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Dear Colleagues,

Radiation oncology continues to advance through innovation, collaboration, and an unwavering commitment to patient-centered care. Across India, our specialty is evolving with the adoption of modern technologies, precision treatment approaches, and multidisciplinary integration, all aimed at improving outcomes and quality of life for patients.

This issue of the newsletter reflects the collective efforts, experiences, and academic contributions of our fraternity. It highlights our shared mission to strengthen cancer care delivery and expand the reach of safe, effective, and accessible radiotherapy services across the country.

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REPRINT

Advances in molecular and genetic profiling of meningiomas for improved diagnosis, prognosis, and targeted therapy

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Advances in molecular and genetic profiling of meningiomas for improved diagnosis, prognosis, and targeted therapy

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Background: Meningiomas exhibit substantial biological and clinical heterogeneity, and traditional histopathology alone often fails to accurately predict tumor behavior. Advances in molecular and genetic profiling have significantly enhanced diagnostic precision, prognostic assessment, and therapeutic decision-making. Methods: This review synthesizes current evidence on genomic, epigenomic, transcriptomic, and liquid biopsy–based approaches used to characterize meningiomas. Results: Key developments include characterization of NF2 and non-NF2 driver mutations, refinement of DNA methylation-based classification systems, identification of high-risk markers such as TERT promoter mutations and CDKN2A/B deletions, and the emergence of targeted therapeutic strategies. Liquid biopsy and circulating biomarkers further enable non-invasive disease monitoring and molecular risk stratification. Conclusion: Molecular profiling has transformed meningioma classification and risk prediction, supporting a shift toward precision neuro-oncology. Future progress will depend on integrated multi-omic diagnostics, improved biomarker-guided surveillance, and development of targeted therapeutic options for aggressive molecular subgroups.

KEYWORDS
meningioma, molecular profiling, DNA methylation, targeted therapy, liquid biopsy, neuro-oncology, prognostic markers

1 Introduction

Meningiomas account for approximately 37% of all primary central nervous system tumors, and their incidence continues to rise, likely influenced by increased imaging use and the aging global population (1). Although most meningiomas are classified as World Health Organization (WHO) grade 1 and are generally benign, a significant proportion—estimated at 20% to 30%—display atypical or anaplastic features corresponding to grade 2 and grade 3 disease, respectively (2). These higher-grade tumors exhibit more aggressive clinical behavior, including an increased propensity for recurrence, local progression, and, in rare cases, metastatic spread. Traditionally, clinicians have relied on histopathological grading as the primary method of predicting meningioma behavior. However, considerable biological heterogeneity exists even within individual histologic grades, and several studies have

demonstrated that histology alone does not reliably distinguish indolent tumors from those with high recurrence risk, even when gross total resection is achieved (3).

The past decade has witnessed a paradigm shift in meningioma research and clinical management, driven largely by the integration of molecular techniques such as next-generation sequencing, DNA methylation profiling, copy-number assessment, and transcriptional analysis. These tools have enabled investigators to uncover the genomic and epigenomic underpinnings of meningioma biology, revealing previously unrecognized tumor subgroups and refining prognostic assessments. Moreover, the development of liquid biopsy techniques and multi-omic classifiers offers promising opportunities for non-invasive tumor characterization and real-time disease monitoring. Collectively, these advances represent a significant evolution in precision neuro-oncology. This review provides a comprehensive overview of the molecular and genetic developments that have transformed diagnostic accuracy, prognostic assessment, and targeted therapeutic strategies in meningioma.

2 Somatic driver mutations and molecular pathogenesis

2.1 NF2 alterations and chromosomal instability

The earliest and most extensively studied genetic event in meningioma pathogenesis involves alterations in NF2, located on chromosome 22q (Figure 1 illustrates the genomic driver landscape, distinguishing NF2-mutant and NF2-independent molecular pathways). NF2 mutations or deletions occur in approximately 40% to 60% of sporadic meningiomas and represent the dominant driver alteration in this disease (4, 5). The NF2 gene encodes Merlin, a tumor suppressor protein that regulates contact inhibition and cytoskeletal organization. Loss of Merlin function disrupts these regulatory processes and leads to activation of multiple oncogenic pathways, including Hippo and focal adhesion kinase (FAK) signaling, promoting tumor growth and proliferation. Clinically, NF2-mutant meningiomas tend to arise along the convexities or parasagittal regions of the brain. They frequently exhibit widespread chromosomal instability, often characterized by monosomy 22 as well as losses in chromosomes 1p, 6q, 10q, 14q, and 18q (6). These copy-number alterations contribute substantially to the aggressive potential of NF2-mutant tumors, with high copy-number burden correlating strongly with recurrence and poor outcomes.

2.2 Non-NF2 molecular subgroups

Advances in sequencing technologies have revealed a diverse array of NF2-independent genetic alterations that define biologically and clinically distinct molecular subgroups of meningiomas. Rather than constituting a single homogeneous category, these non-NF2 alterations encompass genes with diverse molecular functions and are associated with preferential anatomical locations, characteristic histologic subtypes, and differing prognostic implications. In general, these mutations are mutually exclusive with NF2 loss and are particularly enriched in skull-base meningiomas, which often display more stable genomic profiles and indolent clinical behavior.

