictive analytics, and treatment optimization, enhancing clinical decision-making. Organ Support: Advanced life support systems, such as Extracorporeal Membrane Oxygenation and Continuous Renal Replacement Therapy provide better organ support. Infection Control: Innovative infection control measures to combat emerging pathogens and reduce healthcare-associated infections. Ventilation Strategies: Precision ventilation modes and lung-protective strategies to minimize ventilator-induced lung injury. Sepsis Management: Early recognition and aggressive management of sepsis with tailored interventions. Patient-Centered Care: A shift towards patient-centered care focusing on psychological and emotional well-being in addition to medical needs. We conducted a thorough literature search on PubMed, EMBASE, and Scopus using our tailored strategy, incorporating keywords such as critical care, telemedicine, and sepsis management. A total of 125 articles meeting our criteria were included for qualitative synthesis. To ensure reliability, we focused only on articles published in the English language within the last two decades, excluding animal studies, in vitro/molecular studies, and non-original data like editorials, letters, protocols, and conference abstracts. These advancements reflect a dynamic landscape in critical care medicine, where technology, research, and patient-centered approaches converge to improve the quality of care and save lives in ICUs. The future of critical care promises even more innovative solutions to meet the evolving challenges of modern medicine.

SPEAKERS

Dr. Pratap Bhadur Singh

Urologist

Bladder cancer, a prevalent malignancy affecting the urinary system, arises from the tissues of the bladder, a hollow organ responsible for storing urine. Urothelial carcinoma is the most frequent type, constituting over 90% of cases in industrialized nations. This type of cancer is notably common among older adults, with risk factors including smoking, chemical exposure, and chronic bladder inflammation. Presenting symptoms such as gross or microscopic hematuria, urinary frequency, and pelvic pain lead to its detection.

Early diagnosis and treatment are crucial for improving outcomes, as bladder cancer can range from noninvasive forms, which are confined to the inner layers of the bladder, to invasive types that penetrate deeper and can spread to other parts of the body. Treatment primarily involves transurethral resection and intravesical chemotherapy instillations but may also include laser ablation, Bacillus Calmette-Guerin bladder treatments, radiation therapy, chemotherapy, or surgical removal of part or all of the bladder.

This activity reviews the intricacies of bladder cancer, from its etiology to its therapeutic strategies. The latest advancements and comprehensive knowledge in diagnosing, treating, and managing this prevalent malignancy are discussed. Participants gain enhanced diagnostic acumen, learn evidence-based treatment protocols, and are updated on emerging research, fostering a deeper understanding of bladder cancer's evolving landscape and ultimately improving clinical practice.

The activity also highlights the interprofessional team's role in improving care for patients with this malignant condition. Collaborating with an interprofessional team enhances patient outcomes by integrating diverse expertise, ensuring comprehensive treatment plans, and providing holistic care that addresses all aspects of the patient's health, thereby improving the overall management and prognosis of bladder cancer.

Biography: A president gold medal winner and a renowned urologist, Dr. Pratap Bahadur Singh was instrumental in establishing a well-equipped department of Urology with all subspecialties. With his pioneering efforts, he successfully established a renal transplant programme at Banaras Hindu University in Varanasi. He is an influential member of the Indian Society of Organ Transplantation, Indian Society of Lymphology, SIU, Urological Society of India, East & North Zone Chapter of Urological Society of India and British Transplant Society. He was a Common Wealth Medical Fellow at the Transplant Unit in Cardiff Royal Infirmary in UK. He was also a fellow at the International College of Surgeons. During his academic years, he pursued MBBS from Banaras Hindu University and went on to pursue MS (General Surgery), M.Ch (Urology). He was also a fellow at the International College of Surgeons. Before becoming the Director of Max, he has been a senior resident of surgery and a professor and head of Urology at Institute of Medical Sciences (Banaras Hindu University) in Varanasi.

Prof M.L.B. Bhatt

Director, Kalyan Singh Super, Speciality Cancer Institute LUCKNOW

Lung cancer is one of the most common malignant cancers in most countries and is the leading cause of death among cancer diseases worldwide. Despite constant progress in diagnosis and therapy, survival rates of patients diagnosed with lung cancer remain unsatisfactory. Numerous epidemiological and experimental studies conducted as early as the 1970s confirm that the most important risk factor for the development of lung cancer is long-term smoking, which remains valid to this day. In the paper, the authors present the latest data on the epidemiology, pathogenesis, treatment and molecular aspects of this cancer. In the last decade, many molecular alterations that are effective in the development of lung cancer have been discovered. In adenocarcinoma, tyrosine kinase inhibitors were developed for EGFR mutations and ALK and ROS1 translocations and were approved for use in the treatment of advanced stage adenocarcinomas. In the case of squamous cell carcinoma, the evaluation of these mutations is not yet being used in clinical practice. In addition, there are ongoing studies concerning many potential therapeutic molecular targets, such as ROS, MET, FGFR, DDR-2 and RET. Constant progress in diagnostic and therapeutic methods gives rise to hopes for an improved prognosis in patients with lung cancer.

Biography:

Prof. Dr. Madan Lal Brahma (M.L.B.) Bhatt is the Director of the Kalyan Singh Super Speciality Cancer Institute in Lucknow and the former Vice-Chancellor of King George's Medical University (KGMU). His career spans decades in radiation oncology, medical administration, institutional development, and academic research.

The Kalyan Singh Super Speciality Cancer Institute is a state-of-the-art cancer centre that was envisaged by the Government of Uttar Pradesh under the guidance of leading cancer specialists of India, to provide comprehensive under-one-roof facilities for cancer management, research, and education.

The institute is slated to become an advanced national-level referral and treatment facility, a center of excellence for networking with national and international institutions, and a premier hub for basic and applied oncology research.

The sprawling hundred-acre campus provides treatment facilities with 750 beds in the first phase and 1250 beds in the second phase. OPD, IPD, OT, emergency, and day-care services are fully operational. The Institute currently has 45 faculty members, over 100 residents across 20 departments, and more than 1000 staff members.

SPEAKERS

Palliative Care Management of an Advance Head and Neck Cancer Patient in a Resource Poor Area: A Case Report

Uttam Changmai

Background: Managing advanced head and neck cancers in remote geographic areas presents significant clinical and socioeconomic challenges, often necessitating flexible, home-based palliative interventions.

Case Presentation: A 53-year-old male residing 67 kilometers from a tertiary treatment facility presented to the OPD of the Deptt. Of Head and Neck Oncology with a tracheostomy tube in situ. He was diagnosed with Squamous Cell Carcinoma (SCC) of the right Aryepiglottic Fold and Pyriform Sinus. Initial oncological intervention with curative intent was delayed due to concomitant tuberculosis, which required a course of Anti-Tubercular Therapy (ATT).

Management and Challenges: Following ATT clearance, the patient received Neoadjuvant Chemotherapy (Docetaxel and Cisplatin); however, the disease showed no clinical response. Although a total laryngectomy was planned, surgical fitness was repeatedly denied due to hypertension. The multidisciplinary group subsequently altered the plan to Palliative Radiotherapy, administering 30 Gy in 10 fractions.

The patient's clinical course was severely impeded by repeated treatment defaults caused by financial constraints, regional flooding, and poor road connectivity in hilly terrains. To ensure continuity of care, the patient was enrolled in a home care program. A total of 7 home visits have been conducted till date, providing critical interventions including right neck ulcer dressings (3 instances) , T-Tube changes (2 instances) , antifibrinolytic injection deliveries (4 instances) alongside analgesic optimization.

Additionally, the caregivers of the patient were also motivated for holistic care of the patient with an informal gathering. The medical social workers successfully intervened and provided Rs. 48,400 through Government financial aid scheme.

Conclusion: This case highlights the necessity of integrating robust, home-based palliative care programs and social support systems to manage advanced cancer patients facing geographical and financial barriers.

Biography: Dr. Kabindra Bhagabati, Uttam Changmai, Dr. Samujjhal Bharadwaj, Tarun Sonowal. Views of the family members about letting the patient know the diagnosis – An experience in a tertiary cancer centre in North East India. 8th Public Health Palliative Care International Conference. Palliative Care and Social Practice 2024; 18: 98.

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• Dr. Kabindra Bhagabati, Uttam Changmai, Dr. Samujjhal Bharadwaj, Dipankar Das, Ashtha Baruah. Patient Support Programme – An Experience in a Tertiary Cancer Centre in North East India. 3rd International Seminar on Public Health Research in Palliative Care, Belfast, Northern Ireland (Nov 2023).

Prof. Vinita Singh

Former HOD eye dept. KGMU, Former academic director RIO Sitapur, Professor Emeritus RIO Sitapur, Consultant VPIMS, Lucknow

Retinoblastoma is the most common primary intraocular cancer in children, comprising 2.5%–4% of all pediatric malignancies. It may be unilateral (60%–70%) or bilateral (30%–40%); bilateral tumors are usually heritable and present at an earlier age as compared to unilateral ones, 18–24 months vs. 36 months in India. 4 out of 10 children with retinoblastoma have heritable changes in the RB1 gene.

High prevalence rates, delayed presentation, and inaccessibility to healthcare lead to worse outcomes in developing countries.

The past few decades have seen a paradigm change in the treatment of retinoblastomas, shifting from enucleation and external beam radiotherapy to less aggressive modalities for eye salvage.

In hereditary retinoblastoma the gene change occurs early in development before birth and cells throughout the body have changes changes in the tomour suppressor gene, the RB1 gene. Inherited gene changes in RB1 are most likely in children with a family history of retinoblastoma, newly diagnosed bilateral or multifocal retinoblastoma.

In non-hereditary retinoblastoma the RB1 gene changes affect only the tumor cells, and cannot be passed on to future children.

A better understanding of the genetics and molecular basis of retinoblastoma has opened up the path for potential targeted therapy. Integrating genetic insights into clinical practice enhances precision, addresses recurrence risks, reproductive options, long-term cancer monitoring, reduces morbidity and healthcare costs. Genetic counseling, and screening of at-risk family members is yet to be incorporated as essential part of management.

This presentation highlights a comprehensive overview of retinoblastoma with an emphasis on recent advances in approach to management in view of the genetic insights.

SPEAKERS

Methylmercury induces pro carcinogenic effects through epitheliomesechymal transitions in the kidneys of Wistar rats

Dr. Preeti Bajpai
Associate Professor,
Life Sciences, Zoology, Lucknow

The ability of MeHg to cross placental and blood brain barriers increases the risk of its harmful effect in both neonates and adults. Organomercury, a possible carcinogen enters the food chain through various sources and bioaccumulate to toxic levels. Exposure to human occur through consumption of mercury contaminated food and water. Reno toxic effects have been reported in several cases of mercury toxicity yet no sufficient scientific data is available about the mechanism of mercury induced toxicity. Gestational exposure to mercury and its toxic effects onnervous system are well documented. However, the harmful effects of MeHg on kidneys of developing foetus is less examined. For our study, we have performed both in vitro and in vivo experiment to analyse mercury induce carcinogenesis. In vitro exposure of methyl mercury is given in 0.5uM, 1uM, 5uM concentration to NRK52E cells grown in DMEM-F12 media. For in-vivo experiment, 0.5 mg/kg and 5 mg/kg of methyl mercury was orally administered to pregnant rats from gestational day 4 GD4 till Gd21.

With increasing concentration of methyl mercury in NRK52E cell, cellular proliferation, dislodging and clubbing of cells was observed. Oxidative stress and kidney toxicity panel biomarker (Calbindin, MCP-1, KIM-1, osteopontin, and VEGF) was also assessed. Real-time PCR analysis of several genes such as cMyc, VEGF, RB1, MMP9 show significant up regulation. These genes are markers of epitheliomesenchymal transition (EMTs) and also act as proto-oncogenes. Cellular migrations in trans well plate culture was also seen in MeHg. Histopathologicalanalysis of kidneys is characterised by defective tissue morphology in mercury treated group. We performed immunohistochemistry and western blot analysis to further confirm our result. Increasein the level of pro-oncogenes foundin both in vitro and in vivo studies are proposed to be responsible for the abnormal structure and function of kidneys thus providingan understanding about the carcinogenic potential of methyl mercury

Keywords: -Methylmercury, Epitheliomesenchymal, oxidative stress, MMP9

The Next Frontier in Cancer Treatment: Integrating Radiation and Immunotherapy

Dr Pritanjali Singh
Professor & Head, Radiation Oncology Dept.

The integration of radiation therapy with immunotherapy has emerged as a paradigm-shifting strategy in cancer treatment, expanding the role of radiation beyond local tumor control to a powerful modulator of systemic antitumor immunity. Radiation induces immunogenic cell death, enhances tumor antigen release, promotes dendritic cell activation, and facilitates T-cell priming, thereby transforming the tumor microenvironment into a platform for immune activation. These effects provide a compelling biological rationale for combining radiotherapy with immune checkpoint inhibitors and other immunotherapeutic approaches.

Growing clinical evidence has demonstrated improved outcomes with radioimmunotherapy across multiple malignancies, including non-small cell lung cancer, melanoma, head and neck, gastrointestinal, and genitourinary cancers. However, several critical questions remain regarding optimal radiation dose and fractionation, treatment sequencing, target volume selection, patient stratification, biomarker development, and management of immune-related toxicities.

This keynote presentation will review the evolving scientific principles underlying radioimmunotherapy and examine landmark clinical trials that have shaped current practice. It will discuss emerging strategies to enhance therapeutic synergy, including stereotactic body radiotherapy, adaptive radiotherapy, FLASH radiotherapy, spatially fractionated radiotherapy, and the integration of artificial intelligence, radiomics, and molecular biomarkers for precision treatment planning. The presentation will also explore future opportunities involving novel immunotherapeutic agents, including bispecific antibodies, cancer vaccines, and cellular therapies.

By integrating advances in radiation biology, immunology, and precision oncology, radioimmunotherapy is poised to redefine multidisciplinary cancer care. A deeper understanding of the biological mechanisms and clinical applications of this evolving therapeutic partnership will be essential to optimize patient selection, maximize durable responses, and improve survival outcomes in the era of personalized oncology.

Biography:

Dr Pritanjali Singh

Professor & Head ,

Radiation Oncology Dept. ,

All India Institute of Medical Sciences Patna.

Ex. Assistant Professor IGIMS Patna.

Ex. Senior Resident TMH , Mumbai.

AROI Fellowship at MD Anderson Cancer Center Houston & Peter MacCallum , Melbourne.

Established the department of Radiation Oncology at AIIMS Patna with the first Linac in a govt setup at Bihar.

Fields of Interest: Breast , GI,

Gynecological malignancies.

SPEAKERS

Bladder Carcinoma: Quality of life after Urinary Diversion

Prof. P.B. Singh

Radical cystectomy is treatment of choice for invasive bladder carcinoma. It can be carried out by open surgery/laparoscopic and robotic surgery. Minimal invasive surgery reduces post-operative pain and duration of stay but quality of life depends upon type of urinary diversion after surgery.

Commonest urinary diversion has been ileal conduit where both ureters are implanted in a loop of ileum and one end is brought out side abdomen as an ostomy and patient has to bear an appliance throughout his life which effect quality of life.

Patients wish is if he can void through natural passage as before operation. This has been possible by creation of urinary bladder using intestine (Neo-bladder). Author has experience of more than 400 ileal neo-bladder with follow-up more than 25 years.

Creation of neo-bladder require special skill particularly if it is done robotically. Majority of robotic cystectomy are ending with ileal conduit and in the name of minimal invasive surgery quality of life is compromised.

For patients few days of extra hospitalization, pain and an incision does not matter if he gets a good quality of life.

Author will present his long experience on quality of life after radical cystectomy

Biography: Prof PBSingh renowned urologist of the country completed his medical education from Banaras Hindu University and then joined as faculty in department of urology and continued till 2010 as Professor and Head of the department.

He moved to Delhi in 2010 as Director Urology & Renal Transplant at Max health Care Delhi and continued till 2016. He then served Nayati Healthcare Mathura and Yashoda Hospital NCR as chairman Urology and Renal transplant.

Now he created a 100 bedded PBS Super specialty hospital Varanasi and serving as its chairman.

His field of interest is reconstructive urology, uro-oncology and renal transplantation. He has credit of performing Ileal Neo-bladder for the first time in India in 1990 and has one of the longest individual experience of more than 400 ileal neo-bladder with follow-up of more than 25 year. He also has experience of more than 600 renal transplant.

He has been past President of Urological Society of India, Executive council member of Banaras Hindu University. He has many awards, orations to his credit.

DR RAVINDRA SINGH

M.B.B.S., M.C.H., M.C.A., Ph.D., MNAMS

Non-Communicable Diseases (NCDs) are now a leading cause of mortality and morbidity globally, and mental illness is a significant part of it. In India, the treatment gap for common mental disorders is over 80%. In order to bridge this gap, mental health treatment models recommend task-shifting to non-specialists and integration of mental health care into general healthcare services. Other NCDs are being managed effectively by non-specialist healthcare workers (HCWs) at primary care, and mental illness and substance misuse are highly comorbid with other NCDs; hence, integrating mental health care within the NCD services and care framework seems logically feasible and effective. However, country-specific characteristics pose a significant challenge to the implementation of integrated care for mental disorders and NCDs. The primary objective of this study includes the development and implementation of a service delivery model that would result in at least 70% coverage of screening, linkage to care, and management of common mental disorders and substance use disorders (MSUD) among persons seeking care for NCDs at public health facilities.

Secondary objectives include assessment of the feasibility of adoption of the implementation model by the health care system and to evaluate the cost of the mental health service strengthening intervention package from the health system's and the patient's perspectives. It will be a multi-site implementation research study, employing a mixed-methods quasi-experimental, within-site, three-phase, single-arm, interrupted time series design. The implementation model comprises screening, treatment, and linkage of mental health services integrated into NCD care in at least three blocks in each of the seven selected districts of the seven selected states of India, which are geographically far apart. The expected outcome would be to increase the proportion of patients screened and managed for MSUDs among persons seeking care for NCDs at the public health facilities. The results of this implementation research will provide a roadmap for scaling up of integrated MSUDs services within general healthcare.

Biography: Dr. Ravinder Singh, a medical graduate (M.B.B.S., M.C.H., M.C.A., Ph.D., MNAMS) has been trained in public health and medical research. Currently, I am working as Sr. Scientist, Division of Non-Communicable Diseases, Indian Council of Medical Research, New Delhi & Head, WHO Collaborating Centre for Policy Research in Assistive Technology.

SPEAKERS

Less-is-More: De-escalation of Breast Cancer Surgery- Achieving Better Cure and Control with Personalized Treatment

Dr. Gaurav Agarwal

Professor & Head, Department of Endocrine & Breast Surgery, Sanjay Gandhi Postgraduate Institute of Medical Sciences (SGPGIMS), Lucknow INDIA

Breast cancer is the commonest cancer in urban women globally, and in India. It accounts for approx. 30% of all cancers in women in our country, with >50% of patients being in 25 to 50 years age group. It can very well be detected in earlier stages by breast cancer awareness, Self-examination, and Screening for breast cancer in the community. If detected early and treated properly, can be cured.

Currently, in our country- close to half all breast cancer patients are diagnosed in early stage, and the remaining in locally advanced or metastatic stages. Early stage breast cancers are curable in majority, and with appropriate treatment a large number of women who are cured of their breast cancers are now living in the country. In such patients, preserving quality of life is as important as cure of the disease. Guided by the philosophy of "Adding life to years and not just years to life", we take help of Precision medicine approach to treat our breast cancer patients, which is replacing the Blunderbuss approach to cancer Tt of the yesteryears. While less treatment can be offered to carefully selected patients with early stage and favorable biological subtypes of breast cancer patients, those with advanced stage disease still require more intensive and extensive treatment. On the other hand, those with incurable metastatic disease and poor expected survival chances need to be treated again with minimal treatment with palliative intend to provide them good symptom control and reasonable quality of life, though recognizing the fact that they will not be cured.

Contemporary breast cancer management is essentially multi-disciplinary, and surgery, radiotherapy, chemotherapy, targeted biological treatment, immunotherapy and hormone treatment all play important roles towards achieving cure in selected patients. However, not all treatment modalities are necessary for every patient. Based on the disease stage and biology, and also on response to initial treatment, the disease treatment can be de-escalated to a large extent to suit the needs of an individual patient.

What can be done "less" Without compromising cure:

• Less evaluation- limiting investigations as per clinical stage.

• Less Surgery of the breast and axilla- Breast conservation rather than mastectomy, i.e. Lesser number of mastectomies, preserving breasts in majority; Partial mastectomy, skin/nipple sparing mastectomy, with less radical excisions by having a Less extensive/ stringent margins excisions at BCS ('No ink on tumor' clearly confirmed as standard). There is sufficient data now available which proves quite convincingly that breast conservation surgery is better than mastectomy in every possible way- it provides better oncological cure, while also providing cosmetic and psychological satisfaction to the patient.

• Neo-adjuvant systemic treatment (NAST) is a major game changing strategy being employed in large proportion of patients, specially in triple negative and HER-2neu enriched cancers. In patients treated with NAST, a response adapted surgical resection within new limits after NAST too provides a major de-escalation opportunity.

• Less disfigurement by utilizing the modern oncoplastic surgery and reconstruction treatment is also being achieved more and more.

• Less axillary surgery: Surgery of the axillary nodes is an important component of breast cancer treatment. While as full axillary lymph node dissection (ALND) used to be a norm in past, with better understanding of the disease spread patterns and modern methodologies, Less axillary surgery in form of sentinel lymph nodes biopsy is equally successful in curing the patients, while preserving quality of life by preventing complications of surgery.

Sentinel LN biopsy rather than complete ALND for node negative patients is possible even in selected node positive cases, with use of NAST and downstaging of the axillary disease, thus avoiding complete ALND.

• Less / shorter Radiotherapy: Accelerated partial breast irradiation, Hypofractionation (3 weeks rather than 5)- are now the standard of care, replacing the extended standard fraction radiotherapy.

• Less Systemic Treatment: by personalization of treatment using various risk stratification and response prediction tools is now practiced widely. Multi-gene prediction tools such as Oncotype-DX and Mammaprint are used by oncologist to reliably avoid chemotherapy in low-risk, hormone responsive patients. Hormone treatment is an effective strategy in all ER/PR positive patients. Targeted/ biological therapy are offered only to high risk patients likely to benefit from such treatment.

Risk stratified comprehensive treatment planning for breast cancer patients by multi-disciplinary teams is a vital component of modern oncology practice. With careful consideration of the disease stage, biology and expected response to treatment, a safe de-escalation of treatment is possible. Less is indeed More- while treating early stage breast cancer patients today.

ORAL PRESENTATION

Role of Integrative Oncology in Reducing Chemotherapy and Radiation-Induced Toxicities through Lifestyle Modification, Yoga, and Satvik Nutrition: Bridging Ancient Vedic Wisdom with Modern Cancer Care

Dr. Mustafa Khan

MPH, MHA, HXMDP (AIIMS New Delhi), MDP (IIHMR Jaipur), LSSBB (Anexas Europe), CPHQ (USA)

Cancer remains one of the leading causes of morbidity and mortality worldwide. Although chemotherapy and radiotherapy have significantly improved survival outcomes, their associated adverse effects—including fatigue, nausea, mucositis, neuropathy, anxiety, depression, sleep disturbances, and compromised quality of life—continue to challenge comprehensive cancer management. Integrative Oncology, an evidence-informed patient-centered approach, combines conventional cancer therapies with scientifically validated complementary interventions to improve treatment tolerance, symptom control, and overall well-being.

Objective: This review explores the potential role of Integrative Oncology in minimizing chemotherapy- and radiation-induced toxicities through lifestyle modification, yoga, stress management, Satvik nutrition, and traditional Vedic health principles, while emphasizing their integration into evidence-based modern oncology practice.

Methods: A narrative review of published literature from international oncology guidelines, peer-reviewed journals, systematic reviews, and clinical studies was undertaken. Evidence related to yoga-based interventions, plant-based dietary practices, meditation, physical activity, circadian lifestyle regulation, and traditional Indian health philosophies was synthesized to evaluate their impact on supportive cancer care.

Results: Emerging evidence suggests that structured yoga interventions significantly reduce cancer-related fatigue, anxiety, depression, insomnia, and treatment-associated stress while improving physical functioning and quality of life. Lifestyle modifications, including regular physical activity, adequate sleep, stress reduction, tobacco and alcohol cessation, and maintenance of healthy body weight, contribute to improved treatment adherence and better clinical outcomes.

Satvik dietary principles—characterized by fresh fruits, seasonal vegetables, whole grains, legumes, nuts, seeds, milk products (where appropriate), and minimally processed natural foods—are rich in antioxidants, phytochemicals, dietary fiber, and anti-inflammatory nutrients. These dietary patterns may reduce systemic inflammation, support gut microbiota, strengthen immunity, and enhance recovery during cancer therapy.

Ancient Vedic concepts emphasizing disciplined daily routines (Dinacharya), balanced nutrition, mindful eating, meditation, ethical living, and harmony between mind and body closely align with current scientific understanding of lifestyle medicine and psycho-oncology. When integrated responsibly alongside standard oncological treatment, these interventions demonstrate potential to reduce treatment-related toxicities without compromising therapeutic efficacy.

Discussion: Integrative Oncology does not replace surgery, chemotherapy, radiotherapy, targeted therapy, or immunotherapy. Rather, it complements evidence-based cancer treatment by addressing physical, psychological, nutritional, social, and spiritual dimensions of patient care. Multidisciplinary integration involving oncologists, nutritionists, physiotherapists, psychologists, yoga therapists, and palliative care specialists can facilitate holistic cancer management. Future multicentric randomized controlled trials are required to establish standardized integrative protocols suitable for diverse populations.

Conclusion: Integrative Oncology represents a promising paradigm for comprehensive cancer care by combining modern scientific advances with validated lifestyle interventions rooted in traditional wisdom. Incorporating yoga, healthy lifestyle practices, Satvik nutrition, stress management, and selected Vedic health principles into routine oncology care has the potential to reduce chemotherapy- and radiation-related adverse effects, improve quality of life, enhance treatment adherence, and promote patient-centered healing. The future of oncology lies not only in curing disease but also in optimizing the overall well-being of individuals living with cancer.

Keywords: Integrative Oncology; Cancer Care; Chemotherapy Toxicity; Radiation Toxicity; Yoga Therapy; Lifestyle Modification; Satvik Diet; Vedic Medicine; Supportive Oncology; Quality of Life; Holistic Cancer Care.

ORAL PRESENTATION

Systematic identification of plant-derived lead compounds for lung cancer: A step towards cost-effective therapy

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Lung cancer continues to be the cause of a high mortality rate despite advances made in the field of surgery, chemotherapy, radiotherapy, immunotherapy and targeted therapy. With systemic toxicities from current therapies together with increased costs of novel drug developments, there has been a tremendous rise in research into exploring alternatives through affordable therapeutics. The development of natural product-based chemotherapeutic agents accounts for about 60 % of currently approved anti-cancerous medications. Senna tora is one among them, being a traditionally used medicinal plant rich in several types of secondary metabolites such as terpenoids, flavonoids, phenols, steroids and anthraquinones.

In our systematic study, we identified an optimal solvent for extractions yielding maximum recovery of both the phenol and flavonoid-rich constituents from this plant, followed by validation using GC-MS/MS analysis, which showed the presence of major phytocompounds with reported anticancer activities, namely hexadecanoic acid, 5-hydroxy-7-methoxyflavanone and ethyl isoallocholate.

Computational evaluations of the identified phytocompounds with clinically relevant oncogenic drivers such as EGFR and KRAS, which are primarily involved in lung cancer progression, were carried out. Results indicated favourable binding affinity and stable interactions between the protein-ligand complexes with standard FDA-approved anticancer drugs. The cytotoxicity tests were performed, demonstrating their potential, exhibiting a half-maximal inhibitory concentration value of the extracts (IC50 = 202.72 ± 26.9 µg/mL) along with increased ROS generation suggestive of oxidative-stress-mediated cell death. In conclusion, the findings suggest that systemisation of phytochemical extraction incorporating computational approaches could be considered as a promising strategy towards the identification of cost-effective plant-based lead compounds useful in the development of future therapeutics against lung cancer.

Keywords: Medicinal plants; lung cancer; EGFR; KRAS; phytocompounds; alternative therapies; cytotoxicity

A novel prognostic and therapeutic target in lung cancer: RAS/MAPK-FOXM1-SPC25 signaling axis

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Lung cancer remains a leading cause of cancer-related mortality worldwide (~18%), with aberrant activation of the RAS/MAPK pathway playing a critical role in tumor initiation and progression. The MAPK cascade, via its downstream effector FOXM1, regulates transcription of genes essential for cell-cycle progression. Concurrently, SPC25, a core component of the NDC80 kinetochore complex, has recently emerged as a novel oncogenic marker in lung cancer, promoting chromosomal instability and uncontrolled proliferation. Although the role of MAPK signaling is well established, and SPC25 is increasingly recognized as a prognostic biomarker, the mechanistic link between these two factors remains unclear. This study investigated the role of the RAS/MAPK-FOXM1-SPC25 signaling axis in urethane-induced lung carcinogenesis, with the aim of identifying novel molecular interactions that contribute to cell-cycle dysregulation, EMT, and aggressive tumor phenotypes.

Lung cancer was induced in rats using urethane, and disease progression was evaluated through physiological, histopathological, biochemical, and molecular analyses. Urethane-treated rats exhibited reduced body weight, altered hematological parameters, visible pulmonary pluripotent cancerous nodules, and disrupted lung architecture. Histopathological examination revealed hyperplasia, inflammatory infiltration, and neoplastic lesions. Alcian blue staining indicated increased mucin secretion, while toluidine blue and Masson's trichrome staining demonstrated enhanced mast cell infiltration and collagen deposition, respectively.

Oxidative stress analysis showed significantly elevated MDA levels, accompanied by reduced SOD, catalase, and GSH, indicating impaired antioxidant defense during tumor progression. Immunohistochemistry, immunofluorescence, and western blot analyses demonstrated marked upregulation of EGFR, KRAS, phosphorylated ERK1/2, phospho-FOXM1, and SPC25 in lung tumor tissues. Increased p-ERK expression was associated with elevated phospho-FOXM1 levels, while phospho-FOXM1 overexpression correlated with enhanced SPC25 expression, suggesting activation of a proliferative RAS/MAPK-FOXM1-SPC25 signaling cascade.

Collectively, these findings identify the RAS/MAPK-FOXM1-SPC25 axis as a potential contributor to lung cancer progression and provide a basis for its future exploration as a therapeutic target.

ORAL PRESENTATION

pRb-E2F1 complex as a target of cisplatin for inhibiting cell cycle progression in acute leukemia cells

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Acute Myeloid Leukemia (AML) is a type hematological abnormality characterized by uncontrolled proliferation of hematopoietic stem cells inside bone marrow. It constitutes around ten percentage cases of different type of cancer. Cisplatin (CDDP) has been used in chemotherapy of AML patients but patients experienced poor prognosis, relapse, and lower five-year survival rate. It inhibits growth of AML cells through diverse mechanisms for instance, cytotoxicity, regulation of cell cycle progression, and apoptosis. However, its exact molecular mechanism of CDDP action in acute leukemia cells mostly remain elusive. We propose CDDP inhibits growth of acute leukemia cells through targeting novel, pRb-E2F1 complex and regulation of interaction of pRb and E2F1 interaction . In-silico Biochemical, molecular and fluorescent microscopy method has been used for revealing the proposed new target of action of CDDP in acute leukemia cells. pRb and E2F1 structure was generated using bioinformatics software and we tried to predict the changes in interaction between pRB and E2F1 in presence of cisplatin. CDDP has changed the interaction of pRb and E2F1 at different residues (in silico data) and modulated expression of pRb and E2F1 (mRNA level) both at mRNA and protein level in a concentration dependent manner in NB4 and HL-60 cells as seen through PCR and immunocytochemistry. Above proposed mechanism of action of CDDP may be used in AML pathophysiology and drug designing.

Keywords: - Acute Promyelocyte Leukemia, CDDP, pRb, E2F1

B3GNT5 as a Biomarker and Therapeutic Target in Triple-Negative Breast Cancer

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Triple-negative breast cancer (TNBC) represents a highly aggressive subtype of breast cancer characterized by the absence of estrogen receptor, progesterone receptor, and HER2 amplification, leading to limited therapeutic options and poor prognosis. Glycosylation, a critical post-translational modification, has emerged as a key regulator of tumour progression and metastasis. B3GNT5, a β-1,3-N-acetylglucosaminyltransferase, plays a crucial role in glycosphingolipid biosynthesis and has been implicated in cancer progression. However, its specific role in TNBC remains unexplored, particularly in the context of the Indian population, which exhibits distinct genetic and environmental factors influencing disease biology.

In this study we have demonstrated that a depletion of B3GNT5 employing lentivirus packaging techniques in MDA-MB 231 resulted in a loss of proliferative capability in TNBC cells. In addition, we found that knockdown of B3GNT5 in TNBC cells inhibited cell migration, invasion, as well as stemness of cancer stem cells and enhanced chemotherapy sensitivity to the Neo-adjuvant chemotherapy (NACD) drugs given to the patients. Mechanistically, knockdown of B3GNT5 led to an increase in p-53 and PAPRP1. Depletion in B3GNT5 also led to an arrest in cell cycle at G2-M-phase. We further observed downregulation in crucial signalling pathways including p-mTOR, p-chk1and ERK1/2, signifying the role of B3GNT5 in cancer progression and proliferation. Our work confirmed a tumor-promotive role of B3GNT5 in TNBC pathogenesis. We have also analysed the expression of B3GNT5 in human breast cancer patients through qRT-PCR and Immunohistochemistry (IHC), subsequently B3GNT5 was found to be upregulated.

ORAL PRESENTATION

OncoMap: A Multi-Scale Biophysical Simulation Framework for Dynamic Tumor Invasion Modeling

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Background: Predicting spatial tumor expansion in bone marrow microenvironments and from from magnetic resonance imaging (MRI) remains a major challenge in precision oncology. Static transcriptomics and uniform growth models fail to account for anisotropic tissue density, solid mechanical stress, and invasive boundary complexities.

Methods: OncoMap, is a five-stage biophysical framework driven entirely by non-invasive multiparametric MRI (mpMRI) radiomics:

Image Ingestion & Feature Extraction: Multiparametric MRI scans (Diffusion-Weighted DWI, ADC maps, and T2-FLAIR) are processed to extract spatial cellular density tensors DADC(x)and tissue heterogeneity matrices.

Scale Mapping & Initialization: Spatial density gradients map to physical mesh coordinates. Initial 3D nodes are positioned deterministically using Laplacian Eigenmap spectral graph theory on voxel adjacency matrices.

Anisotropic Physics Engine: Tumor cell migration is simulated via an anisotropic reaction-diffusion PDE along MRI diffusion pathways. Mechanical force relaxation uses a softened Velocity-Verlet integrator to prevent numerical instability.

Concave Boundary Profiling: An -concave hull mesh reconstructs 3D invasion fronts from segmented MRI contours, yielding a non-parametric Boundary Instability Index (Bii)derived from 95th-percentile radial variance.

Validation: 4D mesh deformations simulate 6–24 month tumor expansion timelines, validated against longitudinal MRI follow-ups and clinical overall survival using Kaplan-Meier analysis.

Results: In synthetic and clinical validation cohorts, OncoMap maintained numerical stability without force accumulation divergence, accurately reconstructing 3D lesion morphology. The model effectively stratified indolent focal lesions(Bii<0.15)from aggressive, invasive osteolytic expansion (Bii0.15). Incorporating MRI-guided anisotropic diffusion and local viscoelastic relaxation significantly improved boundary spicule resolution compared to classical linear spring models.

Conclusion: OncoMap provides a robust, physics-grounded computational framework for mapping 3D osteolytic microenvironments and predicting structural expansion risk in MM patients. By integrating poro-elastic stress relaxation with MRI-guided anisotropic transport, this platform bridges functional imaging and computational biomechanics for personalized clinical decision-making.

IMPA2 Is a Potential Biomarker and Novel Target for Triple-Negative Breast Cancer

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Triple negative breast cancer is the most common aggressive type of breast cancer in which the expression of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor (HER) are absent. Inositol monophosphatase is an enzyme that converts myoinositol monophosphatase to myoinositol through dephosphorylation. IMPase can be two types: IMPA1 and IMPA2. IMPA2 has a paradoxical role in cancer. Studies have shown that IMPA2 acts as a proto-oncogene in different cancers, including breast cancer. However, the role of IMPA2 in TNBC is unclear. In this present study we showed that shRNA-mediated gene silencing of IMPA2 suppressed cellular migration and proliferation in the MDAMB231 and HCC1937 in vitro. Immunohistochemical studies (IHC) and qRT-PCR analysis of tumor tissues from Indian TNBC patients showed higher IMPA2 expression in tumor tissues in comparison to normal adjacent tissues. Knockdown of IMPA2 also induced apoptosis. Western blot analysis revealed that IMPA2 knockdown suppressed the AKT/mTORC1 signaling pathway, increased autophagy initiation through increased levels of autophagy markers like beclin1 and LC3, and decreased levels of p62. Besides this, reduced expression of IMPA2 showed a high level of E-cadherin and a low level of N-cadherin, and snail expression depicts that IMPA2 promotes the EMT process. Collectively our study revealed that IMPA2 functions as an oncogene, driving proliferation and migration in triple-negative breast cancer, and represents a potential target for therapeutic intervention.

ORAL PRESENTATION

Upregulated Fetuin-A expression persuades lung cancer cell migration

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Lung Cancer is one of the catastrophic diseases with a high rate of mortality worldwide. Fetuin-A, also known as alpha-2-Heremans-Schmid glycoprotein (AHSG), is a 63-kDA liverderived hepatokine that acts as a chemotactic factor and plays a role in tumor progression in glioblastoma, pancreas, and prostate cancers. However, little is known about the mechanism behind Fetuin-A-mediated cancer cell migration. Therefore, we systematically analysed AHSG expression using GEPIA, HPA, STRING, and UACAN databases and found that the Fetuin-A gene was upregulated in several cancers, including lung cancer (LUAD), compared to normal tissues. The expression of AHSG was correlated with tumor stages and prognosis of lung adenocarcinoma. Besides, AHSG expression induces immune cell infiltration and shows a positive correlation with TLR4 and Nrf2 expression in LUAD. When we performed in vitro analysis, for the first time, we found that Fetuin-A protein promoted non-small cell lung cancer (NSCLC) cell migration via activation of the TLR4 downstream MAP- kinase (ERK ½ and JNK) pathway. These MAP-kinases act as upstream kinases of Nrf2, which phosphorylates Nrf2 at serine 40 and allows its nuclear translocation. Activation of Nrf2 facilitated NSCLC cell migration via the expression of a battery of Nrf2 driven genes, including HIF1α. Inhibition of TLR4 or ERK1/2, or Nrf2 via siRNA significantly abrogated Fetuin-A mediated NSCLC cell migration. Besides, inhibition of Nrf2 by Trigonelline, a plant alkaloid, significantly abrogated Fetuin-A mediated NSCLC cell migration. Therefore, understanding the basic mechanism of FetuinA-mediated cancer cell migration via Nrf2 is key to developing novel therapeutic approaches for metastatic cancers. Keywords: Fetuin-A, AHSG, LUAD, TLR4, cell migration, metastasis

FOXM1–FOXO Based Prognostic Target in Meningioma: A futuristic therapeutic gateway

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Meningioma is a central nervous system tumour that exhibits notable biological and histological variability. More than half of cases are caused by changes in the mTOR pathway and the neurofibromatosis type 2 (NF2) gene. The two main therapies for meningioma are surgery and radiation therapy, however these methods only address very particular pathways of meningiomas. Even after macroscopically complete resection, meningiomas often return. Effective long-term management is still difficult, even with advancements in treatment. Recently, forkhead box protein M1 (FOXM1) has been identified as a biomolecular marker linked to poor clinical outcomes and cell proliferation. On the other hand,Forkhead box class O (FOXO) transcription factors typically repress tumours, albeit depending on the cellular environment, some subgroups may play oncogenic functions.The protein product of NF2 gene merlin regulates Cell motility, proliferation, and survival , and it also inhibits the PI3K/AKT/mTOR signalling pathway. Meningioma involves several molecular pathways, hence it is crucial to conduct study on particular regulatory axis in meningioma. The FOXM1–FOXO axis has been investigated in many cancer studies, however its function in meningioma has not been thoroughly investigated. Protein expression,molecular interaction and pathways were investigated in silico using databases like NCBI BLAST, KEGG, STRING, and UniProt. Most important pathways examined were the Hippo signalling system and the PI3K/AKT pathway. The molecular interaction between FOXM1, FOXO3, and FOXO6 were investigated in molecular level. Unique results related to meningioma were observed using in silico research. Nevertheless, wet-lab tests such as meningioma gene expression study using personalised primers for qPCR are necessary to validate in silico results. Although computational data provide accurate predictions, their use in experiments may be difficult . This axis may be used as a targeted prognostic marker for meningioma ,if it is clinically validated. Keywords:-Meningioma, forkhead box protein M1 , Forkhead box class O ,Neufibroblast type 2, PI3K/AKT/mTOR signalling pathway.

ORAL PRESENTATION

Uncommon histologic variants of Oral carcinoma & their implications with case discussion

DR.SONALEE SHAH

Most oral cancers are conventional squamous cell carcinomas (SCC) which are histologically categorised as well-differentiated, moderately differentiated or poorly differentiated. Some OSCC cases show rare histopathological deviations which are not commonly found in conventional OSCC. These variants of OSCC represent 10–15% of cases of OSCC. They differ from conventional SCC in histopathology, behaviour, and prognosis. Recognizing these rare subtypes is crucial for diagnosis and management.

Key variants include-

* Verrucous carcinoma

* Spindle cell(sarcomatoid) carcinoma

* Adenosquamous carcinoma

* Papillary carcinoma

* Carcinoma cunniculatum

* Adenoid/acantholytic carcinoma

* Basaloid carcinoma

* Clear cell carcinomas

Verrucous carcinoma has broad bulbous pushing rete ridges with parakeratotic plugging. Spindle cell carcinoma has malignant epithelial cells showing spindling/sarcomatoid appearance. Adenosquamous carcinoma is a biphasic tumor showing true glandular differentiation along with squamous differentiation. It has a relative poorer prognosis as compared with conventional SCC. It shows approximately 2-year survival rate of approximately 55%. Adenoid (acantholytic) squamous cell carcinoma has a pseudoglandular pattern with acantholytic tumor cells. Basaloid squamous cell carcinoma is a biphasic tumor showing basaloid malignant islands with peripheral palisading and comedonecrosis .It is regarded as a high-grade tumor with an increased propensity for distant metastasis. It requires aggressive multimodality treatment. The 2-year survival rate is 40%. Carcinoma cunniculatum is a deeply infiltrative, keratin-filled branching tunnels resembling rabbit burrows. It is frequently misdiagnosed as verrucous carcinoma but has a distinct architecture.

Histomorphologically every variant shows a unique appearance. Which raises an opportunity for the different diagnostic consideration with the precise management decision. Many times atypical lesions are misdiagnosed as the histologic variants which affects the prognosis of patient. Appropriate systemic approach is required for correct diagnosis.

The prognosis, metastatic potential, survival rate and treatment of each of the variants are diverse, thus mandating their distinction.

Evaluating Dilution Adequacy in Over-Range Tumour Markers Reruns: An Analytical Study of Automated Stepwise Dilution

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Background and Objectives: In high-burden oncology centres, tumour marker concentrations scale with disease burden and frequently exceed the analytical measurement range (AMR), requiring dilution to restore a reportable, linear result. We studied the dilution adequacy against the AMR and tested whether the dilution requirement is analyte driven or case specific.

Methods: We reviewed 5,575 immunoassay results on Roche cobas e 801 across eight analytes (CEA, AFP, CA 19-9, CA 125, CA 15-3, PSA, Ferritin, and Thyroglobulin) over 5 weeks and 135 over range samples were analysed. The minimum required dilution was the recovered result divided by the upper AMR limit, and dilution adequacy the diluted result as a fraction of that limit. The variation in required dilution was partitioned within and between markers to obtain the intraclass correlation (ICC).

Results: Dilution adequacy was less than 1.0 (median 0.16, IQR 0.05-0.31). Thirty-two of 135 (24%, 95% CI 17-32%) required a further dilution step, generating 37 additional runs, primarily for carbohydrate antigen 19-9 (n=24) and alpha-fetoprotein (n=6). Only one-fifth of the variation was explained by tumour marker type (ICC=0.20), with the remaining 80% attributable to case specific tumour burden. Quality control data for CA 19-9 and AFP showed modestly higher low range variability (CV up to 3.9% and 5.5% respectively with bias within 3%).

Conclusion: A uniform per marker dilution tier addresses only the baseline differences between assays; for example, raising the CA 19-9 first dilution would remove 22 additional runs but over-dilute 33 previously adequate samples. An adaptive reflex dilution protocol, validated prospectively, is preferable to uniformly higher fixed tiers.

ORAL PRESENTATION

Healing Beyond Medicine: Peer Support Communities as Catalysts for Emotional Resilience in Cancer

Dr. Mudra Binju, PhD
(Ovarian Cancer) & Founder at Vistrit Wellbeing (Together We Can)

Background: Cancer impacts not only physical health but also emotional and psychological well-being. Neuroplasticity—the brain's ability to adapt and heal—plays a vital role in resilience and recovery, particularly when supported by belief, motivation, and meaningful human connection. Objective: To highlight the importance of peer-based emotional support in enhancing patient resilience, especially during critical stages such as diagnosis and relapse. Methods: Drawing from lived experiences of cancer warriors and caregivers, alongside community-based engagement initiatives, we believe that shared understanding and connection influence coping, motivation, and overall well-being. Support is facilitated through structured social interactions including group discussions, book clubs, game nights, and informal meetups.

Results: Patients who engage with peers who have lived similar experiences report reduced feelings of isolation, improved emotional strength, and renewed motivation to continue treatment. Caregivers benefit from safe spaces that allow vulnerability, validation, and emotional release. Unlike conventional encouragement, support rooted in shared experience fosters deeper trust and hope. Conclusion: Healing extends beyond medical treatment. Communities built on empathy, trust, and positivity empower cancer warriors to reclaim purpose and caregivers to find closure. By shifting focus from illness to living, such environments strengthen emotional resilience and improve quality of life. Peer-led support systems represent a powerful complement to clinical care, reminding individuals that they are not alone—and that there is more to life beyond cancer.