Mutations in TRAF7, an E3 ubiquitin ligase, occur in approximately one-quarter of all meningiomas (5) (Figure 1). TRAF7-mutant tumors frequently co-occur with the KLF4 K409Q mutation, a combination that is nearly pathognomonic for secretory meningioma (5), a distinct histologic subtype characterized by eosinophilic pseudopsammoma bodies. These TRAF7/KLF4- mutant tumors typically arise from the anterior skull base, are most often classified as WHO grade 1, and generally demonstrate a benign clinical course.

Alterations in the PI3K/AKT/mTOR signaling pathway represent another major NF2-independent subgroup. Mutations in AKT1, particularly the E17K variant, result in constitutive pathway activation and are most commonly observed in midline skull-base meningiomas, including those of the planum sphenoidale

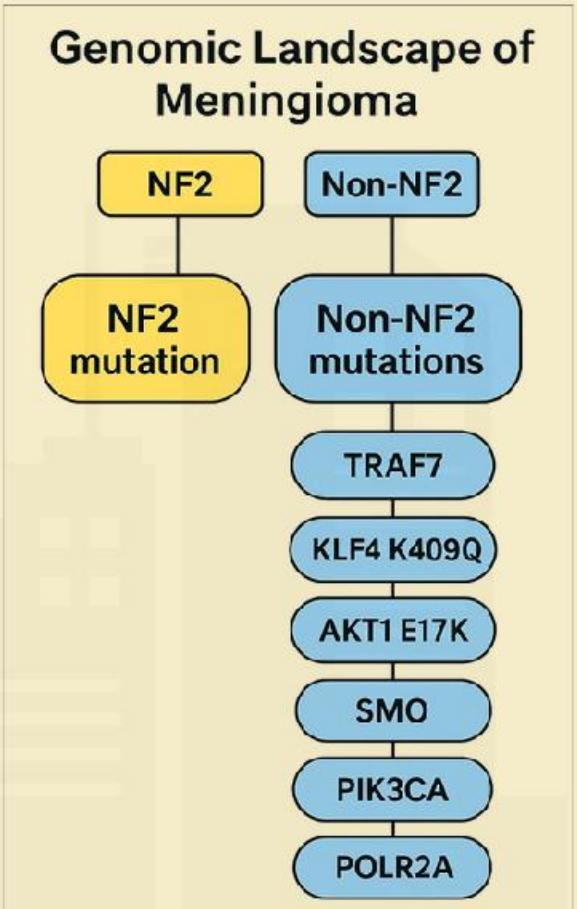

FIGURE 1
Genomic landscape of meningioma showing NF2-mutant and non-NF2 molecular subgroups and their major driver mutations.

and olfactory groove (7). Although these tumors are frequently WHO grade 1 by histology, they exhibit distinct radiographic and biological characteristics and have emerged as rational candidates for targeted pathway inhibition. Similarly, PIK3CA mutations occur in approximately 7% of meningiomas and are enriched in non-NF2 skull-base tumors (7). These alterations further support the biological distinction of this subgroup and provide a therapeutic rationale for PI3K or mTOR inhibition in selected patients.

Activating mutations in SMO, a key component of the Hedgehog signaling pathway, are identified in approximately 5% of meningiomas and show a strong predilection for the olfactory groove and anterior midline skull base (7). SMO-mutant tumors form a molecularly defined subgroup with characteristic transcriptional profiles and represent one of the clearest examples of a genotype-directed therapeutic opportunity in meningioma.

Beyond skull-base–predominant alterations, several additional NF2-independent mutations are associated with specific histopathologic phenotypes and prognostic implications. POLR2A mutations define a transcriptionally unique subset of largely benign meningiomas with low recurrence risk (8). SUFU mutations, though rare, implicate upstream dysregulation of Hedgehog signaling and may have implications for pathway-directed therapies (9). In contrast, loss of BAP1 function is strongly associated with rhabdoid meningioma, a high-grade variant characterized by aggressive clinical behavior and poor prognosis (10). Germline or somatic loss of SMARCE1 is characteristic of clear cell meningioma, a subtype frequently occurring in younger patients and associated with a higher risk of recurrence, particularly in spinal locations (11). Finally, mutations in ARID1A highlight the role of SWI/SNF chromatin-remodeling dysfunction in a subset of biologically aggressive and higher-grade meningiomas (12). Together, these observations underscore that NF2-independent meningiomas represent a heterogeneous collection of molecularly, anatomically, and clinically distinct entities, rather than a single category. Recognition of this heterogeneity allows refinement of meningioma classification beyond traditional histopathology, improves prognostic stratification, and facilitates the development of personalized, genotype-informed therapeutic strategies (Table 1 integrates driver mutations with clinical, anatomical, and prognostic associations, serving as a translational reference.).

3 Epigenetic profiling and DNA methylation classification

3.1 Development of methylation-based classification

One of the most transformative advances in meningioma research has been the incorporation of DNA methylation profiling into tumor classification (Figure 2 depicts DNA methylation-based classification and its correlation with WHO grading and prognosis). Sahm et al. (13) pioneered a methylation-based grading system that stratifies meningiomas into biologically distinct classes with far superior prognostic accuracy compared to WHO histologic grade alone. This approach integrates genome-wide methylation signatures, copy-number patterns, and

TABLE 1 Driver Mutations and Clinical Associations.