Effects of Obesity on Serum Tumour Markers: A Cross-Sectional study at a Tertiary Care Hospital in Eastern India

Dr. Sahithya Kumar Jasu

Background: Obesity can affect the interpretation of serum tumour markers used in oncological diagnostics. Although obesity is a known risk factor for malignancy, its effect on the serum level of common tumour markers remains relatively underexplored in the Indian population. Two opposing physiological mechanisms— hemodilution and chronic inflammation—may alter tumour marker interpretation.The purpose of this study was to evaluate the influence of obesity on serum levels of Carcinoembryonic Antigen (CEA), Carbohydrate Antigen 19-9 (CA 19-9), Prostate-Specific Antigen (PSA), and Cancer Antigen 125 (CA 125) to determine whether obesity-related variations may affect interpretation of standard reference ranges.

Methods: A cross-sectional case-control study was done on 117 subjects (59 obese cases and 58 non-obese controls) in a tertiary care hospital in EasternIndia. Chemiluminescence Immunoassay (CLIA) was used to measure serum tumour marker levels. Statistical analysis included descriptivecomparisons, independent sample Mann-Whitney U tests, , Box and Whisker plots were used to compare distribution in both groups, ROC curve analysis to assess diagnostic power, and Q-Q plots were used to check for Normality.

Results: The findings suggested different patterns across markers, likely reflecting differences in their underlying biology. Consistent with the hemodilution hypothesis, obese individuals exhibited lower mean levels of CA 19-9 (15.35 ± 12.88vs. 19.52 ± 14.43 U/mL) and CEA (2.20 ±1.75 vs. 2.45 ± 1.8 ng/mL) compared to controls. In contrast, markers that may be influenced by inflammatory pathways showed the oppositetrend: obese males exhibited higher PSA (1.98 ± 2.04vs. 1.25 ± 1.52 ng/mL), and obese females showed higher CA 125 (13.84 ± 12.13 vs.10.51 ± 4.53 U/mL) with significantly wider variance (147.09 vs. 20.50). Conclusion: Obesity appears to exert a dual effect on tumour markers in the Indian population. While hemodilution may lower the concentration of CEA and CA 19-9, potentially masking early-stage disease (false negatives), the pro-inflammatory profile often described in Asian Indian phenotype may contribute to higher PSA and CA 125, increasing the probability of false positives. These findings suggest that obesity may affect the interpretation of tumour marker levels in clinical settings. Keywords: Obesity, Tumour Markers, Hemodilution, PSA, Asian Indian Phenotype, Cancer Screening.

ORAL PRESENTATION

Smart Intraoral Thermographic Device With Artificial Intelligence For Early Detection Of Oral Potentially Malignant Disorders And Oral Cancer

Dr. Purusharth Kumar Sharma, Dr. Manoj Meena, Dr.Neha Laskar

Early detection of oral potentially malignant disorders (OPMDs) and oral cancer remains a major challenge in dental and medical practice, particularly in high-risk populations. Conventional diagnostic approaches such as clinical examination, biopsy, and histopathological analysis are either subjective, invasive, or limited in their ability to detect early molecular and physiological changes.

Diseases such as Oral Squamous Cell Carcinoma, Oral Leukoplakia, Oral Submucous Fibrosis, and Erythroplakia often demonstrate altered metabolic activity and increased vascular perfusion prior to the appearance of overt clinical changes. These biological alterations produce localized temperature variations on the mucosal surface that can be detected using thermal imaging technologies.

The present invention proposes a Smart Intraoral Thermography Device, a portable and non-invasive diagnostic system designed for real-time detection of abnormal thermal patterns in the oral cavity. The device integrates a high-sensitivity infrared thermal sensor, a high-resolution optical imaging camera, an artificial intelligence based diagnostic processing unit, and a wireless communication module within a compact handheld intraoral probe.

The infrared sensor captures thermal radiation emitted from oral tissues and converts it into high-resolution thermal maps, while the optical imaging component simultaneously records visual images of the lesion.

These data are processed using machine learning algorithms capable of identifying abnormal temperature gradients, vascular heat signatures, and asymmetrical thermal distributions associated with premalignant and malignant lesions.

The system employs a convolutional neural network (CNN) based analysis model trained on thermal and optical datasets of normal mucosa and pathological lesions.

The algorithm evaluates temperature differentials, morphological patterns, and lesion boundaries to generate a diagnostic probability score, classifying lesions into categories such as normal tissue, inflammatory lesions, suspicious premalignant lesions, or high-risk malignant lesions.

The results are displayed in real time through an integrated interface and may be transmitted wirelessly to a smartphone or cloud based clinical database for documentation, remote consultation, and longitudinal monitoring.

The proposed device offers several advantages over existing diagnostic approaches. It provides rapid chairside screening, radiation-free imaging, non-invasive operation, portability, and cost-effectiveness, making it suitable for routine dental examinations, community oral cancer screening programs, and tele-dentistry applications.

The technology is particularly valuable in countries such as India, where the incidence of oral cancer is among the highest globally and early detection remains a critical public health priority. In conclusion, the Smart Oral Thermography Device represents a novel integration of infrared thermographic imaging and artificial intelligence based diagnostic analysis for early detection of oral potentially malignant disorders and oral cancer.

This technology has the potential to significantly enhance early diagnosis, improve clinical decision making, and reduce morbidity and mortality associated with oral malignancies

ORAL PRESENTATION

Mucosal Melanoma Of Anus : A Case Study

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INTRODUCTION:

Mucosal melanoma of the anal region is a rare and highly aggressive malignancy; accounting for less than 0.01% of all mucocutaneous melanomas and a very small percentage of anorectal cancers. It is often associated with poor prognosis due to delayed diagnosis. We report this case because of its rarity.

CASE STUDY:

A 54 year old female presented with pain and mass in the anorectal area, associated with difficulty in defecation for the past six months. She was initially managed conservatively with medical therapy but showed no clinical improvement. In view of persistent symptoms and suspicion of a neoplastic lesion, a wide local excision was performed, and the specimen was submitted for histopathological examination. Microscopic evaluation showed an ulcerated proliferation of epithelioid cells exhibiting high N/C ratio, prominent nucleoli, atypical mitosis and bizarre nuclear forms along with melanin pigment. Tumor cells were positive for S100, HMB 45 and Melan-A and negative for LCA and CK PAN.

DISCUSSION & CONCLUSION:

Anorectal melanomas frequently mimic benign conditions such as hemorrhoids or rectal polyp clinically; leading to delayed recognition and advanced disease at presentation. Histopathological examination supported by immunohistochemistry markers plays a crucial role in establishing the definitive diagnosis.

In conclusion, anorectal melanoma, though rare, should be considered in patients presenting with persistent anorectal symptoms or atypical growths. Early biopsy and histopathological evaluation are essential for timely diagnosis and appropriate management.

Machines Assist, Doctors Decide: The Need for Medical Expertise in oncology Labs: A case series based approach in a cancer hospital

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Aim: Identification of abnormal parameters in the laboratory based on preanalytical, analytical, and post-analytical error to produce correct results for the patients safety.

Methodology: Samples were identified in the laboratory based on sample characteristics such as hemolysis, dilution, reference range, and altered parameters. Haemolysed and diluted samples were traced back by identifying abnormal values by comparison with the delta values. Post-analytical errors were identified during reporting, and LIS-related issues were identified and corrected. A case series was derived from the analyzed data.

Results: Errors across the pre-analytical, analytical, and post-analytical phases were identified and corrected. Mainly, hemolyzed, diluted, and incorrect sample transport were identified in the pre-analytical phase; in the analytical phase, only a few reports were identified; and in the post-analytical phase, mostly LIS-related issues were identified.

Discussion: 70% of the clinical decisions are made based on the laboratory results produced. It becomes the immense responsibility of medical biochemists to ensure the proper release of accurate results in cancer patients. The role of automatic analyzers in today's era has been a boon for the medical field. However, the role of medical biochemists cannot be compromised. Machine work is intended to ease analysis techniques, reduce workload, improve precision and accuracy, and ultimately benefit patients. But machines also require a pre-analytical, analytical, and post-analytical supervision. Introducing incorrect samples will yield incorrect results. If results are not scrutinized at each step, wrong results may be generated.

Conclusion: The role of the machine is appreciable, but the role of medical biochemists in the oncology setting is essential. The machine helps in most ways, but a medical biochemist shows the way. Proper handling of the samples in all three phases, i.e., pre-analytical, analytical, and post-analytical, will reduce the errors and help in the proper management of the oncology patients.

ORAL PRESENTATION

Exploring PTEN-mediated regulation of Ferroptosis in Glioblastoma Multiforme

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Glioma represents one of the most aggressive forms of primary brain malignancy arising from glial cell lineages within the central nervous system. Its highly infiltrative nature and marked resistance to conventional therapies contribute to poor clinical outcomes. A critical feature underlying this therapeutic resistance is the ability of glioma cells to evade apoptosis, often driven by tumour-suppressor alterations such as phosphatase and tensin homolog (PTEN) loss or mutation. This limitation necessitates the exploration of alternative non-apoptotic cell death mechanisms. Among these, ferroptosis, an iron-dependent form of regulated cell death characterised by lipid peroxidation of cellular membranes, has emerged as a promising therapeutic target. Importantly, ferroptosis is a biologically relevant cell death mechanism that occurs under both physiological and pathological conditions, contributing to the maintenance of cellular metabolic homeostasis. PTEN is a key negative regulator of the PI3K/AKT signalling axis and plays an essential role in maintaining cellular metabolic homeostasis, including redox balance, lipid metabolism, and iron handling. However, its contribution to ferroptotic regulation in glioma remains insufficiently characterised. In this study, we aim to delineate the molecular mechanism through which PTEN influences ferroptosis in glioma. Transcriptomic datasets from The Cancer Genome Atlas were stratified based on median PTEN expression to generate PTEN-high and PTEN-low datasets. Differential expression analysis was performed, and ferroptosis-associated genes curated from FerrDb were integrated to identify ferroptosis-related differentially expressed genes. These findings were further validated using an independent single-cell transcriptomic dataset. Protein–protein interaction network analysis using STRING enabled the identification of central ferroptosis-associated regulatory nodes. Functional validation was carried out in glioblastoma cell lines with distinct PTEN status. Pharmacological induction of ferroptosis using erastin revealed enhanced susceptibility in PTEN-proficient cells, whereas PTEN-deficient cells exhibited relative resistance. This phenotype was supported by differential accumulation of reactive oxygen species, lipid peroxidation, and intracellular ferrous ions, assessed using DCFH-DA, BODIPY 581/591 C11, and FerroOrange assays, respectively. Increased expression of antioxidant and iron-storage regulators in PTEN-deficient cells further indicated adaptive metabolic rewiring. Collectively, these findings suggest that PTEN loss contributes to ferroptosis resistance in glioblastoma, highlighting ferroptosis modulation as a potential strategy to overcome apoptosis-associated therapeutic resistance.

Keywords: Glioblastoma multiforme, ferroptosis, PTEN, NACD

Mechanistic Evaluation Of Pterin-induced Apoptosis In Triple-negative Breast Cancer Cell Lines

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Triple Negative Breast Cancer (TNBC) is an aggressive type of breast cancer that lacks HER2, ER, and PR receptors with poor prognosis, unusual metastatic patterns, unique molecular profile, and lack of targeted therapy. Pteridines are low-molecular-weight heterocyclic compounds existing in different forms, such as BH4, BH2, and oxidized forms. Tetrahydrobiopterin (BH4) is the most reduced form, playing a major role as a cofactor in nitric oxide production and amino acid synthesis. This work aims to use 2-Amino-4-hydroxypteridine (Pterin) as an anticancer drug against TNBC by promoting apoptosis through the induction of NO levels. The potential use of pterin as a drug was predicted using in-silico methods and pharmacokinetic profiles. Then pterin was treated in MDA-MB-231 and L929 cells. The cell proliferative efficiency, migration were studied using clonogenic assay and scratch assay. The apoptosis was studied using DAPI, Hoechst, AO/EtBr, and MMP techniques. The RNA was isolated, and cDNA was synthesized. The primers of target genes were optimized, and pathway hypothesized with STRING. The ADME studies revealed that pterin has high GI absorption that acts as oral drug candidate. The toxicity profiles revealed pterin under class 4. Further, pterin was docked with both DHFR and DHPR, the results showed that DHPR had a better binding affinity with pterin, indicated by a glide score of -6.19. The MTT assay revealed IC50 was 85 µg/ml and concentration was not toxic in L929 cells. The total RNA was isolated with high purity, and the synthesized cDNA was used to optimize target gene primers. This unique study demonstrates the anti-cancer potential of pterins, which can act as a therapeutic agent in breast cancer.

Keywords: Pterin, Tetrahydrobiopterin, Nitric Oxide, p53, TNBC.

ORAL PRESENTATION

Repurposing Statins in Oncology: A Structural and Therapeutic Comparison of fungal derived Lovastatin and synthetic Atorvastatin in Triple-Negative Breast Cancer

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Triple-negative breast cancer (TNBC) is an aggressive subtype representing 10–20% of breast cancers, characterized by poor prognosis and limited targeted therapies due to the absence of ER, PR, and HER2 expression. A popular class of hyperlipidemic medications called "statins" is frequently used for decreasing serum cholesterol in the treatment and prevention of hypercholesterolemia. Lovastatin and Atorvastatin are the members of the statin class of drugs known as 3-hydroxy-3-methyl-glutaryl-CoA (HMG-CoA) reductase inhibitors. These drugs have been shown to have pleiotropic cardiovascular and anti-atherosclerotic effects, including cholesterol-lowering and anti-oxidant activities, immunomodulation, regulation of inflammatory response, anticancer effects, etc. Drug repurposing is a promising path, and statins, which are well-known HMG-CoA reductase inhibitors, have garnered attention for their possible anticancer properties. This study aims in the production and purification of lovastatin from Aspergillus niger CTLSF7, its structural comparison with synthetic atorvastatin In silico and elucidation of their anticancer potency against triple negative breast cancer cell line MDA MB 231 In-vitro. Based on the results, fungal derived lovastatin were more efficient when compared with synthetic atorvastatin against MDA MB 231 cell line. Thus, the results support the repurposing of lovastatin and atorvastatin in TNBC, emphasizing the need for further mechanistic studies which paves the way for their therapeutic applications.

Keywords: Triple-negative breast cancer (TNBC), Lovastatin, Atorvastatin, Anticancer effects, Drug repurposing.

The Newly Synthesized Copper-Based Anti-Cancer Drug Triggers Mitochondrial-Mediated Apoptosis in Lung Cancer Cells

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To develop a potential theranostic metal based drug effectively inhibit the cancer cell growth. In this study, we evaluated the mitochondrial mediated apoptosis induced by newly synthesized copper complex, complex (2) [Cu(L1) Cl] Cl·$2H_2O$, where L1 = 1-[2-hydroxybenzyl(2-pyridylmethyl)amino]-3-(1-naphthyloxy)-2-propanol, against human lung carcinoma (A549) cells. Cell viability, assessed using the MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide) assay, revealed that complex (2) exhibited significantly higher cytotoxic activity compared to cisplatin, with IC_{50} values of 26.5 ± 1.1 μM and 203 ± 1.2 μM, respectively. Apoptotic cell death was confirmed through flow cytometric analysis of sub-G1 populations and Annexin V/Propidium Iodide staining. Morphological and ultrastructural alterations, including plasma membrane blebbing, reduced microvilli density, chromatin condensation and fragmentation, mitochondrial damage, and endoplasmic reticulum enlargement, were observed using scanning and transmission electron microscopy. Mitochondrial dysfunction was further validated using JC-1 staining, which demonstrated a significant loss of mitochondrial membrane potential in treated A549 cells. Additionally, elevated activation of caspase-12 indicated the involvement of endoplasmic reticulum stress in the apoptotic process. Overall, these findings suggest that complex (2) exerts potent anticancer effects by inducing mitochondria-mediated apoptosis, highlighting its potential as a promising therapeutic candidate for lung cancer treatment.

ORAL PRESENTATION

Insilico Structural Characterization and Ligand Interaction Analysis of Pterin Deaminase from Agrobacterium tumefaciens through Homology Modelling and Molecular Docking

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Pteridine metabolism represents an important biochemical pathway. Pterin deaminase catalyzes the hydrolytic deamination of Pterin derivatives into the Lumazine intermediates; however, the structural information for this enzyme remains poorly characterized in several bacterial species. In this study, an In-Silico structural and functional characterization of Pterin deaminase from Agrobacterium tumefaciens was performed using homology modelling and molecular docking approaches. The amino acid sequence was obtained by the NCBI BLAST from the whole genome sequence of Agrobacterium tumefaciens, by using the Arad amino acid sequence as a reference sequence. A three-dimensional structural model was constructed using SWISS-MODEL based on a homologous template structure. The predicted model was validated through stereochemical and structural assessment, indicating a reliable and stable conformation suitable for interaction studies. To explore substrate recognition and catalytic interactions, molecular docking analysis was performed using PyRx docking engine. The ligand substrates and inhibitors were docked into the predicted active site to evaluate binding affinity and interaction patterns. Docking results demonstrated stable ligand binding with favourable binding energies and key hydrogen bonding interactions involving conserved active-site residues. Comparative structural analysis revealed similarities with other amidohydrolase superfamily enzymes, such as Guanine deaminase, suggesting evolutionary conservation of the catalytic mechanism. Importantly, pterin derivatives are closely associated with cellular redox regulation and folate-related metabolic pathways that influence tumor cell proliferation and metabolic adaptation in cancer biology. Therefore, structural insights of Pterin deaminase in the present study provide the first comprehensive structural and docking-based characterization from A. tumefaciens, offering new insights into substrate recognition, catalytic mechanisms, and potential biomedical relevance of pterin metabolism.

Keywords: Agrobacterium tumefaciens, Pterin deaminase, Gene annotation, In silico analysis, Amidohydrolase superfamily, Homology modelling, Enzyme – Ligand Interactions, Anti-tumor activity

Chitosan Nanoparticle-Mediated Delivery of Laetiporus versisporus Extracts Modulates Pulmonary Tumorigenic Signaling with In Silico Validation of Key Compounds

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Lung cancer remains a leading cause of cancer-related mortality worldwide, driven by genomic instability, dysregulated signaling pathways, and altered cellular metabolism. The identification of effective biomarkers and targeted therapies is critical for early diagnosis and improved clinical outcomes. Edible mushrooms, such as Laetiporus versisporus, are rich in bioactive constituents with anticancer potential. This study investigates the diagnostic and therapeutic potential of L. versisporus extracts against lung cancer through integrated biochemical, molecular, and computational analysis of its bioactive constituents. Crude aqueous and ethanolic extracts were prepared to selectively isolate polar polysaccharides and phenolics and cytotoxic compounds, respectively. Chitosan nanoparticle formulations of the extract were prepared and characterized by DLS, zeta potential, TEM, UV-VIS spectroscopy, and FTIR to enhance bioavailability and cellular uptake. Antioxidant assays showed superior activity in the chitosan nanoparticle formulations (DPPH 80%, ABTS 78%, FRAP 600 µM Fe^{2+} equivalents), which significantly outperformed both the aqueous and ethanolic extracts. Cytotoxicity assessed by MTT assay on A549 lung cancer cells revealed improved efficacy of nanoparticle formulations with an IC_{50} of approximately 25 µg/mL compared to crude extracts. Apoptosis induction and metabolic inhibition were confirmed via DNA fragmentation, comet assay, mitochondrial membrane potential disruption, and reduced glucose uptake. RT-PCR revealed upregulation of tumor suppressor genes (p53, Bax) and downregulation of oncogenes (Bcl-2, EGFR, KRAS) at the mRNA level. GC-MS and LC-MS identified multiple bioactive constituents, which were further evaluated via molecular docking, showing strong binding affinities (-7 to -10 kcal/mol) toward key lung cancer targets and favorable ADMET profiles. Overall, these findings highlight the potent antioxidant, cytotoxic, pro-apoptotic, and metabolic inhibitory effects of L. versisporus, with ethanolic extract and chitosan nanoparticle formulations showing superior activity, supporting its potential for lung cancer biomarker discovery and targeted therapy, warranting further in vivo validation.

Keywords: Laetiporus versisporus, Chitosan nanoparticles, Lung cancer, Molecular docking, Apoptosis

ORAL PRESENTATION

Role of MAD2 in Modulating Etoposide Sensitivity and DNA Damage Signaling in Breast Cancer Cells

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Background: DNA damage induced chromosomal instability is a major cause of cancer progression and chemoresistance Mitotic Arrest Deficient 2 (MAD2) protein is a critical component of spindle-mitotic checkpoint complex, which is frequently overexpressed in several cancers and also contributes to chemoresistance. High MAD2 expression is associated with poor prognosis and survival in breast cancer patients. Emerging evidence suggests functional cross-talk between MAD2 and the tumor suppressor p53 during the DNA damage response (DDR), although the underlying mechanisms remain unclear. In this study, we investigated the effect of genotoxic stress on the p53–MAD2 regulatory axis using Etoposide, a topoisomerase II inhibitor that induces double-strand DNA breaks. Using MAD2 overexpression models in breast cancer cells, we analyzed transcriptional and signaling responses following etoposide treatment. Our finding show that altered MAD2 expression modulates genes involved in cell-cycle progression, apoptosis, chromatin organization, and DNA repair, suggesting that MAD2 influences p53-dependent responses to chemotherapy-induced DNA damage.

Methods: MCF-7 cells (ER+/PR+) were transfected with empty vector or a full-length MAD2 construct. After 24 h, cells were treated with DMSO or etoposide for another 24 h, followed by immunoblotting and RNA sequencing. Differential expression was analyzed using DESeq2 with GO and KEGG enrichment.

Results: MAD2 overexpression broadly altered transcriptional profiles in MCF-7 cells. Etoposide downregulated cancer-related genes and pathways, and MAD2 significantly reshaped this etoposide-driven response.

Conclusion: MAD2 status critically influences ER+/PR+ breast cancer cell survival and sensitivity to DNA-damaging agents. Screening MAD2 levels may help predict drug response and guide strategies to overcome chemoresistance.

Characterization Of Pterin Deaminase From Zebrafish (danio Rerio) Tissues: Exploring Its Potential As A Metabolic Target For Cancer Therapeutics

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Pterin deaminase is an amidohydrolase enzyme belonging to the hydrolase family that catalyzes the deamination of pterin compounds to produce 2, 4-dihydroxypteridine with the release of ammonia. It acts on various pterin substrates producing lumazine derivatives and has been reported in both prokaryotic and eukaryotic organisms. In zebrafish, pteridine compounds play an important role in pigmentation, indicating a possible involvement of pterin deaminase in pigment metabolism. The association of pterin metabolism with folate-dependent pathways governing DNA synthesis and cellular proliferation underscores its potential significance in anticancer drug development. This study aims to isolate, purify and characterize the enzyme from zebrafish tissues. The enzyme was biochemically characterized to evaluate its activity under different parameters. Enzyme kinetic parameters were determined to assess its catalytic efficiency and inhibitor studies were conducted to evaluate enzyme sensitivity. Enzyme activity assays showed that catalytic activity varied with pH, temperature, and incubation time, indicating optimal conditions for enzyme function. Kinetic analysis demonstrated substrate-dependent catalytic behaviour using folic acid, while inhibitor studies revealed differential sensitivity to chemical agents, providing insights into the enzyme's catalytic mechanism. These findings provide significant biochemical and metabolic insights of pterin deaminase in the zebrafish model. As pterin–folate metabolism is closely associated with DNA synthesis and cell proliferation, this enzyme may represent a potential metabolic target in anticancer research. The study further enhances the understanding of folate-related metabolic pathways and may facilitate the development of novel therapeutic strategies for cancer drug development.