Driver mutation	Genomic effect/ pathway	Clinical association	Typical location	Histologic correlation	Prognostic notes
NF2	Loss of Merlin → activation of FAK, Hippo pathway dysregulation	Chromosomal instability; aggressive behavior	Convexity, parasagittal, falx	Fibrous, transitional meningiomas	High CNV burden correlates with recurrence; seen in higher-grade tumors
TRAF7	E3 ubiquitin ligase mutation	Anterior skull base meningiomas	Anterior/medial skull base	Often combined with KLF4 K409Q (secretory subtype)	Generally benign; rarely progresses
AKT1 (E17K)	Activates PI3K/AKT/mTOR pathway	Midline skull base meningiomas	Planum sphenoidale, olfactory groove	Meningothelial WHO grade 1	Potential target for AKT inhibitors; stable behavior
KLF4 (K409Q)	Transcription factor alteration	Secretory meningioma	Skull base	Secretory subtype with pseudopsammoma bodies	Indolent behavior; consistent molecular signature
PIK3CA	Activates PI3K pathway	Subset of non-NF2 skull-base tumors	Skull base	Meningothelial	Targetable with PI3K inhibitors
SMO	Hedgehog pathway activation	Olfactory groove tumors	Anterior midline base	Often meningothelial	May respond to SMO inhibitors (vismodegib)
POLR2A	RNA polymerase II subunit mutation	Distinct benign molecular class	Variable	Benign transcriptional subtype	Usually low recurrence
BAP1	Loss-of-function; chromatin remodeling defect	Rhabdoid meningiomas	Variable	Rhabdoid morphology	High-grade; very poor prognosis
SMARCE1	SWI/SNF chromatin remodeling	Clear cell meningioma (often hereditary)	Spinal canal predominance	Clear cell subtype	High recurrence risk
SUFU	Hedgehog pathway regulator	Rare SUFU-related subgroup	Variable	Uncommon	May affect Hedgehog-directed therapy
ARID1A	SWI/SNF complex alteration	Aggressive meningioma subset	Variable	High-grade histology	Associated with progression

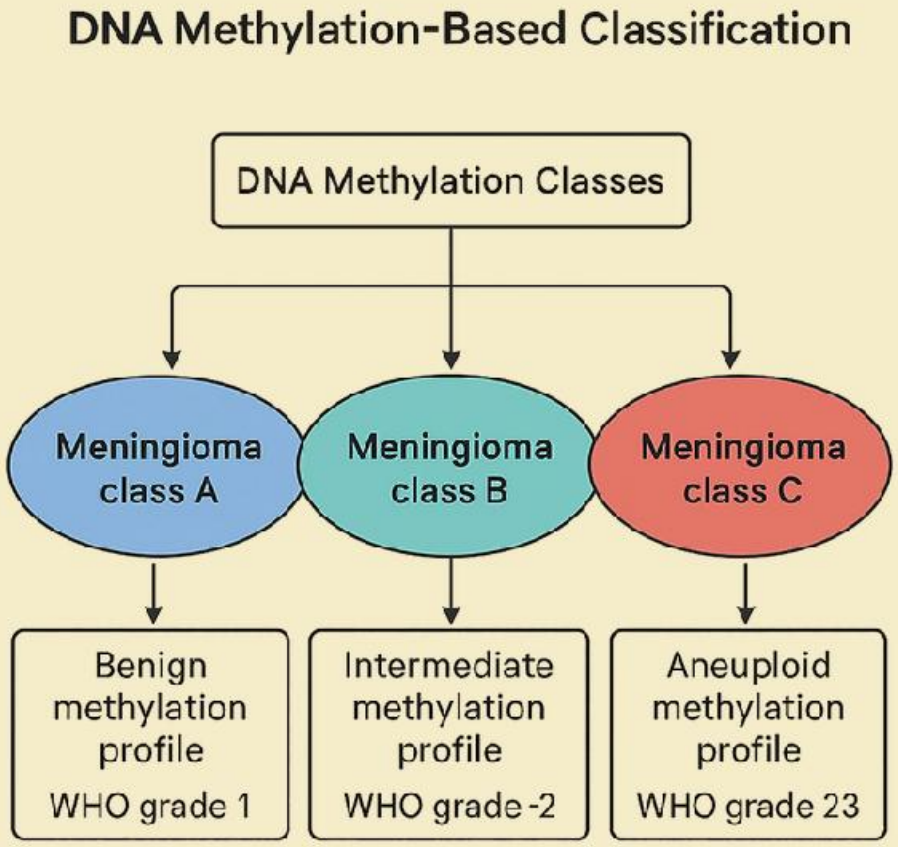

FIGURE 2
DNA methylation–based classification of meningiomas demonstrating biologically distinct classes correlated with WHO grade and prognosis.

transcriptional programs to identify tumors with similar biological behaviors, regardless of histologic appearance.

3.2 Consensus molecular groups

Further refinement occurred through the work of Nassiri et al. (14), who conducted a large multi-institutional analysis combining methylation, transcriptomic, copy-number, and mutational data. Their study identified three major consensus molecular groups (CMGs) (Figure 2): a Merlin-intact group associated with favorable outcomes, an immune-enriched group characterized by inflammatory microenvironments and intermediate prognosis, and a proliferative group marked by high cell-cycle activity and poor clinical outcomes. These CMGs predict progression-free survival with greater accuracy than histologic grade and reveal biological features that may have therapeutic implications, such as immune signatures or proliferative pathway activation.

3.3 Clinical implications

Methylation profiling increasingly informs real-world clinical decision-making. It aids in predicting recurrence risk following gross total resection, identifies histologically benign-appearing tumors with aggressive molecular features, guides the need for adjuvant radiotherapy, and assists in selecting patients for molecularly tailored clinical trials. For recurrent, atypical, and morphologically ambiguous meningiomas, methylation profiling is rapidly becoming an essential diagnostic complement.

4 High-risk prognostic molecular markers

Advances in molecular diagnostics have identified specific genetic and epigenetic alterations that independently predict prognosis in meningiomas (15).

Mutations in the TERT promoter (TERTp), particularly C228T and C250T substitutions, occur in approximately 3% to 5% of meningiomas (Figure 3 summarizes high-risk molecular markers that independently predict recurrence and survival) and are strongly associated with early recurrence, malignant progression, and significantly reduced survival. These mutations are now regarded as an adverse prognostic feature sufficiently powerful to define WHO grade 3 in some contexts, even when histology is low-grade.

Homozygous deletions of CDKN2A/B, which encode critical cell-cycle regulators, represent another robust predictor of poor

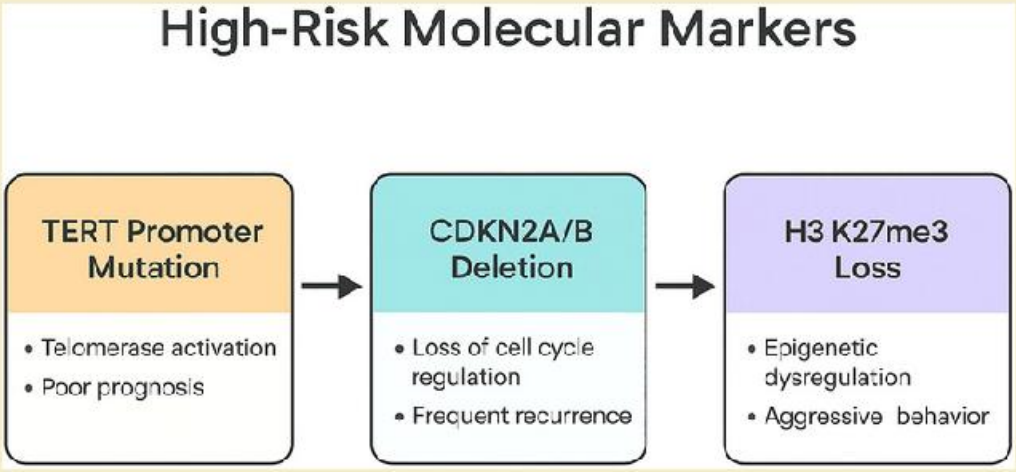

FIGURE 3
High-risk molecular markers associated with aggressive behavior including TERT promoter mutation, CDKN2A/B deletion, and H3K27me3 loss.

outcome and have been incorporated into the WHO classification as a molecular marker of grade 3 meningioma. These deletions frequently co-occur with NF2 alterations and high chromosomal instability, reinforcing their association with aggressive behavior.

Epigenetic markers such as loss of H3K27me3, a histone modification associated with transcriptional repression, also correlate with poor prognosis, particularly among atypical meningiomas. Tumors lacking H3K27me3 expression demonstrate significantly shorter recurrence-free survival. This readily assessable immunohistochemical marker enhances risk stratification when combined with molecular and histologic data.

Global copy-number burden further contributes to prognostication. Losses involving chromosomes 1p, 6q, 10q, and 14q are associated with aggressive behavior, and tumors with high copy- number variation show markedly increased recurrence rates (15).

5 Liquid biopsy and circulating biomarkers

Liquid biopsy has emerged as a promising adjunct to conventional imaging and tissue-based diagnostics in meningioma. Circulating tumor DNA (ctDNA) can be detected in plasma and carries tumor-specific genetic and epigenetic information (Figure 4 presents the workflow of liquid biopsy and its potential clinical applications). Several studies have demonstrated that meningioma-specific methylation signatures can be identified in plasma-derived cell-free DNA (cfDNA), providing a non-invasive approach to tumor detection and molecular classification (16). In addition, liquid biopsy enables real-time monitoring of tumor dynamics, with ctDNA levels increasing in parallel with tumor progression and, in some cases, allowing earlier detection of recurrence compared with conventional MRI imaging (17). Serial plasma sampling also permits assessment of molecular evolution over time, including the acquisition of adverse alterations such as TERT promoter mutations during disease progression (18).

Beyond ctDNA, circulating microRNAs—including miR-21 and miR-34a—have shown differential expression between benign and aggressive meningiomas, suggesting potential roles as minimally invasive biomarkers (19). Proteomic markers such as PD-L1, VEGF-A, and proinflammatory cytokines have likewise been explored for their prognostic and therapeutic relevance (20).

Despite these encouraging findings, important limitations remain. Sensitivity of liquid biopsy is reduced in low-volume disease and may vary according to tumor size, location, and vascularity. Furthermore, most available evidence is derived from retrospective or exploratory studies, underscoring the need for prospective validation in larger, clinically annotated cohorts before routine clinical implementation.

6 Therapeutic implications and targeted treatment

The characterization of specific molecular subgroups has provided a foundation for targeted therapeutic strategies in meningioma, although effective systemic treatments remain limited.

Tumors harboring SMO mutations represent an actionable subgroup for Hedgehog pathway inhibitors. Early-phase clinical trials of agents such as vismodegib and sonidegib have demonstrated modest disease stabilization in selected patients, underscoring the need for molecularly enriched cohorts (7).