Keywords: Pterin Deaminase, Zebrafish, folate metabolism, Anticancer drug target

ORAL PRESENTATION

Elucidating anti-cancer properties of synthesized benzyl thiocyanate derivatives

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Background: Breast cancer is the leading cause of death in women globally. The existing chemotherapeutic drugs have many limitations, so we are exploring the potential of synthetic thiocyanate (SCN) derivatives which are readily found in human body. Researchers have reported that aromatic SCN compounds inhibit colorectal cancer cell proliferation whereas organochalcogenocyanates inhibit breast cancer cell proliferation. However, limited research has been done on SCN in cancer. Hence, 31 derivatives were synthesized using benzyl thiocyanate molecule as a scaffold and tested in breast cancer cell lines.

Objectives: Screening of benzyl thiocyanate derivative(s) for their cytotoxicity. Evaluating the functional aspects of the selected benzyl thiocyanate derivative. Analysing the expression of proteins involved in the activity of selected molecule.

Methodology: Breast cancer cell line and normal breast epithelial cell line was cultured to carry out in vitro experiments. Screening of compounds was conducted using cytotoxicity assays. Further, functional assays were performed to check for apoptosis, cell cycle arrest and ROS production. The clonogenic property and migration of cancer cells was evaluated post treatment. Mechanistic assays like western blotting were also performed.

Results: Th-1 exhibited cytotoxicity in cancer cell line in a dose dependent manner. It induced significant apoptosis and arrested cells at the G2/M phase, validated by the regulation of related proteins. An increase in the Median Fluorescence Intensity of the cells producing ROS was observed. It inhibited the colony formation and migration property of cancer cells post treatment by Th-1. Conclusion: The results imply that Th-1 can be a potent halogenic benzyl thiocyanate derivative. Further, functional and mechanistic studies will be conducted for the non-halogenic derivative, T-12.

Expression Profiling of m6A RNA Methylation Regulators in Oral Squamous Cell Carcinoma: Uncovering Novel Biomarker Candidates

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Background: Oral squamous cell carcinoma (OSCC) is one of the most prevalent types of head and neck cancer. Despite advancements in diagnosis techniques, detection of OSCC remains a significant challenge due to late-stage diagnosis and limited molecular biomarkers. Methylation marks arise early in tumourigenesis and can destabilise the expression of cancer-critical genes. Recently, N6-methyladenosine (m6A), an RNA modification regulated by writer, eraser, and reader proteins, regulates diverse post-transcriptional mechanisms that result in tumour development, yet its expression landscape in OSCC is not fully characterised. This study aims to evaluate the expression patterns of specific m6A regulators and their potential as diagnostic markers for the early detection of OSCC.

Methods: A total of 61 tumour and 19 normal adjacent tissues were collected from patients with informed consent and subjected to RNA isolation and cDNA conversion. Further, gene-specific primers were designed and used to profile the expression of a panel of m6A regulators, including writers (RBM15, WTAP, METTL3, METTL7, METTL14, VIRMA), erasers (FTO, ALKBH5), and readers (IGF2BP2, FMR1, YTHDC1, HNRNPA2B1), by RT-PCR. The beta-actin gene served as the internal control for data normalisation, and relative fold change was determined by using the Livak method followed by p-value calculation using Student's t-test.

Results: RT-PCR-based expression profiling elucidates that m6A regulators WTAP, METTL3, METTL14, IGF2BP2, FMR1, and ALKBH5 were significantly upregulated, while METTL7 was significantly downregulated in tumour samples. Whereas RBM15, FTO, VIRMA, YTHDC1 and HNRNPA2B1 showed no significant change. P-values were calculated using Student's t-test (*$p<0.05$, $p<0.001$, *$p < 0.001$; ns, non-significant).

Conclusion: OSCC displays selective, non-uniform dysregulation of the m6A regulators, with METTL3, METTL14, WTAP, ALKBH5, IGF2BP2, and FMR1 emerging as the most robustly altered regulators. These findings nominate a panel of genes that may serve as signature diagnostic biomarkers and mechanistic targets warranting further functional validation.

ORAL PRESENTATION

Neoadjuvant and Perioperative Systemic Therapy in Muscle-Invasive Bladder Cancer: A Systematic Review and Meta-Analysis

Prathamesh Jha

METHODS

Search Strategy and Study Selection

We conducted a systematic review following PRISMA 2020 guidelines. Details of the search strategy, databases searched, and PRISMA flow diagram are provided in Supplementary Figure S1.

Statistical Analysis

All analyses were performed using R version 4.5.1 with the meta (version 8.2-1) and metafor (version 4.8-0) packages.

Primary Outcomes

For randomized controlled trials (RCTs) reporting time-to-event outcomes, we pooled hazard ratios (HRs) for event-free survival (EFS) or disease-free survival (DFS) and overall survival (OS) using the generic inverse-variance method. Log-transformed HRs and their standard errors were calculated from reported 95% confidence intervals. We applied restricted maximum likelihood (REML) estimation for between-study variance (τ^2) and the Hartung-Knapp adjustment for random-effects confidence intervals given the small number of studies.

Secondary Outcomes

Pathologic complete response (pCR, defined as ypT0N0) and downstaging rates (defined as ≤ypT1N0 or <ypT2) were pooled using Freeman-Tukey double arcsine transformation to stabilize variance for proportions. We calculated simple pooled rates (Σevents/Σn) with Wilson confidence intervals for reporting, while forest plots display individual study proportions with 95% CIs.

Safety Analysis

Grade ≥3 treatment-related adverse events (TRAEs) were pooled across trials reporting this outcome. Event counts were estimated from reported percentages and denominators when not directly provided.

Heterogeneity Assessment

Statistical heterogeneity was quantified using the I^2 statistic and Cochran's Q test. We considered I^2 values of 25%, 50%, and 75% as representing low, moderate, and substantial heterogeneity, respectively. Prediction intervals were calculated for random-effects estimates when $k \geq 3$.

Subgroup and Sensitivity Analyses

Pre-specified subgroup analyses examined treatment effects by:

Treatment setting (neoadjuvant vs perioperative vs adjuvant)

Cisplatin eligibility status (eligible vs ineligible vs mixed)

Intervention type (ICI monotherapy vs ICI + chemotherapy vs ADC-based regimens)

Subgroup differences were tested using the Q-statistic for interaction.

Meta-Regression

We explored potential effect modifiers using mixed-effects meta-regression models with REML estimation for:

PD-L1 positivity rate (continuous)

Variant histology proportion (continuous)

Publication Bias

We assessed publication bias visually using funnel plots and statistically using Egger's regression test when $k \geq 10$. For analyses with fewer studies, we reported results with appropriate caution regarding potential small-study effects.

Certainty of Evidence

We assessed the certainty of evidence for each outcome using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) framework, evaluating risk of bias, inconsistency, indirectness, imprecision, and publication bias.

RESULTS

Study Characteristics

We identified 14 trials meeting inclusion criteria, comprising 6 Phase 3 RCTs and 8 Phase 2 studies (Table 1). The trials enrolled a total of 4,279 patients across neoadjuvant (n=6 trials), perioperative (n=5 trials), and adjuvant (n=3 trials) settings. Six trials enrolled cisplatin-eligible patients, four enrolled cisplatin-ineligible patients, and four included mixed populations.

Among the Phase 3 RCTs, five evaluated immunotherapy or antibody-drug conjugate (ADC)-based regimens: NIAGARA (durvalumab + gemcitabine-cisplatin), CheckMate-274 (adjuvant nivolumab), AMBASSADOR (adjuvant pembrolizumab), IMvigor010 (adjuvant atezolizumab), and EV-303 (enfortumab vedotin + pembrolizumab). VESPER compared two chemotherapy regimens (dose-dense MVAC vs gemcitabine-cisplatin) without immunotherapy and was analyzed separately.

ORAL PRESENTATION

Role of Lysosome-associated membrane protein-3 (Lamp3) mediated Autophagy in immunotherapy and radiotherapy in breast cancer

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Breast cancer is the most common malignancy among women and exhibits significant heterogeneity in treatment response and prognosis. Radiotherapy (RT) is a cornerstone of breast cancer management and is administered to nearly 70% of patients, achieving local tumor control primarily through irreversible DNA damage. With advances in tumor immunology, immunotherapeutic strategies have emerged as promising treatment options. However, RT can also induce immunosuppressive components. Additionally, tumor hypoxia contributes to radioresistance, limiting RT efficacy. Although single-agent immunotherapy has shown limited success in breast cancer, RT can induce immunogenic cell death, enhance tumor antigen release, remodel the tumor microenvironment, and stimulate systemic anti-tumor immunity, making RT–immunotherapy combinations an area of active investigation

Lysosome-associated membrane protein 3 (LAMP3) has been reported to be dysregulated in multiple malignancies and is associated with radioresistance in breast cancer, although its mechanistic role remains poorly defined.

LAMP3 has also been implicated in immune regulation, particularly through its association with regulatory T cell enrichment and immune checkpoint pathways. Moreover, LAMP3 exhibits context-dependent roles in autophagy, yet the interplay between LAMP3-mediated autophagy, radioresistance, and immune modulation is not fully understood.

In this study, radioresistant breast cancer cell models were established to investigate LAMP3 expression, autophagic activity, and immune checkpoint protein levels using western blotting. shRNA-mediated knockdown of LAMP3 was performed to assess radiosensitization, and its relationship with autophagy was validated using confocal microscopy, western blotting, and qRT-PCR. Immunohistochemical analysis of patient tissue samples was conducted to evaluate LAMP3 expression. Together, these findings aim to clarify the role of LAMP3-mediated autophagy in radioresistance and immune regulation, with potential implications for improving combined RT–immunotherapy strategies in breast cancer.

Hospital-Based Epidemiological Trends of Gastric Cancer in Bihar, India: A Five-Year Retrospective Cohort Study

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Background: Gastric cancer is still the fourth leading cause of cancer-related death globally. Late diagnosis is common in resource-limited areas, leading to disproportionately poor outcomes. Region-specific epidemiological data from Bihar, India, remains scarce despite its high reported burden of gastric malignancies.

Objective: To determine the demographic, clinicopathological, and geographic patterns of gastric cancer among patients from Bihar registered at Mahavir Cancer Sansthan and Research Centre (MCSRC), Patna, over a five-year period.

Methods: A retrospective hospital-based analysis was conducted on gastric cancer patients registered at MCSRC between years 2020 and 2024. Patients inhabiting in the state of Bihar were selected for the study. Collection of data related to age, gender, stage at diagnosis, district of origin, and tumor morphology of the patients were extracted from medical records and analyzed descriptively.

Results: Out of 1,209 patients screened, 1,135 met the inclusion criteria. The cohort showed a male predominance (65.6%; M:F ≈ 1.9:1), with a mean age of 55.1 ± 13.9 years (range 1.5–90). Moreover, 80.7% of gastric cancer patients were diagnosed in very advanced stage (Stage III–IV), with Stage IV alone accounting for 66.5% of cases. Adenocarcinoma was the dominant histology (>85%). Among the studied demographic districts Patna, Nalanda, East Champaran, Saran, and Gaya respectively contributed the highest patient volumes.

Conclusion: This is among the largest region-specific studies of gastric cancer from Bihar and presents a strikingly high proportion of advanced stage presentation. These findings highlight the need for public awareness campaigns for early symptom identification and for needful medical interventions.

Keywords: Gastric cancer, Bihar, epidemiology, stage of diagnosis, retrospective study

ORAL PRESENTATION

MK3 Attenuates PI3K/PDK1 Signalling Axis to Restrict Prostate Cancer Bone Metastasis

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Castration resistant prostate cancer (CRPC) predominantly metastasizes to bone, leading to increased mortality and therapeutic resistance. The bone microenvironment plays a vital role in promoting prostate cancer bone tropism and regulating tumor cell behavior. This study investigates the potential of carbazole alkaloid MK3 in inhibiting prostate cancer metastasis and delineates the underlying molecular mechanisms. Overlap analysis of human proteome of metastatic prostate cancer identified PDK1, MIF and EPHX2 as potential drug targets of MK3. Molecular docking revealed that MK3 exhibits a high druggability score of 0.97 with PDK1 by forming stable interactions. Molecular dynamic simulation studies were performed between MK3 and PDK1 which confirmed the stability of the complex over 100 ns. MK3 was isolated through solvent extraction and purified through column chromatography. The efficacy on metastatic CRPC cell lines (PC3 and DU145) was found to be dose and time dependent; the IC50 was found to be 50nM for 48 hours. The migratory and invasive abilities were also significantly inhibited upon MK3 treatment. Key metastatis-associated differentially expressed genes (TAGLN, MIF, SLC2A1, CSRP1) were identified and validated by qRT-PCR and Western blotting with/without the effect of MK3. This revealed that MK3 significantly downregulates genes responsible for cancer cellular proliferation, migration and invasion. PDK1 was cloned and expressed in plasmid vector pcDNA3.1 for assessing the effect of PDK1-overexpression on migration and invasion in MK3 treated/untreated CRPC cells. Subsequently, migration and invasion assays were performed in PDK1-overexpressing stable cell lines untreated/treated with MK3 (50 nM, 48 nM hours). PDK1-overexpressing stable cell lines did not exhibit reduced migratory or invasive abilities upon MK3 treatment, underscoring the critical role of PDK1 in driving prostate cancer metastasis to bone. PDK1 was cloned into pET28a vector and protein expression was successfully induced with 1.5 mM IPTG. The recombinant PDK1 was purified using Ni-NTA affinity chromatography for subsequent drug–protein interaction studies and phosphorylation assays.

Keywords: Carbazole alkaloid, Molecular docking, Cloning, Gene expression

Cognitive And Behavioral Gaps In Cervical Cancer Awareness: A Psycho-Oncological Assessment Of Women In Bengaluru

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Background: Cervical cancer remains a significant public health challenge in India, where late-stage diagnosis often results from psychological and cognitive barriers to screening. Objective: As part of a larger research initiative in preventive healthcare and psycho-oncology, this phase of the study quantitatively evaluated cervical cancer awareness and knowledge gaps among women in Bengaluru. Methods: A cross-sectional survey was conducted with 360 participants using a validated Cervical Awareness Measure (CAM) adapted for the local context. Awareness was scored across five domains: unprompted symptom recall, age-related risk knowledge, unprompted risk factor recall, closed-ended risk factor recognition, and HPV vaccine awareness. Data were analyzed according to a strict scoring protocol where missing or 'don't know' responses were excluded from the awareness totals to ensure accuracy. Results: The findings revealed a profound deficit in unprompted health literacy. The mean total awareness score was 4.80 out of a possible 32. While participants demonstrated moderate success in prompted recognition of risk factors (mean 3.39/8), unprompted recall for symptoms (mean 0.28/11) and risk factors (mean 0.24/11) was critically low. Overall, 96.7% of the cohort (n=348) fell into the 'Low Awareness' category, with only 3.3% showing moderate awareness. Conclusion: From a health psychology perspective, the significant disparity between prompted recognition and unprompted recall suggests that while basic information is present, it is not 'top-of-mind,' creating a barrier to early symptom detection and proactive diagnosis seeking. These results emphasize the need for psycho-educational interventions that move beyond passive information to active, unprompted health literacy to improve early diagnosis outcomes for women.

Keywords: Cervical Cancer Awareness; Psycho-Oncology; Preventive Healthcare; Health Psychology.

ORAL PRESENTATION

Osteopontin Regulates the Activation of the Hippo Pathway and Its Implication in Breast Cancer Growth

Samikshya Mahapatra

Breast cancer remains a prevalent malignancy in women around the world. Its high rate of mortality is due to metastasis and resistance to conventional treatments. Understanding the molecular pathways behind breast cancer growth is critical for identifying potential therapy targets. OPN is a secreted glycoprotein that plays numerous biological functions and has been linked to tumor migration, metastasis, and poor prognosis in breast cancer patients; however, the exact biochemical interactions of OPN with the Hippo signaling cascade and autophagy are not entirely understood. Hippo pathway maintains tissue homeostasis by modulating apoptosis, organ size, and cell proliferation. This pathway primarily depends on the kinases MST1/2 and LATS1/2, as well as the downstream effectors YAP/TAZ. The transcriptional activity of YAP/TAZ is inhibited when the Hippo pathway becomes active, because once phosphorylated, YAP gets retained in the cytoplasm. YAP/TAZ translocate to the nucleus and stimulate carcinogenic transcriptional events when the pathway is dysregulated, which results in unchecked cell proliferation and metastasis.

Currently, the dysregulated Hippo signaling pathway has also been connected to a variety of malignancies, including breast, liver, lung, gastric, prostate, and colorectal cancers.

Apart from its function in regulating cell proliferation, the Hippo pathway also governs autophagy, a cellular mechanism that degrades and recycles damaged proteins and organelles. Autophagy is known to show a dual role in cancer. On one hand, it tends to clear the oncogenic proteins and unfolded or misfolded proteins, preventing tissue damage and genomic instability. On the other hand, tumor formation leads to an increased autophagic flux that often facilitates the growth and survival of tumor cells. This makes autophagy an interesting target for researchers and clinicians. We propose that OPN plays a crucial role in breast cancer progression and metastasis by regulating the activation of the Hippo signaling pathway and autophagic flux induction.

Esr2-mediated Modulation Of Estrogen Signaling In Ovarian Cancer And Therapeutic Potential Of Quercetin As A Phytoestrogen

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Ovarian cancer (OC) is the deadliest gynecological cancer often associated with asymptomatic progression and late diagnosis, complex and variable molecular etiology, and with chemotherapeutic failures or resistance. The importance of estrogen-estrogen receptor (ER) axis especially ER-alpha (ESR1) modulation in OC has been reported; but the significance of differential ER-beta (ESR2) expression needs investigation because of documented enigmatic roles in different cancers.

Aim and methodology: The present study involving SKOV3 cell line with mutated ESR1 alpha and normal ESR2 was utilized to understand the above lacunae in OC using quercetin which is a phytoestrogen and partial selective estrogen receptor modulators (SERM) in comparison to the established drug tamoxifen; based on cellular in vitro and molecular assays and tools.

Result: The importance of estrogen-ER axis was evaluated using estradiol stimulation in SKOV3 cell line; which indicated an increased trend of cell proliferation, migration, size etc., as well as increased ESR2 expression which strongly correlated with key downstream oncogenic markers like MAPK1, MAPK3, AKT1, PI3K, CCND1 etc., and downregulated p53 expression; thereby strongly indicating its significance in oncogenesis. Quercetin was observed to positively modulate ESR2 mediated cell signaling in presence of estradiol but antagonistically to estradiol. Quercetin (150 μM) effectively killed SKOV3 cells, restricted cell proliferation and migration (scratch assay) etc., as well as downregulated the key oncogenic signatures at mRNA levels. It also increased ESR1 re-expression making the cells chemo-susceptible to ER based drug targeting. The quercetin drug complemented or at some levels even bettered the results obtained with SERM tamoxifen (1 μM).

Conclusion: The study strongly indicates the significance of ESR2 mediated signaling and its specific modulators in regulating important cellular and molecular parameters associated with OC. With literature suggestive of tamoxifen resistance, alternatives such as phytoestrogen quercetin may be of clinical relevance and therapeutic significance in ESR1-negative ESR2-positive OC cases.

Keywords: Ovarian cancer, Tamoxifen, Phytoestrogen, Estrogen Signaling, Estrogen receptor

ORAL PRESENTATION

Assessment of compliance with breast cancer screening among health care professionals in a teaching hospital

Sandhya Srinivasan

The objective of this study was to assess awareness and compliance with breast cancer screening among health care professionals in a tertiary care teaching hospital in south India

An observational study was conducted in the months of June to October 2023 in a teaching hospital at Chennai, TamilNadu. The sample group was limited to female doctors above the age of 40 all of whom had completed postgraduate qualifications at the time of study. 90 doctors were asked open ended questions regarding awareness and compliance in a mix of telephonic and in person interviews and their answers recorded.

All doctors in the study were aware of the necessity of screening. However, only 17.7% (16) were aware of the correct screening protocols and were compliant. Among the noncompliant, major reasons for noncompliance were: 32.3% felt repetitive screening unnecessary, 12.3% stated lack of time in their schedule as the reason and 7.8% feared the screening process due to pain and/or radiation exposure.

This study shows that awareness and compliance for breast cancer screening even among health care professionals is poor and needs to be addressed. Doctors may move toward more regular screening by education regarding correct protocols and reassurance of the screening process, which may lend more confidence and result in better compliance.

Sarmistha Jena

Chemotherapy resistance remains a major clinical challenge in the treatment of breast cancer and is a key factor contributing to tumor recurrence, disease progression, and poor patient prognosis. Among commonly used chemotherapeutic agents, resistance to Tamoxifen represents a significant barrier to effective therapy. In the present study, we established a Tamoxifen resistant breast cancer cell line through prolonged and incremental exposure to Tamoxifen and systematically characterized resistance associated alterations.

Comparative analyses between parental and resistant cells revealed remarkable changes in the expression of key breast cancer receptors, including estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2), suggesting molecular reprogramming during the acquisition of chemoresistance. Further comparative analysis demonstrated expression of stemness markers NANOG, OCT4 and SOX2, along with drug resistance marker ABCG2. The tamoxifen resistant cell line showed elevated expression of these markers, indicating enhanced stemness and increased resistance properties.

Functional assays further demonstrated that Tamoxifen resistant cells exhibited significantly enhanced migratory capacity and increased colony forming ability relative to their parental counterparts. These phenotypic changes indicate the emergence of a more aggressive, and therapy-refractory cellular phenotype following long-term chemotherapy exposure.

Collectively, our findings suggest that acquired Tamoxifen resistance is not merely associated with reduced drug sensitivity but is accompanied by broader biological alterations that may promote tumor progression and malignancy. Importantly, this study provides a robust experimental approach for future investigations aimed at elucidating the mechanisms underlying chemoresistance and identifying novel therapeutic strategies to reverse Tamoxifen resistance. Ongoing and future work will focus on resistance-reversal approaches and combination treatment strategies designed to restore chemosensitivity and improve therapeutic outcomes in resistant breast cancer.

Osteopontin Regulates PI3kinase/MAPK-dependent Autophagy that Controls Breast Cancer Progression

Sinjan Khanra

Breast cancer remains the most frequently diagnosed cancer and the second leading cause of cancer-related deaths among women worldwide. Despite advances in early detection and treatment, disease recurrence and metastasis continue to pose major clinical challenges. Understanding the molecular mechanisms that drive breast cancer progression is therefore essential for developing more effective therapeutic strategies.

Osteopontin (OPN) is a multifunctional secreted glycoprotein involved in cell adhesion, migration, immune regulation, and extracellular matrix remodeling. Numerous studies have shown that OPN levels are significantly elevated in various cancers, including breast cancer. High OPN expression correlates with aggressive tumor behavior, enhanced metastatic potential, and poor patient prognosis. OPN contributes to tumor progression by activating several signaling pathways that promote proliferation, invasion, and survival. However, despite its recognized role in cancer biology, one important aspect of OPN function remains poorly understood, which is its relationship with autophagy.

Autophagy is a conserved cellular degradation process that maintains homeostasis by recycling damaged organelles and proteins. In cancer, autophagy plays a dual role: it can suppress tumor initiation by preventing cellular damage. Still, it can also support tumor growth and metastasis by helping cancer cells survive under stress conditions such as hypoxia or nutrient deprivation. The connection between OPN and autophagy remains unclear.

This project aims to investigate how OPN regulates the autophagic pathway in breast cancer cells by analyzing changes in autophagy markers, signaling pathways, and cellular behavior in response to OPN. The study seeks to determine whether OPN acts as an inducer or inhibitor of autophagy. Thus, understanding this interaction may reveal new mechanisms by which OPN drives breast cancer progression and could identify novel therapeutic targets to limit breast tumor growth and metastasis.

ORAL PRESENTATION

Comparative Performance of Friedewald's, Martin-Hopkins', and Sampson's Equations for LDL-Cholesterol Estimation Against Direct Measurement in Oncology Patients: A Tertiary Care Cancer Hospital-Based Retrospective Observational Study

Dr. Shraddha Kailani

Post Graduate Resident, Dept. of Biochemistry, Homi Bhabha Cancer Hospital and Mahamana Pandit Madan Mohan Malaviya Cancer Centre (A Unit of Tata Memorial Centre, Mumbai), Varanasi, Uttar Pradesh

Background: Dyslipidemia, driven by tumor-related inflammation, liver disease, cachexia, and chemotherapeutic effects, is common among cancer patients; consequently, these patients often have hypertriglyceridemia and low LDL cholesterol (LDL-C) levels. Under these circumstances, formula-based LDL-C estimates are unreliable. This can lead to overtreatment with lipid-lowering drugs or to suitable candidates not receiving appropriate treatment.

Objective: To directly compare LDL-C values obtained by direct measurement with those calculated using the Friedewald, Martin-Hopkins, and Sampson formulas in patients with cancer and to assess the impact on treatment risk classification.

Methods: Lipid profiles from 72 cancer patients with data for both direct LDL-C and all three formulas were analyzed retrospectively. Correlation among methods was assessed using Pearson/Spearman correlation, bias and limits of agreement (Bland-Altman), paired t-test, and risk category agreement using Cohen's kappa, with risk categories defined according to NCEP ATP III criteria and TG subgroups.

Results: Correlation was highest with Martin-Hopkins ($r = 0.976$), followed by Sampson's ($r = 0.939$) and Friedewald's ($r = 0.902$). All three equations significantly underestimated LDL-C ($p < 0.0001$), with mean biases of -7.85, -10.55, and -16.10 mg/dL, respectively. NCEP ATP III risk category agreement was highest for Martin-Hopkins (79.2%; $\kappa = 0.71$) and lowest for Friedewald (59.7%; $\kappa = 0.44$).

Conclusion: Formula-based LDL-C estimates, especially Friedewald's, have been shown to underestimate directly measured LDL-C levels in cancer patients; this underestimation is even more pronounced in the presence of hypertriglyceridemia, which is prevalent among cancer patients. The Martin-Hopkins and Sampson equations show better correlation with NCEP ATP III criteria than Friedewald's and should be used in place of Friedewald's.

Development and Validation of Culturally Tailored Pamphlets on Cervical Cancer Prevention for Rural and Migrant Populations in Greater Noida

Dr. Shruti Singh

Background: Cervical cancer remains a leading cause of mortality among women in marginalized Indian communities. Effective Information, Education, and Communication (IEC) materials are crucial for health promotion, but they must address hyper-local barriers to drive screening uptake. This ongoing 3-month study (concluding August 1, 2026) outlines the development and community validation of context-specific IEC pamphlets designed for the unique rural and migrant demographics of Greater Noida.

Methodology:

This study employs a two-phase qualitative design:

1. Phase 1 (Month 1 - Development): Three focus group discussions (FGDs) were conducted with 18 frontline health workers (7 ANMs and 11 ASHAs) to map community barriers and draft the initial IEC material.

2. Phase 2 (Months 2 & 3 - Validation): The draft pamphlets are currently undergoing field validation (June–July 2026) via in-person interviews with 12–15 women aged over 30 years from the target rural and migrant communities. These interviews evaluate comprehension, visual appeal, and cultural acceptability.

Results: Phase 1 findings revealed critical barriers that shaped the initial draft: the necessity of a universal screening message to bypass risk-factor stigma; transparently addressing that testing involves a pelvic examination to mitigate clinic-site fear; integrating HPV vaccination for adolescent daughters; and reassuring the community of high cure rates to counter cancer phobia.

Phase 2 field validation is currently active. Preliminary interview insights indicate a strong preference for simplified local dialects and relatable visual graphics over dense text. The final compiled results, user feedback themes, and the field-modified pamphlet will be fully presented at the conference.

Conclusion: Developing and validating IEC materials by leveraging both the systemic insights of frontline workers and the direct lived experiences of the target community ensures clinical outreach aligns with community reality. This validated tool will offer a scalable, culturally safe framework for oncology prevention programs among vulnerable migrant and rural populations.

ORAL PRESENTATION

Metastatic hepatocellular carcinoma: Epigenetic silencing of claudin-4 by EZH2/H3K27me3 promotes hypoxic Wnt/β-catenin signaling and metabolic reprogramming

Smriti Verma

Division of Toxicology and Experimental Medicine, CSIR-Central Drug Research Institute (CSIR-CDRI), Lucknow, 226031, India

Background: Extrahepatic hematogenous metastasis is a major cause of hepatocellular carcinoma (HCC) progression, with the lung being the predominant metastatic site. Although Claudin-4 dysregulation has been implicated in HCC, its epigenetic regulation and association with hypoxia-induced β-catenin signaling and metabolic reprogramming during metastasis remain unclear. The current study investigated the role of epigenetic modulation of Claudin-4 in regulating HIF-1α/β-catenin signaling and metabolic adaptation in HCC lung metastasis.

Approach and result: In Vitro Immunofluorescence on HepG2 cells showed that pharmacological inhibition of EZH2 using tazemetostat restored Claudin-4 and suppresses β-catenin and EMT. In vivo DEN and NMOR-induced HCC metastasis SD rat model exhibited a marked reduction in body and organ weights, accompanied by significant alterations in liver function markers and GGT levels, along with notable hematological changes as determined from serum and blood sample analyses. Gross morphology and histopathological analyses confirmed a well-established HCC lung metastasis model, characterized by multiple small hepatic nodules coalescing into large tumor, along with black metastatic foci and squamous cell carcinoma-like features in lung tissues. Increased expression of EZH2, H3K27me3, β-catenin, LEF-1, VEGF, S100A4, and EMT markers (Snail, Vimentin, and CTNNAL-1), along with reduced Claudin-4 and E-cadherin expression, promoted carcinogenicity, epithelial–mesenchymal transition, and metastasis, as confirmed by immunohistochemistry, immunofluorescence, and immunoblot techniques, resulting in the loss of cell–cell adhesion, imparting migratory potential to HCC cells. TUNEL assay demonstrated suppressed apoptosis in invasive HCC cells. Moreover, the hypoxic tumor microenvironment stabilizes Hypoxia-Inducible Factor-1α (HIF-1α), supports tumorigenesis and metabolic adaptation.

Furthermore, high-field 800 MHz, LC-MS/MS, 1H Nuclear Magnetic Resonance (NMR) based untargeted tissue metabolomics analysis in time dependent manner of liver tissue lysates identify significant alterations in metabolites associated with the progression of HCC and extrahepatic metastasis. Collectively, these findings demonstrate that EZH2/H3K27me3-mediated Claudin-4 repression promotes β-catenin-driven metabolic rewiring during HCC metastasis and identifies potential metabolomic biomarkers and therapeutic targets for metastatic HCC.

Integrated Discovery and Translational Evaluation of Conjugated Small molecules for Anticancer Drug Development

Soma Mandal

School of Biological Sciences, National Institute of Science Education and Research, HBNI, Jatni, Odisha 752050, India

Cancer remains a major global health challenge owing to persistent dysregulation of proliferative signalling pathways, therapeutic resistance, and tumor adaptive mechanism that collectively compromise treatment efficacy and long-term clinical outcomes. Despite substantial advances in precision medicine and targeted therapeutics, the development of novel small molecules capable of producing robust biological responses with improved translational potential remains a significant challenge in contemporary anticancer drug discovery.

In the present study, a series of structurally modified small-molecule conjugates were rationally designed, synthesized and systematically evaluated using an integrated experimental workflow encompassing phenotypic screening, mechanistic investigations, computational analyses, and in vivo validation. Initial biological screening across multiple cancer models identified several active candidates demonstrating significant growth inhibitory effects and modulation of tumor associated cellular phenotypes.

Subsequent functional characterization revealed suppression of proliferative capacity accompanied by alteration in cellular processes associated with tumor progression, survival and cellular adaptation. Integrating mechanistic and computational investigations provided evidence supporting modulation of molecular network associated with proliferative regulation and therapeutic response, offering preliminary insights into the biological basis underlying the observed anticancer effects. Collectively, these findings suggest that the biological activity of these conjugated molecules may extends beyond simple cytotoxic effects and may involve broader modulation of cancer-associated cellular processes.

Furthermore, translational evaluation in a murine breast cancer model demonstrated meaningful suppression of tumor progression with favourable tolerability profiles. Overall, these findings identify conjugated small molecules as biologically relevant scaffolds that warrant further investigation toward the development of improved anticancer therapeutics with improved translational potential.

ORAL PRESENTATION

Community-Based Mobile Cancer Screening for Early Detection of Common Cancers: Experience from Rural Villages in Northern India

Suprakash Mandal

Background: Cancer problem in India is an increasing public health challenge. Rural areas and low resource settings have limited access to screening services for cancerous and precancerous lesion. Community based mobile screening program can fill this gap and ensures early detection and timely referral for common cancers.

Objective: This study described the implementation and programmatic outcomes of a community-based mobile cancer screening initiative conducted across rural villages in Northern India.

Methods: This was a descriptive program evaluation conducted from data of Mobile Cancer Screening Unit. The unit was a bus equipped for clinical examination, oral, breast, and cervical cancer screening, chest X-ray, mammography, and laboratory investigations. Team consisted of medical officer, dentist, nurses, laboratory and radiology technicians, patient trackers, attendants, drivers, and community mobilizers. Villagers attending camps were registered, screened for common malignancies, and were referred for further diagnostic evaluation and management. Camp data were reviewed to present participant characteristics, screening outcomes, and referral patterns using descriptive statistics.

Results: Total 38 camps conducted between December 2025 and June 2026, screening 2,311 individuals (mean age 41.0 ± 16.8 years); 52.4% were male and 73.8% aged ≥30 years. Nearly all participants (99.6%) never had cancer screening before, and 95.7% received neither HPV nor hepatitis-B vaccination, 32.9% had raised blood pressure, 34.8% overweight/obese, and 53.1% high waist circumference. Oral examination revealed suspicious lesion in 49 participants (2.1%), more among males (3.1% vs. 1.0%), clinical breast examination of 398 women detected abnormalities in 12 (3.0%). Overall, 155 participants (6.7%) were referred for further evaluation, with oral potentially malignant disorders (4.4%) being the most common indication, followed by suspicious breast lesions (0.5%).

Conclusion: Community-based mobile cancer screening model was feasible and effective for early detection. Apart from identifying individuals with suspected cancer and premalignant lesions, it also identified participants with cardiometabolic risk factors. Improving community participation and stronger referral may further enhance the impact.

An Uncommon Hematological Malignancy in Down Syndrome: A Case of Pediatric Acute Promyelocytic Leukemia

Suhani Sinha

Introduction: APL(Acute Promyelocytic Leukemia) is a rare leukemia comprising less than 10 percent of Pediatric AML(Acute Myeloblastic Leukemia). Down Syndrome is associated with a 15 times increased risk of leukemia, the most frequent of which is acute megakaryoblastic leukemia. APL is very rarely found with Trisomy 21 and only a few cases have been published in medical literature so far.

Case Description: Here we describe a case of a 12-year-old female with Down Syndrome who presented with easy fatigability and pallor for 2 weeks and menorrhagia. Examination revealed no lymphadenopathy . Complete blood count showed pancytopenia with hemoglobin of 3.5 g/dL. Peripheral smear demonstrated 47% atypical/buttock cells with Auer rods. Serum LDH(Lactate Dehydrogenase) was markedly elevated. Bone marrow biopsy revealed hypercellular marrow with 81% blasts and diffuse promyelocytic infiltration with suppression of erythropoiesis, granulopoiesis, and megakaryopoiesis. Cytogenetic analysis showed t(15;17) translocation, while flow cytometry confirmed PML-RARA positive Acute Promyelocytic Leukemia.

The patient was initiated on ATRA(All trans retinoic acid), followed by induction chemotherapy with ATO(Arsenic Trioxide) and ATRA as per AAML 1331 protocol.

During therapy, she developed multiple complications including differentiation syndrome, febrile neutropenia, upper respiratory infection due to Influenza B, tumour lysis syndrome, acute kidney injury and oral candidiasis. She required intensive supportive care including antimicrobials, dexamethasone, rasburicase, allopurinol, ionotropic support, and blood component .

Result: The BMA(Bone Marrow Aspiration)and biopsy done on day 44 of induction were suggestive of morphological remission. The haemoglobin increased to 10.5g/dL and the platelet count increased to 2.8 lacs. Induction chemotherapy was hence stopped on day 48 and the patient was discharged within a week; she is currently undergoing maintenance chemotherapy.

Conclusion: This case highlights the importance of high index of suspicion for APL in Down Syndrome which requires urgent initiation of therapy with a combination of ATRA and ATO. This leads to excellent remission rates however management of complications during the treatment such as differentiation syndrome is crucial.

Keywords: APL(Acute Promyelocytic Leukemia),Down Syndrome, Differentiation Syndrome, PML RARA fusion, ATRA(All Trans Retinoic Acid) therapy

ORAL PRESENTATION

A Rapid UHPLC Method for Simultaneous Therapeutic Drug Monitoring of Imatinib, Nilotinib, Voriconazole, Posaconazole, and Isavuconazole

Mr. Murari Gurjar

Background: Therapeutic drug monitoring (TDM) is essential for drugs with a narrow therapeutic window, significant interpatient variability, and a risk of toxicity when levels exceed the therapeutic range. However, performing TDM in clinical pharmacology is labor-intensive and demands considerable time and personnel. Since the timing of monitoring is critical for patient safety, efforts have focused on reducing turnaround time. In this study, we standardized a method for simultaneous detection of five therapeutic agents including two tyrosine kinase inhibitors and three triazole antifungals regularly being used in cancer patients. The assay requires less time and fewer resources, which also helps lower the overall cost.

Methods: Chromatographic separation was performed on a Shimadzu UHPLC system equipped with a C18 column and PDA detector using a gradient mobile phase of acetonitrile–methanol and ammonium acetate. Imatinib, Voriconazole, Isavuconazole, Nilotinib, and Posaconazole were simultaneously analyzed with a 30 µL injection volume and a 22-minute run time. Human plasma calibration standards (0.25–10 µg/mL) and quality control samples were prepared by spiking known drug concentrations, followed by protein precipitation with acetonitrile and methanol. The method was validated according to Bioanalytical Method Validation Guidance for Industry for selectivity, linearity, accuracy, precision, recovery, carry-over, and stability.

Results: The UHPLC–PDA method achieved complete separation of five drugs within 22 min with excellent selectivity and no endogenous interference. It demonstrated good linearity (0.25–10 µg/mL; $R^2 > 0.99$), high sensitivity (LLOQ: 0.25 µg/mL; LOD: 0.03–0.04 µg/mL), acceptable accuracy and precision (±15% RE, ≤15% CV), and recovery of 74.38–103.80%. Successful analysis of patient plasma confirmed its applicability for routine therapeutic drug monitoring.

Conclusions: The validated UHPLC–PDA assay is a simple, sensitive, precise, and robust method for simultaneous therapeutic drug monitoring of multiple drugs in human plasma, with potential applications in routine clinical practice and pharmacokinetic studies.

Keywords: Therapeutic Drug Monitoring; UHPLC-PDA; Human Plasma; Tyrosine kinase Inhibitors; Triazole Antifungals; Bioanalytical Method Validation.

Community-Based Mobile Cancer Screening for Early Detection of Common Cancers: Experience from Rural Villages in Northern India

Dr. Suprakash Mandal

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Conclusion: Community-based mobile cancer screening model was feasible and effective for early detection. Apart from identifying individuals with suspected cancer and premalignant lesions, it also identified participants with cardiometabolic risk factors. Improving community participation and stronger referral may further enhance the impact.

ORAL PRESENTATION

Efficacy And Safety Of Virus-specific T-cell Therapy For Viral Infections Following Allogeneic Hematopoietic Stem Cell Transplantation: A Systematic Review And Meta-analysis

V. Pavithra, (HOD, Dept of Pharmacy Practice, KMCH COP)

Background: Viral infections remain a significant cause of morbidity and mortality following pediatric hematopoietic stem cell transplantation (HSCT). Virus-specific T-cells (VSTs) have emerged as a promising therapy for infections refractory to standard antivirals. We aimed to synthesize the evidence for VST efficacy and compare donor-derived vs. third-party products.

Methods: We systematically searched PubMed for studies published up to 2024 reporting on VST therapy in pediatric HSCT. Eligible studies included cohorts (n≥5) treating CMV, EBV, or ADV. A random-effects meta-analysis was performed to estimate the pooled overall response rate (ORR). Subgroup analysis was conducted to evaluate the impact of VST source (Donor-Derived vs. Third-Party).

Results: From 38 identified records, 12 studies met the inclusion criteria.

The pooled ORR across all pediatric cohorts was 0.68 (95% CI: 0.62–0.74). Subgroup analysis revealed no statistically significant difference in efficacy (p=0.52) between third-party "off-the-shelf" VSTs (ORR 0.69, 95% CI: 0.61–0.76) and donor-derived VSTs (ORR 0.64, 95% CI: 0.52–0.74). Heterogeneity between subgroups was low (I^2=0%).

Conclusion: VST therapy is a highly effective intervention for refractory viral complications post-pediatric HSCT. Importantly, third-party VSTs demonstrate comparable efficacy to donor-derived products, supporting their use as a rapid, accessible "off-the-shelf" treatment option in the pediatric setting.

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Anjana Madassery Vinod, (HOD, Dept of Pharmacy Practice, KMCH COP)

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Sharan kaarthick B. R. (HOD, Dept of Pharmacy Practice, KMCH COP)

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ORAL PRESENTATION

Identification of Potential DGKα Inhibitors Through Structure-Based Drug Repurposing and Computational Analysis

Akanksha Behera

Department of Biochemistry and Molecular Biology, Pondicherry University, Puducherry 605014, India

Diacylglycerol and phosphatidic acid play multiple oncogenic pathways and has been implicated in the progression of several malignancies, including melanoma, hepatocellular carcinoma, and glioma. The generation of above said signaling molecules are generated by Diacylglycerol kinase alpha (DGKα), which is a lipid kinase. Lack of an experimentally resolved three-dimensional structure of DGKα has limited the rational design and identification of selective inhibitors. In the present investigation, we have integrated structure-based computational approach to bring DGKα structure, further we identified FDA-approved drugs that could potentially be repurposed to target DGKα. A validated full-length DGKα model was employed for molecular docking, molecular dynamics simulations and computational alanine mutation screening, while the shortlisted compounds were evaluated for their pharmacokinetic and drug-likeness properties using ADMET analysis. Camostat mesilate exhibited the highest docking score (−8.034 kcal/mol), whereas acebutolol hydrochloride demonstrated superior conformational stability during molecular dynamics simulations with a docking score of −6.725 kcal/mol. Protein-ligand interaction analysis showed that structurally diverse compounds occupied a conserved catalytic binding pocket, while computational alanine mutation screening identified F407, V406, and L420 as conserved energetic hotspots essential for ligand stabilization across DGKα-ligand complexes. This investigation provided promising compounds with potential inhibitory activity against DGKα, providing a foundation for the development of novel therapeutic interventions and accelerating the translation of existing drugs for cancer treatment.

Computational Repurposing of FDA approved drugs as Potential DGKα Inhibitors for Cancer Chemotherapy Using Molecular Docking, Molecular Dynamics Simulations, and Identification of Hotspots by Alanine Mutation Screening

Abstract

Diacylglycerol kinase alpha (DGKα) is a key regulator of lipid signaling and oncogenic pathways, and has become a promising therapeutic target in Hepatocellular carcinoma (HCC), melanoma, glioma and other cancer types. However, the lack of a fully experimentally solved structure of DGKα has hampered structure-based drug discovery. Hence, the goal of this investigation was to develop full-length structure and identify potential DGKα inhibitors by using an in-silico drug repurposing strategy with FDA approved drugs. The full-length 3D structure was predicted and validated. Virtual screening, ADMET profiling, molecular docking, molecular dynamics (MD) simulations and computational alanine scanning were used to evaluate four FDA approved candidates such as acebutolol hydrochloride, pimavanserin, triamcinolone (kenacort), sotalol HCl and were compared to a known DGKα inhibitor, ritanserin. Among the repurposed candidates, Camostat mesilate showed the best docking score value (−8.034 kcal/mol), while Acebutolol hydrochloride was the best candidate with the docking score (−6.725 kcal/mol) and stable structural stability during molecular dynamics simulations. Alanine mutation screening and binding free energy analyses of mutated complexes revealed that F407, V406, and L420 are important conserved residues that play a significant role in ligand stabilization and DGKα-associated oncogenic signaling. Collectively, these findings provide a comprehensive structural framework for DGKα targeted drug discovery, identify acebutolol hydrochloride as a promising repurposable inhibitor, and establish a foundation for the rational development of next-generation targeted therapies against DGKα-driven cancers.

ORAL PRESENTATION

Detection of Lung Pathologies Using Chest Radiography in a Mobile Cancer Screening Program: Evidence from Rural Western Uttar Pradesh

Dr Anirudh Saxena

Background: Chest radiography incorporated into community-based screening programs can facilitate early identification of pulmonary and cardiothoracic abnormalities among underserved populations. We describe the findings of chest X-ray screening conducted through a mobile cancer screening unit in rural Northern India.

Methods: Portable chest radiography was performed as part of a community-based mobile cancer screening program conducted across 38 rural villages. Chest X-rays were interpreted by qualified radiologists, and abnormal findings were documented. Participants requiring further evaluation were referred to a tertiary care centre.

Results: Total 38 health camps were conducted in the different villages in the rural areas around the catering institute. Total of 2,311 villagers attended the screening camp with a mean (SD) age of 41.0 (16.8) years. Out of them 52.4% participants were male and 73.8% were aged ≥30 years. Nearly all participants (99.6%) had never been screened for cancers before. Among the participants recommended for chest radiography (n=59), emphysematous changes were the most common abnormality (n=15), followed by cardiomegaly (n=12), fibrotic lung changes (n=5), and pulmonary calcification (n=3). Tuberculosis (n=2), pleural effusion (n=2), unfolding of the aorta (n=2), solitary mass/nodule (n=3), left lung collapse (n=1), pneumonia (n=1), and boot-shaped heart (n=1) were also detected. All participants with clinically significant findings were referred for further diagnostic evaluation.