Alterations in the PI3K/AKT/mTOR pathway, including AKT1 and PIK3CA mutations, justify the investigation of pathway inhibitors (Figure 5 highlights actionable signaling pathways and corresponding targeted therapies). AKT inhibitors such as capivasertib and PI3K inhibitors such as alpelisib are being evaluated in molecularly selected patients. The mTOR inhibitor everolimus has shown mixed results, particularly when combined with somatostatin analogues like octreotide (11).

NF2-mutant meningiomas may benefit from FAK inhibitors, given the central role of FAK signaling in Merlin-deficient tumors.

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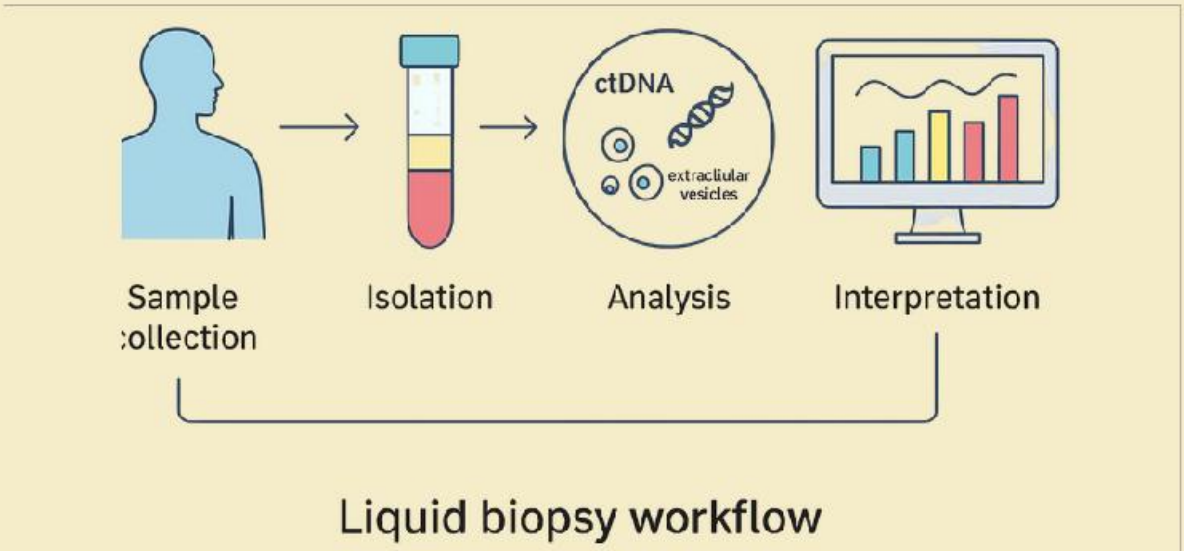

FIGURE 4
Liquid biopsy workflow illustrating sample collection, circulating tumor component isolation, molecular analysis, and clinical interpretation.

Clinical trials of GSK2256098 have shown early evidence of disease stabilization in this subgroup (21).

In tumors characterized by CDKN2A/B deletion or cell-cycle dysregulation, CDK4/6 inhibitors such as abemaciclib have demonstrated promising early activity. Epigenetically dysregulated meningiomas, particularly those harboring SWI/SNF complex alterations such as ARID1A or SMARCB1 mutations, may be sensitive to EZH2 inhibitors, with early-phase trials of tazemetostat supporting further exploration (22).

Although meningiomas typically exhibit low mutational burden, some tumors show immune-rich microenvironments. Immune checkpoint inhibitors such as pembrolizumab and nivolumab have been tested in recurrent high-grade meningioma, demonstrating limited but noteworthy activity (23).

7 Clinical integration of molecular profiling

The integration of molecular profiling into clinical practice has significantly improved diagnostic precision, prognostication, and treatment planning. Tumors with unique molecular signatures—such as secretory meningiomas characterized by combined KLF4

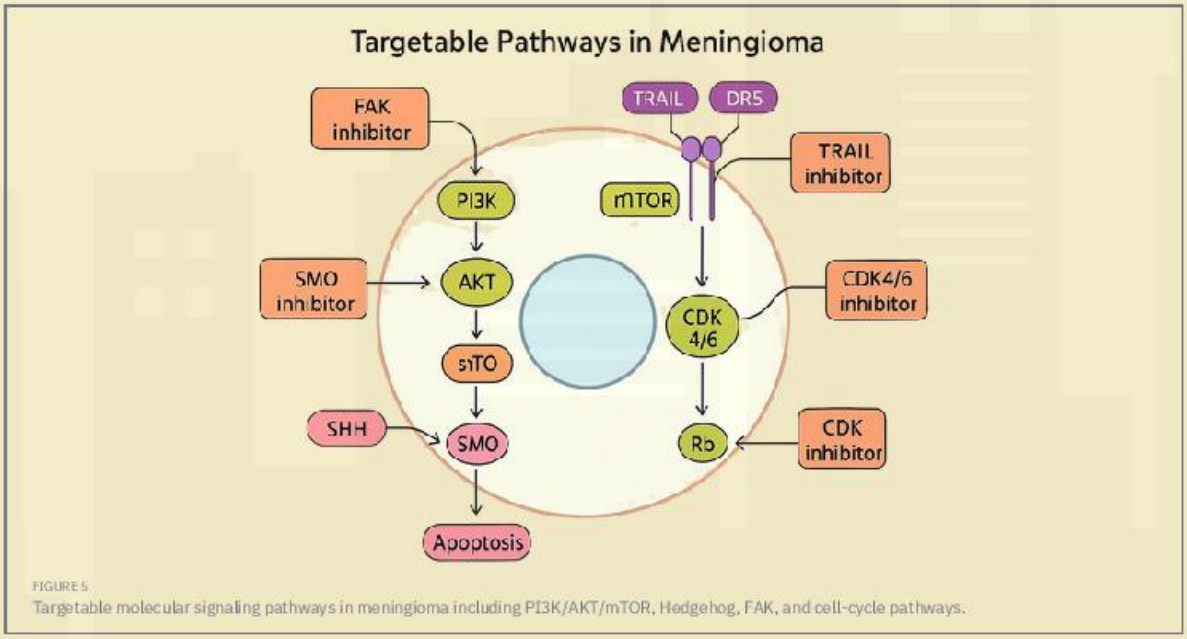

FIGURE 5
Targetable molecular signaling pathways in meningioma including PI3K/AKT/mTOR, Hedgehog, FAK, and cell-cycle pathways.

and TRAF7 mutations, clear cell meningiomas driven by SMARCE1 loss, or BAP1-inactivated rhabdoid tumors—can now be accurately classified based on their genetic alterations, even when histology is ambiguous. These molecular insights aid pathologists and clinicians in refining diagnoses that may influence subsequent management.

Prognostically, markers such as TERT promoter mutation, CDKN2A/B deletion, H3K27me3 loss, and methylation class are increasingly used to guide decisions regarding adjuvant radiotherapy and intensity of surveillance. Tumors classified within aggressive methylation groups or harboring high-risk molecular features may warrant closer imaging follow-up or early postoperative radiotherapy, even when histology suggests low-grade disease.

Perhaps most importantly, molecular profiling facilitates personalized treatment strategies through selection of patients for clinical trials evaluating targeted agents or immunotherapies. The identification of actionable mutations such as SMO, AKT1, or PIK3CA allows patients to be enrolled in genotype-directed therapeutic studies, representing a critical step toward precision medicine in meningioma.

8 Limitations and future directions

Despite significant progress, challenges remain:

- Limited access to methylation profiling in low-resource settings.
- Lack of highly effective systemic therapies; most targeted treatments result in disease stabilization, not regression.
- Intratumoral heterogeneity complicates single-biopsy interpretation.
- Need for standardized molecular reporting systems integrating histology, CNV, methylation, and mutations.
- Requirements for prospective multi-omic trials to validate classifiers and therapeutic responses.
- Future research will likely emphasize combined radiotherapy–molecular inhibitor strategies, liquid biopsy–guided monitoring, and deeper insights into spatial heterogeneity and tumor microenvironment.

Addressing these challenges will require integration of emerging biological insights, minimally invasive molecular diagnostics, advanced computational tools, and collaborative data infrastructures.

8.1 Emerging molecular insights in WHO grade 3 meningiomas

Recent work has further refined the biological understanding of grade 3 meningiomas, highlighting the central role of TERT promoter mutations, CDKN2A/B homozygous deletions, high copy-number burden, and proliferative methylation-defined subgroups in driving aggressive behavior (24). These tumors exhibit marked genomic instability, dysregulated cell-cycle control, and enrichment of DNA repair and proliferation pathways. Recognition of these molecular characteristics supports incorporation of molecular risk factors into treatment algorithms, including consideration of early adjuvant radiotherapy, intensified surveillance schedules, and prioritization for enrollment in molecularly targeted clinical trials.

8.2 Blood-based molecular profiling for surgical decision-making

Liquid biopsy approaches are being explored as adjuncts to tissue-based diagnostics in meningioma. Circulating tumor DNA (ctDNA) and tumor-specific methylation signatures detectable in plasma offer a non-invasive means of tumor classification and disease monitoring (16). Although sensitivity remains limited in low-volume disease, ongoing improvements in assay sensitivity raise the possibility that blood-based molecular profiling may assist in preoperative risk stratification, particularly in tumors located in surgically challenging regions. Integration of plasma-derived molecular data with imaging and clinical parameters may eventually help guide the extent of surgical resection, inform adjuvant therapy decisions, and tailor postoperative surveillance strategies (17).

8.3 Artificial intelligence and computational modeling

Emerging artificial intelligence (AI)–driven modeling approaches are increasingly being integrated with molecular profiling in meningioma research. Machine learning frameworks combining radiomic features, genomic alterations, DNA methylation patterns, and clinical variables have demonstrated potential for improving risk stratification and recurrence prediction. Integrative AI platforms can identify imaging–molecular correlations that are not readily apparent through conventional analysis, enabling non-invasive inference of tumor biology and facilitating longitudinal disease tracking (25). As these tools mature, AI-assisted systems may support personalized surveillance strategies and treatment planning by dynamically integrating molecular, radiologic, and clinical data.

8.4 Need for centralized molecular data repositories

A key limitation in advancing meningioma molecular research is fragmentation of datasets across institutions. Establishment of centralized, multi-institutional repositories integrating genomic, epigenetic, radiologic, and clinical outcome data would substantially accelerate validation of molecular classifiers and therapeutic targets. Such initiatives would require standardized biospecimen processing protocols, harmonized bioinformatics pipelines, secure data-sharing infrastructure, and robust ethical governance frameworks. Centralized repositories could facilitate large-scale validation studies, improve reproducibility, and enable AI-driven predictive modeling, thereby enhancing the translational impact of molecular profiling.