Conclusion: Community-based mobile chest X-ray screening can identify a broad spectrum of clinically important pulmonary and cardiothoracic abnormalities including cancer, supporting early referral and strengthening integrated rural health screening services.

Figure 1. Community-Based Chest X-ray Screening Pathway

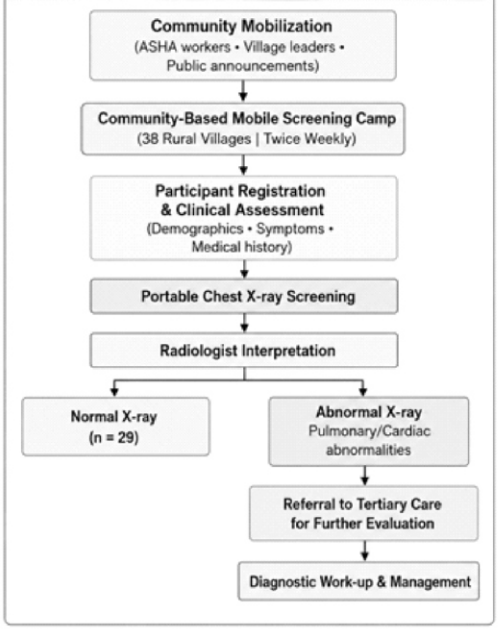
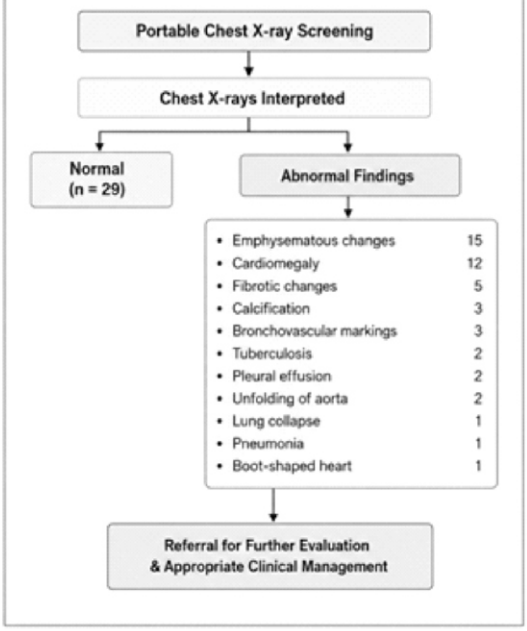


Figure 2. Chest X-ray Screening Outcomes



ORAL PRESENTATION

Neurological symptoms due to isolated brain metastasis in an elderly woman with otherwise resectable distal cholangiocarcinoma: a case report and review of literature

Aishwarya Prasad

Cholangiocarcinoma (CCA) is the second most common primary hepatic cancer, and usually presents as locally advanced disease. Isolated brain metastasis is very rare in CCA. We admitted a 70-year-old woman with progressive, painless, obstructive jaundice and mild pain, and, a palpable gallbladder. Investigations revealed a serum alkaline phosphatase (ALP)= 706 U/L, total bilirubin= 8.1 mg/dL, direct bilirubin= 4 mg/dL, and, serum CA 19-9= 20 U/mL. Triple-phase contrast CT abdomen revealed a "double-duct" sign and an enhancing distal common bile duct mass, without regional lymph nodes or ascites. During hospital stay, the patient developed grade 3 encephalopathy; all metabolic, toxic, septic causes were absent. CT brain showed an irregular, hypodense lesion in the right cerebrum and cerebellum with midline shift, suggestive of metastasis. After optimization, the patient was planned for systemic therapy but preferred hospice care. This is probably the second reported case of resectable dCCA with symptomatic isolated cerebral and cerebellar metastasis.

Introduction: Cholangiocarcinoma (CCA) is the second most common primary hepatic cancer, and, about 3% of all gastrointestinal malignancies.[1] Contemporary anatomical classification includes intrahepatic (iCCA, proximal to second-order bile ducts), perihilar (pCCA, between second-order bile ducts and cystic duct-common hepatic duct junction), and, distal (dCCA, between cystic duct and ampulla of Vater).[2] Indian series have reported a preponderance of proximal (intrahepatic-7% and perihilar-81%) CCA, as compared to about 12% distal CCA. The location of the disease is a decisive prognostic factor, with 67% of Indian patients with iCCA presenting with metastatic disease and only 57% overall being able to complete the intended treatment.[3] In the western world data, dCCA is more common and offers better prognosis mainly due to earlier detection. Nonetheless, all subtypes frequently present with locally advanced disease and lymph node spread rather than distant metastases. This is due to the well-defined directional lymphatic drainage around the biliary tree.[4,5]

Brain metastases in CCA have been reported only in 32 cases (6 case series) over the last three decades.[5-11] There is an invariable occurrence of prior metastases to the lung (66.7%), liver, chest wall, or bone.[10,12] Many of these reported cases have occurred in autopsy series or after completion of locoregional treatment. It is exceptionally rare to diagnose a symptomatic isolated brain metastasis in a resectable CCA awaiting surgical treatment. We present such a case in a 70-year-old woman.

Case report: A 70-year-old woman was admitted with a 3-month history of occasional right upper abdominal pain, jaundice, anorexia, weight loss (~10 kgs), and, recent-onset back pain over 15 days. No other history suggestive of infectious disease, or apparent distant metastases was elicited. Physical examination revealed deep icterus and a palpable, mobile, non-tender gallbladder. No ascites, lymphadenopathy, or, cardiovascular anomaly was detected. Laboratory analysis showed hemoglobin= 5 mg/dL, serum aspartate aminotransferase (AST)= 195 U/L, serum alanine aminotransferase (ALT)= 176 U/L, serum alkaline phosphatase (ALP)= 706 U/L (40–150U/L), total bilirubin= 8.1 mg/dL; direct bilirubin= 4 mg/dL, and, serum CA 19-9= 20 U/mL. Serum creatinine, serum glucose, serum albumin, serum amylase, and prothrombin time were normal. Ultrasound of the abdomen was suggestive of a dilated common bile duct (CBD: proximal 16 mm, mid-CBD= 12 mm, distal obscured) with an overdistended echo-free gallbladder. There was mild central and peripheral intrahepatic biliary radical dilatation (IHBRD). Contrast-enhanced CT abdomen revealed similarly dilated biliary ducts along with a prominent "double-duct" sign and a soft-tissue mass with arterial enhancement protruding into the distal CBD lumen. No regional lymph nodes or ascites were noted. (Figure 1,2) With a provisional diagnosis of distal cholangiocarcinoma, the patient was planned for curative resection (pancreatoduodenectomy) after systemic assessment and optimization.

On the third day of admission, the patient developed lethargy, altered consciousness and slurred speech. No focal neurological signs including cerebellar signs, were elicitable. There was no suggestion of septic or metabolic encephalopathy in the laboratory screening. The GCS deteriorated to 9 (E1V2M6) over 24 hours. An urgent plain CT of the brain revealed a well-defined, irregular-marginated, centrally hypodense lesion in the right cerebral hemisphere and cerebellum with perilesional edema, mass effect and midline shift. (Figure 3) The radiological impression was an intracranial metastasis. Fundoscopy revealed papilledema. The patient was administered oxygen therapy, intravenous dexamethasone and acetazolamide over the next 2 days, with partial improvement over the next week. An endoscopic biopsy of the primary biliary lesion suggested well-differentiated adenocarcinoma, and subsequent palliative chemoradiation was planned due to stage IV disease. The option of pancreaticoduodenectomy with whole brain irradiation was also discussed due to the resectable primary lesion and single isolated brain metastasis (oligometastatic disease). However, the patient's family opted to take the patient to a hospice center close to their residence.

Discussion: Neurological symptoms in a case of malignant jaundice may be multi-factorial. Our patient was a 70-year-old woman who presented with jaundice and developed mild slurring of speech, which progressed to Grade 3 encephalopathy. We could not satisfactorily attribute this finding to hepatic encephalopathy or kernicterus, since the serum bilirubin was only 8 mg/dL and serum ammonia was 25 μg/dL. Other parameters commonly implicated for metabolic encephalopathy like electrolytes, sugars, blood gases and renal biochemistry were normal. CT brain was sought initially suspecting intracranial/ subdural hemorrhage; however, it was a major surprise to find a metastatic lesion. Very few authors have reported or suggested performing brain imaging as standard metastatic work-up for a resectable biliary neoplasm, in the absence of symptoms.[6,10,12]

CCAs usually present at a locally advanced stage with a few cases amenable for curative surgical resection, that too, in specialized units.[7] Distant metastases are rare, especially brain metastases, and are more reported with intrahepatic type of cholangiocarcinoma (iCCA).[8,12] The striking rarity of our case was the occurrence of an isolated brain metastasis as a presenting feature of extrahepatic cholangiocarcinoma. To the best of our knowledge, only 3 cases from 2 authors have previously reported this feature.[13-16]

Fowler et al described a young woman with isolated sixth cranial nerve palsy, which was diagnosed by imaging as a large suprasellar mass. Total resection of the mass revealed a final histopathological diagnosis of adenocarcinoma, with a fibroblast growth factor receptor (FGFR)2-BICC1 gene mutation. A positron emission tomography (PET) scan later identified the primary as a large, locally advanced necrotic iCCA. The patient subsequently received palliative treatment, including FGFR-targeted therapy with pemigatinib.[13] Gravito-Soares et al reported an elderly man with a dCCA causing obstructive jaundice, along with symptomatic cerebellar metastasis. This patient was similar to our case, however, PET scan revealed adrenal and multiple bony metastases.[14] Rico et al identified five cases with neuro-ophthalmologic manifestations; of these, four patients had a known diagnosis of CCA, while one suffered a stroke from the hypercoagulable state of malignancy. They could confirm 3 cases with orbital biopsy.[15] William et al diagnosed a 51-year-old man with locally advanced iCCA, who received palliative chemotherapy. However, the patient was detected to have cerebral and leptomeningeal carcinomatosis one year later.[16] It is evident from the described cases that a surgically-resectable dCCA with isolated brain metastasis (especially cerebellar) is indeed very rare, and our case is unique in this regard. We can also infer that biopsy from both the primary and secondary (metastatic) site is important for immunohistochemical typing and to plan appropriate therapy. We could not offer these further options as the patient's family chose to pursue hospice care.

ORAL PRESENTATION

Structural and functional characterization of promoter G-quadruplex in AJUBA modulates transcription and EMT-associated phenotypes in Triple-negative breast cancer

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Triple negative breast cancer (TNBC), characterised by the absence of estrogen (ER), progesterone (PR) and HER2 receptors, is an aggressive subtype of breast cancer that possesses high metastatic potential, results in poor prognosis and has limited availability of therapeutic targets. An important aspect of this aggressive behaviour is the epithelial-to-mesenchymal transition (EMT), a physiological process linked to cytoskeletal remodelling and altered expression of N-cadherin (CDH2), a mesenchymal marker. In this context, AJUBA, a LIM-domain protein implicated in oncogenic signalling, has emerged as a potential upstream regulator of EMT due to its association with the Hippo signalling pathway, which negatively regulates LATS1 and promotes YAP/TAZ nuclear accumulation and TEAD-dependent transcriptional programs associated with proliferation. Based on this rationale, the present study focused on whether transcriptional regulation of AJUBA can be achieved by targeting the guanine-rich element on the promoter region.

The guanine base enrichment identified in the upstream regulatory region of AJUBA adopts a stable hybrid G-quadruplex conformation, validated by various biophysical assays, namely 1D 1H NMR, circular dichroism, and ThT-fluorescence. The ligand interaction and stabilization of the folded G4 with Pyridostatin (PDS) was examined by melting profile, isothermal titration calorimetry, and gel-shift assay. Stabilization of AJUBA G4 using Pyridostatin (PDS) resulted in a dose-dependent reduction of AJUBA transcripts in MDA-MB-231, a TNBC cell line, as quantified by qRT-PCR. A similar reduction in the expression of N-cadherin, a mesenchymal marker, indicates the attenuation of EMT-associated downstream signalling. In contrast, carboxy-Pydridostatin (cPDS), an analogue of PDS known to engage RNA G4, showed no significant change in the expression of AJUBA or N-cadherin, supporting a DNA-G4-mediated regulatory mechanism. The phenotypic alteration as a consequence of AJUBA and CDH2 repression was further evaluated by phalloidin staining, which revealed altered actin organization, together with reduced lamellipodia extensions visualized by SEM, and was consistent with the reduced migratory potential of the cells.

Collectively, these findings identify that the AJUBA-G4 can function as an exploitable regulatory element in TNBC. The study highlights a novel mechanism of regulating the oncogenic expression of AJUBA and its EMT-associated downstream effect via G4 stabilization, thereby advancing the DNA G4-mediated approach in regulating cancer progression.

Keywords: Triple-negative breast cancer, Metastasis, Transcriptional regulation, G-quadruplex, Pyridostatin, EMT.

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Diffuse large B-cell lymphoma (DLBCL) is a highly heterogeneous and aggressive malignancy, and its association with Epstein–Barr virus (EBV) infection contributes to distinct molecular alterations and poor prognosis. However, the role of ferroptosis, a regulated iron-dependent form of cell death, in EBV-positive DLBCL remains poorly understood. This research addresses this critical gap by integrating transcriptomic data and ferroptosis-related gene (FRG) networks to uncover potential diagnostic biomarkers and mechanistic insights. Differential expression analysis of two independent NCBI-GEO datasets comprising DLBCL and EBV-infected B cells, followed by integration with ferroptosis-related genes from the FerrDb database, identified 41 common differentially expressed ferroptosis-related genes (DE-FRGs). Functional enrichment (GO and KEGG) analyses revealed strong associations with iron–sulphur cluster binding, oxidoreductase activity, ferroptosis, glutathione metabolism, and p53 signalling, highlighting metabolic and redox imbalance as central mechanisms in EBV-mediated DLBCL progression.

Advanced machine learning models—support vector machine–recursive feature elimination (SVM-RFE) and Least Absolute Shrinkage and Selection Operator (LASSO) regression analysis—were applied to refine feature selection and identify key diagnostic markers. The integration of both models revealed 13 pivotal genes—TMBIM4, SLC7A11, BRD4, SLC38A1, MGST1, ATM, MT1G, LPIN1, PGRMC1, DPP4, ACO1, PDK4, and GPX4—with strong diagnostic performance. This study is the first to systematically identify ferroptosis-associated molecular markers in EBV-positive DLBCL, bridging the gap between viral oncogenesis and redox biology. These findings provide novel insights into the ferroptosis vulnerability of EBV-infected DLBCL cells, paving the way for targeted therapeutic strategies and precision diagnostics.

Keywords: Diffuse large B-cell lymphoma; Epstein–Barr virus; Ferroptosis; Machine learning; Biomarkers

ORAL PRESENTATION

5-Azacytidine Modulates the Variation in Genetic Signature of Sox4 and EpCAM Gene in Circulating Tumor Cells Isolated from Breast Cancer Patients using Flowcytometric Based Analysis

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Circulating tumor cells (CTCs) are the frontier area of medicine for early diagnosis of cancer patients. Flowcytometry is a highly sensitive technique used for quantitative analysis of epithelial - mesenchymal transition (EMT) markers expression of Sox4 and EpCAM using monoclonal antibodies in CTCs population, conjugated by PE (phycoerythrin) that detects more than 98.90 % score aftergating.Sox4, is an early transcription factor regulating various developmental process including EMT markers from pre to post metastasis during tumor progression of disease. The mean values of Sox4 showing variation between -ve mAbSox4 (7427.8) and +ve mAbSox4 (6482.3), while, highest frequency (40.00%) were observed between 51-60 years age-group and O.R (4.0) with confidence interval (C.I) at 95% varying between 1.074 -14.896 showing significant ($p<0.002$) differences. EpCAM mean values varying between –ve mAbEpCAM (22050.8) and +ve mAbEpCAM (21251.5) and highest frequency (40.00 %) were observed between 51-61 years age-groups. Significant ($p<0.003$) differences with O.R (2.0) and C.I at 95% that varying between 1.074 - 14.896, suggesting confirm signaling of Sox4 act as transition inducer for CTCs in BC patients. Sensitivity was further evaluated after in-vitro exposure (1.0 ugm/ml) to peripheral blood lymphocytes (PBL) with 5-azacytidine (5AzaC) and compare with controls, that showing increased mean CTCs population in 5AzaC +ve mAbSox4 (302397) and -ve mAbSox4 (251443).Statistical analysis showing observed values of O.R (1.11) and C.I at 95% varies between 0.225 - 5.46, showing lack of significance ($p<0.089$) differences but data of the EpCAM showing decreasing trend of cell population with significant ($p<0.001$) differences, suggesting, EpCAM marker seems to be more sensitive EMT marker during tumorogenesis in BC.

Conclusion : EpCAM seems to be more sensitive and reliable biomarkers for early diagnosis using in CTCs in breast cancer patients. However, our focus on functional aspects to open new avenues of research using short term tissue culture engineering for in vitro study. SOX4, stem cell expression still continue to reverse the transition process i.e from mesenchymal to epithelial by developing new inhibitors or antineoplastic agents to reduce the cellular toxicity and enhance the survival of the tumor bearing patients

Key Words: Circulating tumor cells, Breast Cancer, Epithelial Mesenchymal Markers: Flowcytometric analysis

POSTER PRESENTATION

Decoding the Anti-Glioblastoma Potential of Skimmianine Through Multi-Target Computational Profiling

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Background: Glioblastoma multiforme (GBM) remains the deadliest primary brain malignancy in adults, with a median overall survival of barely 14 to 18 months despite aggressive multimodal therapy. This reality makes the discovery of novel molecular targets and therapeutically promising candidates an urgent biomedical priority. Skimmianine which is a furoquinoline alkaloid mainly isolated from Skimmia japonica and related Rutaceae species consists of well-documented anti-inflammatory, antioxidant, and antiproliferative properties, yet its specific molecular mechanisms in GBM pathobiology have remained largely unexplored.

Methods: A systematic, multi-step network pharmacology framework was applied. GBM associated targets were retrieved from GeneCards (n = 8,268) and OMIM, while Skimmianine targets were predicted independently through SuperPred, SwissTargetPrediction, and ChEMBL. Venny 2.1 intersection analysis identified 72 common shared targets. A high-confidence protein-protein interaction (PPI) network was constructed via STRING (confidence ≥ 0.9) and subjected to topological analysis in Cytoscape 3.10.4 using the CytoNCA and MCODE plugins. KEGG pathway enrichment analysis was conducted at an adjusted $P < 0.05$. Molecular docking of Skimmianine against ten hub proteins which are - AKT1, EGFR, ERK1, ERK2, JAK2, MTOR, PD-L1, PI3K, STAT3, and VEGFR2 was carried out with the help of AutoDock Vina.

Results: PPI topology ranked AKT1, EGFR, ERK1, ERK2, JAK2, MTOR, PD-L1, PI3K, STAT3, and VEGFR2 as the ten foremost hub targets (node degree range 14 to 34). KEGG enrichment highlighted the Prolactin Signalling Pathway, PD-L1/PD-1 Checkpoint Pathway in Cancer, and Proteoglycans in Cancer as the most significantly enriched pathways, corroborating immunomodulatory and oncogenic signalling roles. Molecular docking confirmed favourable binding affinities ranging from −5.1 kcal/mol (PD-L1) to −7.3 kcal/mol (ERK1), with all ten complexes exhibiting thermodynamically stable interactions.

Conclusion: This study provides the first systematic computational evidence that Skimmianine may simultaneously modulate multiple potential GBM pathways including PI3K-AKT-mTOR signalling, JAK-STAT cascades, MAPK/ERK axes, angiogenic VEGFR2 networks, and immune-checkpoint regulation through PD-L1 thereby establishing a rigorous preclinical rationale for experimental validation of Skimmianine as a multi-target anti-GBM candidate.

Keywords: Glioblastoma multiforme; Skimmianine; Network pharmacology; Molecular docking; AKT1; EGFR; JAK2; mTOR; STAT3; VEGFR2; PD-L1; ERK; Furoquinoline alkaloid; PI3K-AKT-mTOR

POSTER PRESENTATION

Clinical Outcomes Following Adjuvant Radiotherapy for Intracranial Meningioma: A Single-Institution Retrospective Analysis

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Background: Surgical resection is the primary treatment for intracranial meningiomas. Adjuvant radiotherapy is frequently indicated for atypical, anaplastic, residual, or recurrent disease. We evaluated the early clinical outcomes of patients treated with Adjuvant RT at our institution.

Methods: This retrospective study included 20 patients with histologically confirmed intracranial meningioma who received Adjuvant Radiation. Baseline demographics, tumor, and treatment characteristics were reviewed, including age, sex, ECOG performance status, WHO grade, MRI-defined Pre-op volume, Post-op Residual tumor volume, Conformity index (CI), and heterogeneity index (HI). The primary endpoint was Local control (LC). Median Overall survival (OS) and Median progression-free survival (PFS) analyses with Acute Radiation related events were taken as Secondary Endpoints

Results: The median age was 43.5 years (IQR 33.5–55.5), with equal representation of males and females. Most patients had ECOG 1 (70%) and WHO Grade II tumors (75%). Among patients with available MRI volumetric data, 40% had tumors larger than 100 cc. The median CI and HI were 0.655 (IQR 0.36–0.76) and 1.07 (IQR 1.06–1.11), respectively. Local control was achieved in 17 of 20 patients (85%), whereas three patients (15%) developed local recurrence. Patients with recurrence were older (median age 57 vs. 39 years) and had a lower median residual tumor volume than those without recurrence.

Conclusion: Adjuvant RT provided encouraging early local control with acceptable treatment planning quality in our institutional cohort. Mature follow-up with Kaplan–Meier analysis of OS and PFS, together with evaluation of other prognostic factors, will further define long-term outcomes.

Dual-Method Immune Deconvolution Reveals Subtype-Specific PD-L1 Drivers and a Robust γδ T Cell–M2 Macrophage Axis in Breast Cancer

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Background: Most computational studies of the breast cancer immune microenvironment rely on a single deconvolution algorithm, risking method-specific blind spots — particularly for rare immune populations. Immune checkpoint blockade benefits in breast cancer remain largely confined to triple-negative disease, and the biology underlying differential PD-L1 utility across subtypes is incompletely understood.

Methods: We applied two complementary deconvolution approaches — xCell and a CIBERSORT-style ssGSEA using LM22 signatures with curated γδ markers (TRGC1, TRGC2, TRDC, TRDV2, TRGV9) — to 1,099 PAM50-classified primary tumours from TCGA-BRCA. Subtype differences, PD-L1–immune correlations, multivariate regression, and Cox survival models were assessed.

Results: PD-L1 expression varied significantly across subtypes (Kruskal–Wallis $p = 4.22 \times 10^{-9}$), highest in HER2-enriched and basal-like tumours. In basal-like disease, PD-L1 correlated strongly with CD8+ T cells ($\rho = 0.65$) and M1 macrophages ($\rho = 0.67$), consistent with IFN-γ-driven adaptive upregulation. HER2-enriched tumours showed simultaneous effector and regulatory associations, while luminal tumours were largely immune-quiet. The γδ T cell–M2 macrophage correlation was non-significant by xCell ($\rho = 0.048$) but strongly significant by CIBERSORT-ssGSEA ($\rho = 0.565$, $p < 2.2 \times 10^{-16}$), holding across all five subtypes and strongest in luminal B ($\rho = 0.59$). A multivariate model explained 49% of PD-L1 variance (adjusted $R^2 = 0.49$); Cox regression gave concordance 0.60.