9 Conclusion

Meningiomas, once viewed as primarily histologically defined tumors, are now recognized as molecularly diverse neoplasms driven by complex genomic, epigenomic, and transcriptomic

alterations. Advances in molecular profiling including driver mutation characterization, DNA methylation classification, and identification of high-risk prognostic markers have dramatically improved diagnostic accuracy and risk stratification. Emerging targeted therapies and liquid biopsy technologies promise to reshape management strategies, particularly for recurrent or aggressive tumors. As molecular diagnostics become increasingly integrated into clinical practice, tailored therapeutic approaches and improved patient outcomes are within reach.

Author contributions

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Conflict of interest

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Sohrai painting is an ancient, matriarchal ritual mural art form from Jharkhand, India, traditionally painted by tribal women on mud walls to celebrate the harvest season and cattle

ARTICLE

Role of Anaesthesia in Radiotherapy: Comprehensive Integration, Advanced Techniques, Safety Frameworks, and Standardized Practice in Modern Oncology

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Keywords

Radiotherapy; Anaesthesia; Pediatric Oncology; Sedation; Brachytherapy; SRS; Patient Safety; Immobilization; SOP

Abstract

Radiotherapy (RT) has evolved into a precision-driven oncologic modality characterized by steep dose gradients, image guidance, adaptive workflows, and highly conformal dose delivery. These advances mandate stringent immobilization, reproducibility, and control of both inter- and intrafraction motion. Anaesthesia, therefore, has transitioned from a supportive adjunct to a critical enabler of accurate radiotherapy delivery in selected patient populations. Its role is particularly significant in pediatric oncology, brachytherapy, stereotactic radiotherapy, and in patients with anxiety, pain, or neurological impairment.
This comprehensive review elaborates on the physiological, technical, and operational rationale for anaesthesia in RT, discusses detailed pharmacological profiles of commonly used agents, and explores scenario-specific applications. The article also delineates safety considerations unique to the radiotherapy environment, outlines the central coordinating role of the radiation oncologist, and provides a detailed Standard Operating Protocol (SOP) aligned with NABH and AERB principles. Emerging trends, including non-pharmacological interventions and AI-based monitoring, are discussed. This manuscript aims to serve as a complete academic and practical resource for radiation oncologists, anaesthesiologists, and multidisciplinary oncology teams.

1. Introduction

The modern era of radiation oncology is defined by technological sophistication and precision. Techniques such as intensity-modulated radiotherapy (IMRT), volumetric modulated arc therapy (VMAT), stereotactic radiosurgery (SRS), stereotactic body radiotherapy (SBRT), and MR-guided adaptive radiotherapy have enabled clinicians to deliver highly conformal doses while sparing adjacent normal tissues. However, the success of these techniques is contingent upon the fundamental assumption of patient immobility and reproducibility of positioning.

Unlike surgical or medical oncology interventions, radiotherapy requires repeated daily precision over several weeks, making even minor variations clinically significant. This challenge is further amplified in specific patient populations such as children, anxious individuals, and those with physical or neurological impairments.

Anaesthesia in radiotherapy thus serves three critical purposes:

1. Geometric stabilization
2. Physiological control
3. Psychological facilitation

The integration of anaesthesia into radiotherapy workflows represents a convergence of two specialties aimed at achieving optimal oncologic outcomes with maximal patient safety.

2. Radiobiological and Physical Basis for Anaesthesia Requirement

2.1 Margin Reduction and Precision Demands

Radiotherapy planning involves expansion from gross tumor volume (GTV) to clinical target volume (CTV) and planning target volume (PTV). With modern imaging and motion management:

·PTV margins have reduced to 3–5 mm (or less in SRS)
·Dose gradients are steep, often >10% per millimeter

Thus, even minimal displacement leads to:

·Significant underdosing of tumor
·Overdosing of critical structures

2.2 Intrafraction Motion: A Critical Determinant

Intrafraction motion arises from:

·Voluntary movement
·Respiratory motion
·Muscle relaxation variability

In paediatric patients or anxious adults, voluntary movement becomes the dominant factor, necessitating pharmacological immobilization.

2.3 Impact on Tumor Control Probability (TCP) and NTCP

Movement-induced errors directly affect:
·Tumor control probability (TCP)
·Normal tissue complication probability (NTCP)

Anaesthesia thus indirectly influences oncologic outcomes.

3. Indications

3.1 Pediatric Radiotherapy

The most important indication.
Challenges

·Cognitive immaturity
·Fear and anxiety
·Inability to tolerate immobilization devices

Clinical Implication

Nearly all children <6 years require anaesthesia; many up to 8–10 years depending on temperament.