Conclusions: Dual-method deconvolution uncovers a baseline γδ–M2 immunosuppressive circuit across breast cancer subtypes, particularly relevant in ER+ disease, and supports subtype-tailored immunotherapy strategies over uniform PD-L1-based selection.

Keywords: breast cancer; tumour immune microenvironment; PD-L1; CIBERSORT; xCell; γδ T cells; PAM50

POSTER PRESENTATION

The Role of Mitochondrial Ion Channels in the Evolution of Anticancer Drug Resistance

Ayush Kulshreshtha

Background: Surgical resection is the primary treatment for intracranial meningiomas. Adjuvant radiotherapy is frequently indicated for atypical, anaplastic, residual, or recurrent disease. We evaluated the early clinical outcomes of patients treated with Adjuvant RT at our institution.

Methods: This retrospective study included 20 patients with histologically confirmed intracranial meningioma who received Adjuvant Radiation. Baseline demographics, tumor, and treatment characteristics were reviewed, including age, sex, ECOG performance status, WHO grade, MRI-defined Pre-op volume, Post-op Residual tumor volume, Conformity index (CI), and heterogeneity index (HI). The primary endpoint was Local control (LC). Median Overall survival (OS) and Median progression-free survival (PFS) analyses with Acute Radiation related events were taken as Secondary Endpoints

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Sebaceous Carcinoma of the left lower eyelid:

Dr. Tanya Soni

Sebaceous carcinoma of the eyelid is a rare but highly aggressive malignancy arising from sebaceous glands, most commonly the meibomian glands. Owing to its ability to mimic benign inflammatory conditions, diagnosis is frequently delayed, increasing the risk of local invasion and recurrence. We report the case of a 38-year-old female who presented with a lesion involving the left lower eyelid. Radiological evaluation with contrast-enhanced CT and orbital MRI demonstrated a localized enhancing soft-tissue lesion in the medial aspect of the left lower eyelid (10x14x10mm) without intraorbital or intracranial extension. The patient underwent wide local excision with regional flap reconstruction. Histopathological examination revealed a moderately differentiated sebaceous carcinoma measuring 1.3 × 0.7 cm, characterized by sebaceous differentiation, infiltrative growth pattern, moderate nuclear pleomorphism,

Depth of invasion- 11mm, focal necrosis, and lymphovascular invasion, with tumour-free surgical margins and no nodal involvement. Given the presence of high-risk pathological features, adjuvant external beam radiotherapy was administered to a total dose of 60 Gy in 30 fractions using three-dimensional conformal radiotherapy (3DCRT). Treatment was well tolerated, with only Grade II dermatitis and radiation conjunctivitis, both of which resolved on 1st follow-up. Post-treatment imaging (MRI Head and Neck) at 3 month follow up demonstrated postoperative changes without evidence of residual or recurrent disease at post-operated bed. This case highlights the importance of early diagnosis, accurate histopathological assessment, complete surgical excision, and appropriately selected adjuvant radiotherapy in achieving durable local control and minimizing recurrence in sebaceous carcinoma of the eyelid.

POSTER PRESENTATION

A Disquisition into Pre-Clinical Diagnostic Delay In Breast Cancer related symptoms through a Tactile Play-based Educational Toolkit

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Breast cancer remains the leading cause of cancer-related mortality among women globally. Survival outcomes are strongly stage-dependent: five-year survival exceeds 90% when disease is detected at a localized early stage but declines to below 30% in cases diagnosed with distant metastasis. In India, a significant proportion of patients present at Stage II or III disease, where survival outcomes are substantially poorer compared to early-stage detection.

Infrastructural barriers such as screening access and affordability have been widely addressed, diagnostic delay frequently originates in the pre-clinical interval, defined as the period between first symptom recognition and initial medical consultation. Studies across resource-constrained countries report patient-related delays ranging from weeks to several months, often attributed to limited symptom literacy, stigmas and sociocultural barriers. For the Indian context, female reproductive health and breast-related discussions are frequently avoided, contributing to concealment or dismissal of early warning signs such as palpable lumps, nipple retraction, skin dimpling or persistent focal pain.

This project explores whether play-based, physical artefacts can function as an accessible medium for improving early symptom recognition and health communication among non-experts. Using a research-through-design methodology, interviews with oncologists, gynaecologists and educators practicing methods of embodied learning, the research resulted in the informed development of a Tactile Physical toolkit that abstracts the symptom patterns and disease progression without encouraging self-diagnosis.

The artefact is intended for strategic deployment in specific contexts like clinic waiting areas (gynaecology and otherwise), community health settings, workshops to facilitate informal engagement and encourage conversation around the topic.

Preliminary user interactions indicate improved recall of key symptom indicators and increased willingness to engage in peer discussion following interaction with the artefact. By targeting behavioural delay within the pre-clinical phase, this intervention proposes a complementary strategy to conventional screening programs, aiming to reduce stigma, enhance symptom literacy and encourage earlier clinical presentation.

By reframing health education through play,the project contributes to discussions on behavioural delay in cancer care and proposes a scalable, low-cost educational model as a complementary strategy for early breast cancer awareness.

Keywords: Breast Cancer, Early Detection, Symptom Recognition, Behavioural Delay, Tactile Learning, Toys & Toolkits, Health Literacy

POSTER PRESENTATION

Role of Adjuvant Radiotherapy in Extraskeletal Myxoid Chondrosarcoma of the Temporal Bone–Parotid Region: A Rare Case Report

Dr. Mehank D Patel

Background: Extraskeletal myxoid chondrosarcoma (EMC) is a rare soft tissue sarcoma with an indolent but locally recurrent behavior. Occurrence in the temporal bone–parotid region is exceedingly uncommon, and optimal management remains challenging because of the proximity to critical neurovascular structures. I present a rare case highlighting the role of adjuvant radiotherapy in local disease control.

Case Presentation: A 62-year-old male presented with left ear pain, hearing impairment, and facial palsy. HRCT temporal bone demonstrated an expansile destructive lesion involving the mastoid air cells, external auditory canal, and temporal bone. MRI revealed an avidly enhancing mass measuring 4.7 × 2.9 × 4.0 cm extending into the middle ear, mastoid region, and deep lobe of the parotid gland, with suspected involvement of the mastoid segment of the facial nerve. Initial biopsy suggested pleomorphic adenoma; however, definitive histopathological examination following surgical excision revealed extraskeletal myxoid chondrosarcoma with chondromyxoid matrix and bony trabecular destruction. Facial nerve tissue was free of tumor infiltration.

Treatment and Outcome: Considering the locally aggressive nature of the tumor, skull base involvement, bony erosion, and risk of microscopic residual disease, the patient was referred for adjuvant radiotherapy. Image-guided radiotherapy (IGRT) was planned to deliver a dose of 60–66 Gy to the postoperative tumor bed while respecting dose constraints to adjacent critical structures. The patient tolerated treatment well with manageable acute toxicities. At follow-up, local control was maintained with no evidence of disease progression and symptomatic improvement.

Conclusion: EMC of the temporal bone–parotid region is exceptionally rare. Complete surgical excision remains the primary treatment; however, adjuvant radiotherapy plays an important role in improving local control, particularly in cases with skull base extension, close margins, or potential residual disease. Multidisciplinary management is essential to achieve favorable oncological outcomes.

Keratin Fusions Reprogram Tumor Cell Identity and Immune Editing Trajectories in Oral Squamous Cell Carcinoma

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Intratumoral heterogeneity is a central obstacle in oral squamous cell carcinoma (OSCC), yet the cytoskeletal determinants that instruct tumor cell identity and immune escape remain largely undefined. Keratin fusion events, traditionally considered structural anomalies, may instead function as active regulators of malignant plasticity. Previously, we identified t(12;17)(q13.13;q21.2) chromosomal rearrangement as the most common fusion event in OSCC, resulting in a variety of inter-keratin fusions. Here, we interrogate keratin fusion-driven transcriptional reprogramming at single-cell resolution to define its role in tumor evolution and immune editing. Using single-cell RNA sequencing of keratin fusion (K6/K14)-derived OSCC subclones, we resolve discrete tumor cell states distinguished by inflammatory signaling capacity, proliferative dominance, and stress-response activation. Fusion-associated clusters exhibit divergent transcriptional architectures: one is enriched for chemokine and cytokine modules linked to macrophage modulation and immune engagement, and proliferative acceleration, chromatin remodeling signatures, and attenuated inflammatory output characterize the other. Trajectory inference reveals state transitions, suggesting that keratin fusion may govern adaptive shifts in tumor fitness under immune pressure. Pathway enrichment analyses demonstrate differential activation of DNA damage-associated signaling, interferon-stimulated gene networks, and epithelial-mesenchymal plasticity programs. Notably, ligand-receptor inference predicts fusion-dependent rewiring of tumor-macrophage communication axes, implicating chemokine gradients and innate immune signaling in microenvironment remodeling. These findings position keratin fusion as a regulatory hub linking cytoskeletal perturbation to transcriptional identity and immune ecosystem restructuring. Collectively, our data challenge the view of keratin alterations as passive structural events and instead identify keratin fusion as a driver of tumor state plasticity and immune editing trajectories in OSCC.

Keywords: Keratin fusion, Oral squamous cell carcinoma (OSCC), Single-cell RNA sequencing, Tumor cell plasticity, Immune editing

POSTER PRESENTATION

In-vitro Analysis of MALAT1, PANDAR, RNU44, POU3F3, and LINCOR Long NonCoding RNAs in Oral Squamous Cell Carcinoma: Exploring lncRNA Biomarkers for Early Detection and Risk Stratification in Tobacco-Associated OSCC

Priyanshi Singh

Background: Oral squamous cell carcinoma (OSCC) is the most common malignancy of the oral cavity and remains a major public health challenge, particularly in populations with high tobacco consumption. Delayed diagnosis contributes significantly to poor clinical outcomes and reduced survival. Long non-coding RNAs (lncRNAs) have emerged as important regulators of carcinogenesis and may serve as promising biomarkers for early detection and prognostic assessment.

Methods: A case-control observational study was conducted involving 60 histopathologically confirmed OSCC patients and 30 healthy controls. Tumor tissue specimens and normal oral mucosal samples were collected following informed consent and institutional ethical approval. Total RNA was isolated using the TRIzol method, converted to cDNA, and the expression of MALAT1, PANDAR, RNU44, POU3F3, and LINCOR was quantified using quantitative real-time PCR (qRT-PCR). Relative expression was calculated using the 2^-ΔΔCt method with appropriate endogenous normalization. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic performance of each lncRNA.

Results: All five lncRNAs demonstrated significant overexpression in OSCC tissues compared with healthy controls ($p < 0.001$). MALAT1 exhibited the highest fold change, followed by POU3F3, PANDAR, RNU44, and LINCOR. Increased lncRNA expression was associated with tobacco exposure and adverse clinicopathological characteristics. ROC curve analysis showed excellent discriminatory ability, with MALAT1 achieving the highest diagnostic accuracy (AUC ≈ 0.99), while the combined five-lncRNA panel further improved diagnostic performance.

Conclusion: MALAT1, PANDAR, RNU44, POU3F3, and LINCOR are significantly dysregulated in OSCC and demonstrate strong potential as molecular biomarkers for early diagnosis and risk stratification, particularly in tobacco-associated disease. A multi-lncRNA biomarker panel may enhance the accuracy of OSCC detection and facilitate earlier clinical intervention. Larger multicentric studies are warranted to validate these findings and support future clinical translation.

POSTER PRESENTATION

Elucidating interaction of HMGB1 as Ligand with EGFR1 receptor to activate MAP-kinase a comparative study with EGF-Ligand and an in silico study

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High Mobility Group Box 1 (HMGB1) that acts as a cytokines and DNA binding biomolecules interacts with Epidermal Growth Factor Receptor (EGFR1) are critical, often interconnected, factors involved in cancer, tumor progression, and metastasis. HMGB1 that is known to be secreted to the extracellular environment interact with TLR4 and RAGE activating different TFs to lead different signal transduction pathway. The 3D-structure of EGFR1 (PDB-ID: 3NJP) and HMGB1 (PDB-ID: 8I9M) is retrieved and molecular docking was performed using ClusPro2.0, complex with higher binding interaction affinity was selected for Molecular Dynamic Simulation (MDS) based trajectory analysis. MDS was further confirmed for stable binding interaction and structural stability over 25ns simulation time.

In current study we predicted, HMGB1 interacted with EGFR1 of Domain-I and Domain-III strongly, same Domain site where present in the bio-molecule EGF that binds to EGFR1 leading to the dimerization and downstream activation of MAP-kinase signal transduction pathway activating different kinases. It was observed that many AAs such as Asn12, Thr15, Gln16, Gly18, Asp22, Glu90, Ser356, Gln384, Phe357, and His409 are common residues of the both EGF and HMGB1 that interacted with EGFR1 receptor. This overall computational study suggests the possible role of HMGB1 other than EGF protein in dimerization and activation of EGFR1 protein leading to activation of MAP-kinase signal transduction pathway.

Key words: EGFR1, EGF, HMGB1, cancer, MAP-kinase

Microbial contamination of Dohra: Edible areca nut product

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Background: Dohra is a mixture of areca nut, lime, kattha, clove, cardamom, as well as peppermint. Few other flavouring agents are added to enhance its taste. It is locally produced and sold in the local market of Jaunpur, Prayagraj and Pratapgarh district of Uttar Pradesh (U.P.), India. Usually it is packed in loose polythene, and sold in market without any warning sign. Mostly the poor people are involved in this business that neither register product, nor pay any form of duties to the government. The users of this preparation are mostly Gen Z who become addicted without knowing its adverse effects on their body, as it does not contain any warning signs. This study aims to study the microbial contamination in Dohra via microbial lab-cultures.

Methods: Different forms of Dohra were collected from Prayagraj districts of U.P., India. The dilution of Dohra was set by adding 1 gm of Dohra in 10 mL of normal saline, and kept on a rotator for 1 hour, followed by centrifugation at 1500 rpm for 5 minutes. 100 µL of dilution is used for lawn culture on MacConkey media. The cultures were observed at 24, 48 and 72 hours. If growth were observed, the confirmatory biochemical tests were performed.

Results: Several pathogenic bacteria were found in Dohra of which predominant were Coagulase negative Staphylococci (CoNS), Staphylococcus, as well as Enterobacteriaceae.

Conclusions: Dohra is having pathogenic bacteria; hence, its use, production and sale should be regulated. A strict regulatory policy regarding the use, production and sale of Dohra preparation is required.

Keywords: Areca nut, catechu, Dohra, policy, tobacco

POSTER PRESENTATION

Decoding the Anti-Glioblastoma Potential of Skimmianine Through Multi-Target Computational Profiling

Somya Gupta

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Background: Glioblastoma multiforme (GBM) remains the deadliest primary brain malignancy in adults, with a median overall survival of barely 14 to 18 months despite aggressive multimodal therapy. This reality makes the discovery of novel molecular targets and therapeutically promising candidates an urgent biomedical priority. Skimmianine which is a furoquinoline alkaloid mainly isolated from Skimmia japonica and related Rutaceae species consists of well-documented anti-inflammatory, antioxidant, and antiproliferative properties, yet its specific molecular mechanisms in GBM pathobiology have remained largely unexplored.

Methods: A systematic, multi-step network pharmacology framework was applied. GBM associated targets were retrieved from GeneCards (n = 8,268) and OMIM, while Skimmianine targets were predicted independently through SuperPred, SwissTargetPrediction, and ChEMBL. Venny 2.1 intersection analysis identified 72 common shared targets. A high-confidence protein-protein interaction (PPI) network was constructed via STRING (confidence ≥ 0.9) and subjected to topological analysis in Cytoscape 3.10.4 using the CytoNCA and MCODE plugins. KEGG pathway enrichment analysis was conducted at an adjusted $P < 0.05$. Molecular docking of Skimmianine against ten hub proteins which are - AKT1, EGFR, ERK1, ERK2, JAK2, MTOR, PD-L1, PI3K, STAT3, and VEGFR2 was carried out with the help of AutoDock Vina.

Results: PPI topology ranked AKT1, EGFR, ERK1, ERK2, JAK2, MTOR, PD-L1, PI3K, STAT3, and VEGFR2 as the ten foremost hub targets (node degree range 14 to 34). KEGG enrichment highlighted the Prolactin Signalling Pathway, PD-L1/PD-1 Checkpoint Pathway in Cancer, and Proteoglycans in Cancer as the most significantly enriched pathways, corroborating immunomodulatory and oncogenic signalling roles. Molecular docking confirmed favourable binding affinities ranging from −5.1 kcal/mol (PD-L1) to −7.3 kcal/mol (ERK1), with all ten complexes exhibiting thermodynamically stable interactions.

Conclusion: This study provides the first systematic computational evidence that Skimmianine may simultaneously modulate multiple potential GBM pathways including PI3K-AKT-mTOR signalling, JAK-STAT cascades, MAPK/ERK axes, angiogenic VEGFR2 networks, and immune-checkpoint regulation through PD-L1 thereby establishing a rigorous preclinical rationale for experimental validation of Skimmianine as a multi-target anti-GBM candidate.

Keywords: Glioblastoma multiforme; Skimmianine; Network pharmacology; Molecular docking; AKT1; EGFR; JAK2; mTOR; STAT3; VEGFR2; PD-L1; ERK; Furoquinoline alkaloid; PI3K-AKT-mTOR

POSTER PRESENTATION

Decoding the Anti-Glioblastoma Potential of Skimmianine Through Multi-Target Computational Profiling

Sukriti Maurya

Department of Forensic Science, School of Basic and Applied Sciences, SGT University, Gurugram, Haryana, 122505, India

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Keywords: Glioblastoma multiforme; Skimmianine; Network pharmacology; Molecular docking; AKT1; EGFR; JAK2; mTOR; STAT3; VEGFR2; PD-L1; ERK; Furoquinoline alkaloid; PI3K-AKT-mTOR

POSTER PRESENTATION

Looking Beyond Conventional Imaging: A Vascular Gallbladder Polyp Unmasked an Occult Gallbladder Malignancy

Dr Shweta Dube

Background: Early gallbladder malignancy frequently eludes diagnosis because patients often present with non-specific symptoms, normal laboratory investigations and equivocal cross-sectional imaging. Identifying subtle radiological clues that warrant oncologic intervention remains a clinical challenge.

Case Presentation: A 61 year old woman presented with vague right upper quadrant pain and reduced appetite. Liver function tests, CEA and CA19-9 were normal. Initial contrast-enhanced CT demonstrated only focal asymmetric gallbladder wall thickening without definite evidence of malignancy. As symptoms persisted, Doppler ultrasonography was performed and revealed a vascular gallbladder polyp measuring over 3 cm, raising suspicion for neoplasia. A repeat CT performed remained essentially unchanged. Despite the absence of convincing biochemical or CT findings, the Doppler finding, together with persistent clinical suspicion, prompted an extended cholecystectomy.

Histopathology demonstrated intracholecystic papillary neoplasm with associated well-differentiated invasive adenocarcinoma (pT1b). Completion radical cholecystectomy with regional lymphadenectomy revealed no residual tumour or nodal metastasis. The patient remains disease-free on follow-up.

Discussion: This case illustrates the potential discordance between pathology and conventional imaging in early gallbladder malignancy. While CT, tumour markers and cytology failed to establish the diagnosis, Doppler-demonstrated intralesional vascularity emerged as the pivotal finding influencing surgical decision making. The case reinforces the importance of integrating multimodality imaging with clinical judgment when evaluating indeterminate gallbladder polyps.

Conclusion: Vascularity within a gallbladder polyp should not be overlooked, even in the setting of reassuring laboratory parameters and equivocal CT findings. Early recognition of this subtle imaging feature may facilitate timely oncologic resection and diagnosis at a potentially curable stage.

Emerging Molecular Biomarkers and Advanced Diagnostic Strategies for HPV-Associated Oral Squamous Cell Carcinoma: Current Perspectives and Future Opportunities

Avantika Dwivedi

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Because of its high frequency, delayed detection, and poor prognosis, oral squamous cell carcinoma (OSCC), which makes up almost 90% of all oral cancers, continues to be a significant public health concern. High-risk human papillomavirus (HPV), especially HPV-16, appears to play a major role in oral carcinogenesis, especially in younger people and non-smokers, although tobacco use, alcohol consumption, and chewing betel quid remain the main etiological factors. Tumor suppressors p53 and retinoblastoma (Rb) are degraded by HPV-mediated expression of the viral oncoproteins E6 and E7, which causes unchecked cell division, genomic instability, and malignant transformation. A number of promising biomarkers, such as HPV DNA, p16, epidermal growth factor receptor (EGFR), TP53 alterations, circulating nucleic acids, salivary proteins, and immune-associated biomarkers, have been discovered recently in molecular oncology. These biomarkers offer improved opportunities for treatment stratification, prognostic evaluation, and early diagnosis. Although conventional histopathology and tissue biopsy remain the gold standards for diagnosing oral cancer, new technologies such as liquid biopsy, salivary diagnostics, next-generation sequencing, immunohistochemistry, CRISPR-based molecular assays, artificial intelligence-assisted image analysis, and nanotechnology-enabled biosensors are transforming the field into one that is less invasive and more sensitive. Customized treatment approaches and real-time sickness monitoring are supported by these instruments. Additionally, it is anticipated that patient-specific therapeutic decision-making would be enhanced by the integration of molecular biomarkers with cutting-edge computational techniques and precision medicine frameworks. Despite so much significant progress in this area there are still issues with the large-scale clinical use, standardization of the diagnostic processes, and the biomarker validation before the researchers which is very complex propbelm. The future interdisciplinary research which will combining the molecular biology, nanotechnology and bioinformatics and also the machine learning will be also be necessary to integrate these outstanding developments into the routine clinical practices which is desired. This review of these will also highlights the current knowledge of the HPV-associated OSCC further recent advancements in the biomarker discovery and also the next-generation diagnostic technologies that may improve the prognosis, early diagnosis, and the precision oncology.

Keywords: Oral Squamous Cell Carcinoma (OSCC), Human Papillomavirus (HPV), HPV-16, Molecular Biomarkers, p16, EGFR, Precision Medicine, Liquid Biopsy, Salivary Biomarkers, CRISPR Diagnostics, Artificial Intelligence, Nanobiosensors, Early Cancer Detection, Personalized Therapy

POSTER PRESENTATION

Differential Expression of AGL and FLT4 Gene in Saliva of Oral Squamous Cell Carcinoma Patients

Priyanka Singh

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Oral Squamous Cell Carcinoma (OSCC) represents a significant health challenge for Clinicians and Scientist to explore the etiopathology in India. Bihar shows high prevalence rates are compounded by later-stage during clinical presentations. Conventional diagnostic protocols is invasive tissue biopsies, that causes delay histopapathologically during diagnosis Present study includes non-invasive liquid biopsy approach by evaluating the DNA methylation status of using two genes, AGL and FLT4, as biomarker from the saliva samples.

Epigenetic alterations, such as promoter hyper methylation, are recognized as early molecular events in carcinogenesis that precede visible physical symptoms. This study includes both saliva and oral swab samples will be collected and preserved in a Tris-NaCl-SDS stabilization buffer to maintain genetic integrity. Following genomic DNA isolation and cDNA synthesis, the methylation-linked expression profiles of AGL (a tumor suppressor) and FLT4 (a lymph genic regulator) will be quantified using SYBR Green-based Quantitative using PCR (qPCR).

This study aims to investigate the DNA methylation-associated molecular profiles of AGL and FLT4 gene in saliva as potential non-invasive biomarkers for the early detection and their clinical significations. These two genes showing differential expression because of different functional nature during tumorogenesis.By correlating the findings, salivary methylation signatures of these genes with clinical stages of OSCC, this study is cost-effective screening for early detection. Ultimately, this research seeks to provide a molecular framework that facilitates timely diagnosis and improves the prognosis for oral cancer patients in high-risk population of Bihar has not been known earlier. Hence, present study is novel study for early diagnosis in the patients for early diagnosis.

Keywords: Oral Squamous Cell Carcinoma: Saliva: DNA Methylation: AGL: FLT4: Liquid Biopsy

POSTER PRESENTATION

Analysis of ssDNA Aptamers Using Computational Tools for the Selective Recognition of Prothrombin Fragment 1.2 as a Biomarker for Cancer-Associated Thrombosis

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Cancer-associated thrombosis (CAT) is a main reason for morbidity and mortality in cancer patients, highlighting the requirement for sensitive biomarkers that can detect hypercoagulable states before the development of clinical thrombotic events. Among thrombin-generation biomarkers, Prothrombin Fragment 1.2 (PF1.2) is generated during the conversion of prothrombin to thrombin in the clotting cascade, making it a potential indicator of in vivo thrombin generation. According to recent evidence, PF1.2 outperforms other coagulation biomarkers for cancer-associated venous thromboembolism (VTE). Parallel advances in aptamer technology have identified aptamers (single-stranded oligonucleotides) as promising alternatives to antibodies for diagnostic applications because of their high specificity, chemical stability, ease of synthesis, low immunogenicity, and compatibility with biosensing platforms. This study presents a computational approach to screen an aptamer with high affinity and specificity towards PF1.2. The PF1.2 structure was initially modelled and validated using in silico tools. Ten aptamers that target proteins with a similar domain to PF1.2 were selected from the literature, and their secondary and tertiary structures were predicted. The generated aptamer models were used in molecular docking with PF1.2 to evaluate binding affinity. To investigate target specificity, a cross-reactivity study was performed with other plasma proteins through docking using the HDOCK server. Overall, this study uses a computational approach for PF1.2 structure modeling, aptamer screening, and docking to reduce the experimental time and cost. This in silico framework provides valuable insights for the development of specific aptamer-based biosensors and diagnostic assays to monitor thrombin generation and cancer-associated thrombosis.

Keywords: Prothrombin Fragment 1.2, Cancer, Aptamer, Computational approach, Molecular docking, Biosensor, Diagnostic

CD6 as a Diagnostic and Prognostic Biomarker in T-Cell Acute Lymphoblastic Leukemia (T-ALL): Structural Architecture, Signaling Dynamics, and Clinical Perspectives

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CD6 (Cluster of Differentiation) is a type I transmembrane glycoprotein belonging to the SRCR SF (Scavenger receptor cystine-rich domain superfamily), majorly expressed on T-cells and also expressed on some fraction of B-cells and NK cells. It is a 105-130 kDa protein encoded by the CD6 gene on human chromosome 11q13. Structurally, it comprises three extracellular SRCR domains (D1, D2, and D3), a transmembrane region, and an intracellular cytoplasmic tail rich in phosphorylatable serine, threonine, and tyrosine residues. CD6 is known to interact with the T-cell receptor (TCR)-CD3 complex to modulate T-cell activation, adhesion, and survival. Various known ligands of CD6 are- CD166, its D1 domain interacts with the D3 domain of CD6, CD318/CDCP1 to domain D1 of CD6, Galectins-1/3, and CD44. The cytosolic tail of CD6 lacks catalytic activity, but upon engagement, CD6 induces downstream signaling via ZAP-70, SLP-76, and the MAPK/ERK cascades. Alternative RNA splicing-regulated by splicing factors such as SRSF1-generates diverse full-length and exon-skipped isoforms (e.g., CD6a, CD6b, CD6c, CD6d, CD6e, CD6Δd3 and CD6f), regulating functional activation thresholds. CD6 is associated with various diseases, including autoimmune diseases such as multiple sclerosis, psoriasis, and rheumatoid arthritis. It is also known to be associated with various types of cancers; primarily, the role of CD6 has been studied with respect to solid tumors, and CD6 surface expression has been extensively studied in various types of blood cancers like CLL (Chronic Lymphocytic leukemia), T-cell lymphomas, but its role in T-ALL (T-cell lymphoblastic Leukemia) remains unclear. Among various types of leukemia, T-ALL is not well classified, while the classification and targeted therapies for B-ALL have advanced significantly. Studies related to the expression of CD6 could be used in diagnosis and prognosis for T-ALL. Harnessing CD6 as a biomarker paves the way for targeted therapeutic interventions, including anti-CD6 monoclonal antibodies.

Keywords: CD6; SRCR; CD166; T-ALL.

POSTER PRESENTATION

Look-Alike Cells, Different Outcomes: A Case of Hematogone Hyperplasia mimicking Acute Lymphoblastic Leukemia

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Introduction: Hematogones are benign B-lymphoid precursors commonly seen in the bone marrow, particularly in response to viral infections (e.g. AIDS,CMV), chemotherapy or marrow injury. Their morphological similarity to lymphoblasts often leads to diagnostic confusion with acute lymphoblastic leukemia (ALL).

Methods: A58-year-old female presented to medicine OPD with complaints of fever, generalized weakness and epistaxis for 15 days. There was a past history of tuberculosis 8 year ago. On examination, petechiae were noted over bilateral upper limb and lower limb along with pallor. No organomegaly was seen. The patient underwent thorough clinical and laboratory evaluation. Investigations included complete blood count with peripheral blood smear, bone marrow aspiration and biopsy, immunohistochemistry application and screening for infection markers.

Results: The complete blood count with peripheral smear revealed pancytopenia (Hb 6.9 g/dL, TLC 630/µL, platelets <10,000/µL). Bone marrow aspirate smears were markedly hemodiluted, while biopsy showed sheets of CD34 positive cells, initially raising suspicion for acute leukemia. However, further work-up with immunohistochemistry demonstrated positivity for CD34, TdT, CD19, with a pattern favoring hematogones rather than leukemic blasts. HSV and Parvovirus B19 assay were positive. The absence of organomegaly and absence of relevant immunophenotypic markers further supported hematogone hyperplasia instead of acute lymphoblastic leukemia.

Conclusion: Hematogones closely mimic lymphoblasts morphologically and can pose a diagnostic dilemma. Accurate distinction requires clinical correlation, immunophenotyping, and Flow Cytometric evaluation to avoid misdiagnosis. Usually hematological follow-up and confirmation with flow cytometry are required for a definitive diagnosis This case highlights the significance of Immunohistochemistry for differentiation of hematogones from leukemis blasts, even when Flow Cytometry evaluation was not available in this case.

Key words: Hematogones, Acute lymphoblastic leukemia, Pancytopenia, Immunohistochemistry

ORAL PRESENTATION

Epidemiology and Survival Trends of Adenocarcinoma of Unknown Primary in New Jersey

Simran Aulakh, Joseph Peters, Jonique Depina, Kyle Miller, Mridu Uprety, Dipika Jayachander, Simcha Weissman

Background: Adenocarcinoma of unknown primary (AUP) is a common histologic subset of cancers of unknown primary with historically poor outcomes. AUP can be precluded by a sequalae of painful paraneoplastic syndromes. Contemporary, state-level data are sparse and lack high powered studies, particularly focusing on non-clinical variables such as demographics and ethnicity. By way of this large retrospective analysis, we aim to characterize temporal patterns, demographics, treatment utilization, and survival for AUP in New Jersey. These data will ultimately help identify factors associated with cancer-specific mortality (CSM) and further reduce overall mortality (OM).

Methods: We conducted a population-based study using SEER (New Jersey) data from 2000–2022. Adults (≥18 years) with AUP (ICD-O-3: C80.9; adenocarcinoma histologies) were included; death-certificate/autopsy-only cases and records with missing key variables were excluded. Baseline characteristics were summarized descriptively. Fine–Gray competing risks models estimated sub-distribution hazard ratios (sHRs) for CSM; Cox regression estimated hazard ratios (HRs) for OM. Multivariable models adjusted for age, race/ethnicity, marital status, county-level income, urbanicity, year of diagnosis, and receipt of chemotherapy, radiotherapy, and surgery.

Results: Among 4,167 patients, 75.1% were non-Hispanic White, 12.8% non-Hispanic Black, and 9.1% Hispanic; 79.7% were aged ≥60 years. Most resided in highly urban counties (86.5%) and nearly half in ≥$100,000 income counties. Chemotherapy was given to 24.3%, radiotherapy to 11.8%, and surgery was recorded in 90.8% (largely diagnostic). In crude analyses, age was the strongest predictor of mortality (vs 0–39 years: CSM HR 1.66 for 60–79; 2.06 for ≥80; both $p<0.001$). Hispanic and "Other" groups had lower CSM than non-Hispanic Whites, while widowed status predicted higher mortality. In multivariable models, older age remained independently associated with higher CSM and OM (e.g., ≥80 years: CSM HR 1.81; OM HR 1.84; both $p<0.001$). Chemotherapy independently reduced mortality (CSM HR 0.88; OM HR 0.85; both $p<0.001$), whereas surgery and radiotherapy showed no survival benefit. Mortality modestly improved in the late 2000s but rebounded in 2022.

Conclusion: In New Jersey, AUP predominantly affects older adults, with persistent sociodemographic disparities and limited therapeutic gains. Systemic therapy remains the principal modifiable factor linked to improved survival. Thus, thus can prevent AUP the sequalae of painful paraneoplastic syndromes prior. These findings support prioritizing equitable access to modern diagnostics and systemic treatment and motivate prospective, molecularly integrated studies.

ORAL PRESENTATION

Development of an Integrated Follow-Up Framework for Patients with HR+/HER2− Early Breast Cancer Receiving Adjuvant CDK4/6 Inhibitors: Expert opinion.

Priya Rathi

Background: Follow-up after curative-intent treatment for early breast cancer is generally guided by symptom-directed surveillance, with major international guidelines recommending clinical assessment and selective investigations based on symptoms or clinical findings. The introduction of adjuvant CDK4/6 inhibitors for selected patients with hormone receptor-positive, human epidermal growth factor receptor 2-negative early breast cancer adds agent-specific safety monitoring requirements that need to be considered alongside routine survivorship care.

Objective: To develop a proposed integrated follow-up framework for patients with HR+/HER2− early breast cancer receiving adjuvant CDK4/6 inhibitors by aligning established post-treatment follow-up recommendations with label-recommended safety monitoring requirements for CDK4/6i.

Methods: A structured review of international early breast cancer follow-up guidelines and approved label recommendations for ribociclib and abemaciclib was conducted. Guidelines from ESMO, ASCO, NCCN, and ISMPO were reviewed. Label recommendations were assessed to identify treatment-specific monitoring needs, including hematologic, hepatic, QTc and electrocardiographic, pulmonary, gastrointestinal, and thromboembolic monitoring. The findings were discussed during an in-person consensus roundtable with multidisciplinary experts from eleven Centres of Excellence across India held in November 2025. Through structured discussion, the proposed framework was refined to define clinically relevant monitoring priorities throughout therapy The discussion focused on key monitoring priorities and practical ways to combine routine survivorship care with treatment-specific monitoring.

Results:

Existing early breast cancer follow-up guidelines largely recommend symptom-directed surveillance, although the frequency and content of clinical assessments vary across guidance documents. In contrast, adjuvant CDK4/6 inhibitors require planned, agent-specific safety monitoring at defined intervals in line with the applicable local label recommendations.

The proposed framework was designed to bring these two elements together by combining symptom-directed follow-up for recurrence and survivorship-related concerns with time-dependent CDK4/6 inhibitor safety monitoring.

The framework recommends more frequent follow-up during the initial months of CDK4/6 inhibitor treatment, when treatment-related toxicities, laboratory abnormalities, and adherence-related concerns may be more likely to arise. Once treatment is stable, follow-up can transition to longitudinal assessments based on individual patient needs, while continuing to assess symptoms suggestive of recurrence, endocrine therapy adherence and continuation, persistent or delayed toxicities, bone and menopausal health, cardiovascular risk, and quality of life. The framework includes hematologic and hepatic monitoring for both ribociclib and abemaciclib; QTc and electrocardiographic monitoring for ribociclib, as per applicable local label recommendations; and focused clinical assessment for diarrhoea, venous thromboembolism, and interstitial lung disease or pneumonitis in patients receiving abemaciclib. The frequency of treatment-specific monitoring should remain aligned with the applicable local label recommendations and individualized according to clinical findings, comorbidities, concomitant medications, and clinician judgment.

Conclusion:

The proposed framework integrates symptom-directed follow-up for patients with HR-positive, HER2-negative early breast cancer with a structured, time-dependent approach to CDK4/6 inhibitor safety monitoring. It emphasizes closer follow-up during the early treatment phase, followed by longitudinal assessment once treatment is stable. This approach may support timely identification and management of treatment-related toxicities, adherence to CDK4/6 inhibitor and endocrine therapy, and ongoing assessment of recurrence-related symptoms and longer-term health concerns. The framework should be applied alongside applicable local label recommendations, institutional protocols, and individualized clinical judgment.

ORAL PRESENTATION

Predictors of Mortality in Patients with Mycosis Fungoides

Simran Aulakh, Natesh Samaroo, Motaz Elwany, Qiuyi Wang, Anusha Pemmasani, Anish Myneni, Dilan Trivedi, Hunter Slosser, Joseph Tafalla, Sahejdeep Chohan, Humbal Omar, Timothy Chon, Jennifer Zheng, Shamirah Johnson, Kyle Miller, Hossein Pourtaghi, Yousef Mzannar, Simcha Weissman

Background: Mycosis fungoides is the most common form of cutaneous T-cell lymphoma, presenting with diverse skin manifestations and potential progression to systemic involvement. Despite its indolent nature in early stages, the disease can be challenging to diagnose and treat due to its clinical heterogeneity. Understanding the clinical characteristics and mortality determinants is crucial for improving patient outcomes as treatment strategies evolve.

Methods: This retrospective cohort study utilized the SEER database to analyze patients diagnosed with mycosis fungoides between 2004 and 2021. The study examined demographic factors, tumor characteristics, treatment modalities, and survival outcomes. Multivariate Cox proportional hazards regression was employed to identify predictors of overall mortality (OM) and cancer-specific mortality (CSM), with a focus on age, tumor stage, and socioeconomic factors.

Results: The analysis included 531 patients, revealing that most were male (60.83%) and aged 60-79 years (42.75%) at diagnosis. Non-Hispanic Whites constituted the majority (67.61%), and 59.51% of cases had localized disease at diagnosis. Radiation therapy was the most common treatment (95.86%), followed by surgery (97.93%). Multivariate analysis identified advanced age (80+ years) and distant tumor stage as significant predictors of higher mortality. Non-Hispanic Black patients exhibited higher mortality rates compared to Non-Hispanic Whites, while higher income levels were associated with lower mortality. An unexpected association was found between radiation therapy and increased overall mortality, warranting further investigation.

Conclusion: This study highlights the significant impact of age, tumor stage, and socioeconomic factors on mortality in mycosis fungoides. The findings underscore the need for early detection and tailored treatment strategies, particularly in older and socioeconomically disadvantaged populations. The unexpected association between radiation therapy and increased mortality suggests the need for careful consideration of treatment modalities and further research to optimize patient outcomes.

POSTER PRESENTATION

VIA Extractortissue disaggregator for AT2 organoid prep in IPF research

Tsuyoshi Takashima1, Prof. Eiichi Morii1
Osaka University, Japan

Organoids derived from alveolar type 2 progenitor cells (AT2) have become essential experimental models for studying the underlying mechanisms of chronic interstitial lung diseases, such as idiopathic pulmonary fibrosis (IPF). This platform has the potential to provide key insights into cellular and molecular processes involved in disease-associated transition of AT2 cells into aberrant basaloid cells.

Successful organoid culture requires viable, highly proliferative cells with intact surface antigens enabling cell enrichment. Isolating enough AT2 cells for organoid culture using manual methods is challenging, especially for those unfamiliar with the process. Cytiva'sOmics bundle -VIA Extractortissue disaggregator, when used with tissue tailored enzymes, is designed to provide gentle and reproducible semi-automated mechanical dissociation maintaining high cell viability and yield for downstream cellular workflows such as organoid culture.

In this study, we evaluated a workflow for establishing human type 2 alveolar epithelial (AT2) organoids using the Cytiva VIAExtractortissue disaggregator in combination with the Beckman Coulter CytoFLEXSRT cell sorter and compared it to an optimisedmanual dissociation method. Following dissociation, we isolated the AT2 positive cells by staining for EpCAM and HTII-280 and sorted cells using Beckman Coulter CytoFLEXSRT. We established organoid cultures on Matrigel and confirmed morphology by immunostaining for HTII-280 and SFTPC.

We achieved cell viability of 85.6%% using the VIA Extractortissue disaggregator with 3 normal human lung tissue samples weighing 300 mg, which yielded in excess of106 cells per tissue sample. The VIA Extractortissue disaggregator provided an efficient and gentle dissociation method evident by the presence and continued expression of the critical surface marker HTII-280, essential for organoid establishment.

A semi-automated system that consistently produces high-quality single cells is a valuable technology to accelerate progress across diverse research fields. Dissociation using VIA Extractortissue disaggregator benefits not only researchers working with lung organoids or expanding into other tissue-derived models and basic research like ours, but also for pharmaceutical companies seeking stable, reproducible data.

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1 Osaka University, Japan

Introduction

In this article, we'll explore research to elucidate the pathophysiology of chronic interstitial pneumonia, particularly an intractable disease called idiopathic pulmonary fibrosis (IPF), and develop therapeutic drugs based on these findings (see Table 1).

Recent advances in developmental biology have brought attention to 3D cultures such as cerebral organoids, which model organogenesis using tissue-derived stem or progenitor cells. While our work focus is lung disease, these insights extend across organ systems, guiding us in establishing organoid models from specific tissue sources, including lung epithelium.

In IPF, the spotlight has been on the aberrant basaloid cells in the pathological exacerbation mechanism. Studies show that human type 2 alveolar epithelium (AT2) transdifferentiates into aberrant basaloid cells, though the details of this process await further clarity. Additionally, inflammation stimulation using AT2 organoids has been found to increase the KRT17 gene expression, a marker of aberrant basaloid cells, prompting further research into culture methods.

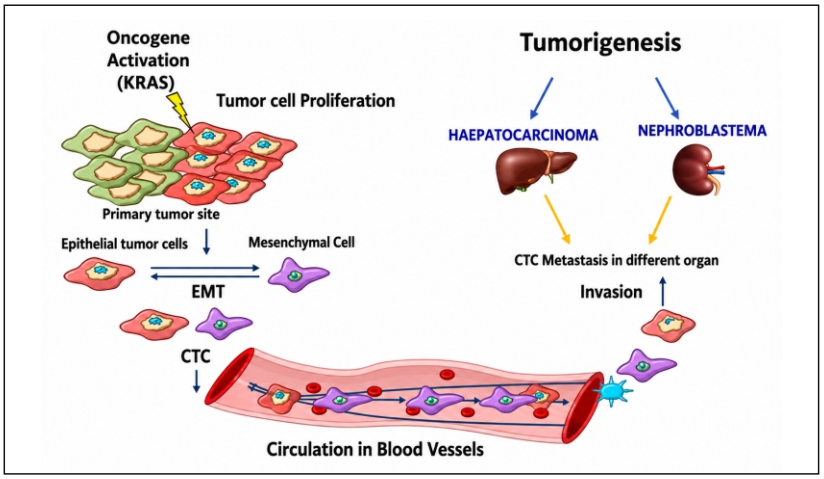
To advance studies in this domain, efficiently establishing and cultivating specific tissue, such as human type 2 alveolar epithelial (AT2) organoids, is crucial. At Osaka University, we've been using a manual method for isolating AT2 cells from healthy human tissue (lung) to establish AT2 organoids in our workflow. However, the introduction to the VIA Extractor tissue disaggregator allows researchers, including those new to this field, to work more efficiently with tissue-derived samples and achieve higher cell viability. Therefore, we will evaluate the efficiency of Cytiva's VIA Extractor tissue disaggregator and Beckman-Coulter CytoFLEX SRT cell sorter with our organoids to compare the performance against the manual method (see Table 1).

Table 1: Setting for each method with 300mg tissue samples

Conclusion

Isolating enough AT2 cells for organoid culture using manual methods is challenging, especially for those unfamiliar with the process, as it requires skilled technique. The most significant advantage of using the VIA Extractor tissue disaggregator in this study was that it provided an easy, efficient way to establish organoids within a single day, even before the enzymes and temperature conditions were fully optimized.

As compared to the manual method, the VIA Extractor tissue disaggregator enabled faster, high-quality single-cell isolation from specific tissue, reducing overall experimental time and improving workflow efficiency. With the ability to customize settings based on the hardness, tissue size, and enzyme type, the VIA Extractor tissue disaggregator delivers consistent results regardless of user experience or tissue condition, whether normal or diseased. Importantly, it maintained the Expression of the critical surface marker HTII-280 essential for organoid establishment; the VIA Extractor tissue disaggregator preserved this marker's brightness, confirming its gentle dissociation process. This reliability benefits not only researchers working with lung organoids or expanding into other tissue-derived models and basic research like ours, but also for pharmaceutical companies seeking stable, reproducible data. An automated system that consistently produces high-quality single cells is a valuable technology to accelerate progress across diverse research fields.



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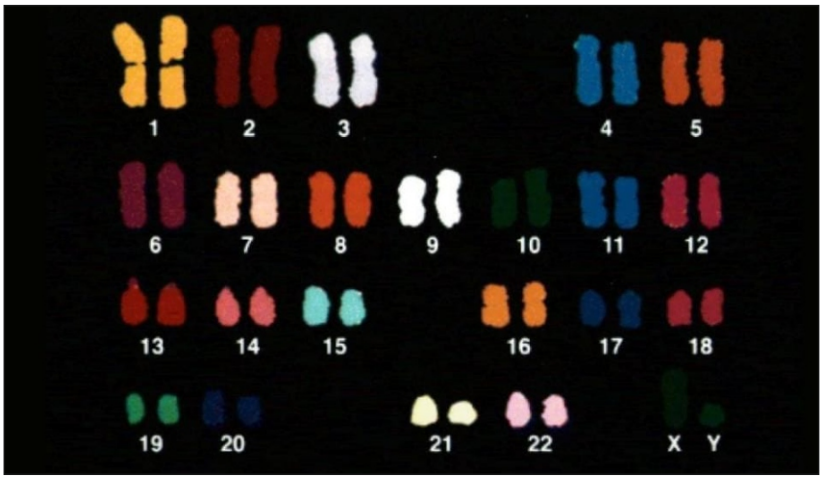

FISH Analysis for Early Diagnosis of Cancer Patients

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