3.2 Adult Indications

A. Psychological

·Severe claustrophobia
·Panic disorders

B. Physical

·Painful conditions
·Movement disorders (Parkinsonism, dystonia)

C. Neurological

·Altered sensorium
·Cognitive impairment

3.3 Procedural Indications

·Intracavitary brachytherapy
·Interstitial brachytherapy
·SRS with rigid immobilization
·Total body irradiation

4. Contraindications and Risk Stratification

Absolute Contraindications

·Unstable hemodynamics
·Severe hypoxia
·Airway compromise

Relative Contraindications

·Recent URTI (pediatrics)
·Difficult airway
·Severe anemia

Risk Stratification

ASA Grade	Risk Category	RT Anaesthesia Implication
I–II	Low risk	Routine Protocol, standard monitoring
III	Moderate	Enhanced monitoring, Pre-Anaesthetic Optimization
IV	High	ICU-level preparedness, Potential inpatient admission

5. Anaesthetic Techniques

5.1 General Anaesthesia

Indications

·Pediatric RT
·Interstitial brachytherapy
·Uncooperative patients

Technique

- General Anaesthesia (GA) - Preferred for Paediatric RT and interstitial brachytherapy.
- TIVA - is often preferred over inhalational for rapid turnover
- Airway:
  - Spontaneous
  - LMA
  - Intubation (complex cases)

5.2 Sedation Continuum

Level	Description	RT Use
Minimal	Anxiolysis	Claustrophobia
Moderate	Conscious sedation	Adult RT
Deep	Near GA	Complex immobilization

5.3 Regional Anaesthesia
Used in:
·Cervical brachytherapy
·Prostate implants
Advantages include superior analgesia and reduced systemic effects.

6. Pharmacology

Table 1. Advanced Pharmacological Profile of Anaesthetic Agents

Drug	Mechanism	Onset	Key Advantages	Limitations	RT Application
Propofol	$GABA_A$ agonist	Rapid	Predictable, antiemetic	Hypotension	Pediatric RT
Dexmedetomidine	α2 agonist	Moderate	Minimal respiratory depression	Bradycardia	Adult sedation
Midazolam	GABA agonist	Rapid	Amnesia	Respiratory depression	Anxiety
Fentanyl	μ-opioid agonist	Rapid	Analgesia	Respiratory depression	Brachytherapy
Ketamine	NMDA antagonist	Rapid	Preserves airway	Delirium	Unstable patients
Sevoflurane (Volatile)	$GABA_A$ agonist	Rapid	Smooth induction	Airway required	Pediatric induction

Agent Selection Logic

- Short duration + repeated sessions → Propofol
- Respiratory compromise → Dexmedetomidine
- Hemodynamic instability → Ketamine

7. Anaesthesia in Specific Clinical Scenarios

7.1 Pediatric Radiotherapy

- Current practice favours a 'De-escalation Hierarchy' where Behavioral Conditioning and Child-Life Specialists are utilized first. Anaesthesia is indicated when these fail.
- Daily GA for 4–6 weeks
- Plan as to reduce 'emergence delirium' and potentially mitigate long-term cognitive sequelae.
- Key principles :
    - Short-acting agents – prefer TIVA
    - Rapid turnover
    - Psychological conditioning

7.2 Brachytherapy

Intracavitary

- Painful → spinal/GA

Interstitial

- Requires deep anaesthesia

7.3 SRS / SBRT

- Requires submillimetric immobilization
- GA in pediatric cases

7.4 MR-Linac / Adaptive RT

- Longer treatment times
- Increased need for sedation

8. Radiotherapy Suite Challenges

Unique Constraints

- Remote location
- Limited access during beam-on
- Radiation exposure

Infrastructure Requirements

- MRI-compatible monitors
- Remote audiovisual systems
- Emergency access protocols

9. Monitoring and Safety Framework

Standard Monitoring

- ECG
- SpO_2
- NIBP
- Temperature

Advanced Monitoring

- BIS

- Capnography
- End-tidal CO_2

Radiation Safety

- Staff outside treatment room
- Remote monitoring mandatory

10. Role of Radiation Oncologist

Clinical Responsibilities

- Identify need for anaesthesia
- Optimize treatment duration
- Select appropriate technique

Operational Role

- Coordinate MDT
- Ensure SOP adherence
- Conduct audits

Strategic Role

- Integrate anaesthesia into workflow
- Improve efficiency and safety

11. Standard Operating Protocol

11.1 Pre-Procedure
- PAC clearance
- ASA grading
- Fasting confirmation
- Consent

11.2 Intra-Procedure
- Equipment check
- Continuous monitoring
- Remote supervision

11.3 Emergency Protocol
- Beam OFF
- Immediate access
- Resuscitation

- Code Blue in Bunker - 1. Beam OFF; 2. Door OPEN; 3. Table OUT
- Bunker-Crash-Cart

11.4 Post-Procedure
- Recovery monitoring
- Aldrete scoring
- Discharge

11.5 Documentation
- Anaesthesia chart
- Drug log
- Incident reporting

11.6 Quality Assurance
- Monthly audit
- Complication tracking
- SOP compliance

12. Complications and Management

Table 2. Advanced Complication Management

Complication	Pathophysiology	Management
Hypoxia	Airway obstruction	Airway reposition, O_2, LMA insertion.
Hypotension	Vasodilation	Fluids, vasopressors
Bradycardia	α2 effect	Atropine
Aspiration	Gastric contents	Intubation
Delirium	NMDA effect	Low-dose benzodiazepines

13. Emerging Trends

- AI-Predictive Monitoring

Emerging trends include the use of AI-driven predictive analytics that monitor subtle changes in EtCO2 or pulse plethysmography to predict airway obstruction before desaturation occurs.

- Virtual Reality (VR)

Pre-conditioning is being used to 'gamify' the immobilization process, effectively reducing the GA requirement in children aged 5–8 by up to 40%

- Dexmedetomidine protocols
- Behavioural conditioning

14. Institutional Implementation Model

Core Components
- Dedicated anaesthesia team
- SOP-driven workflow
- Integrated scheduling

Benefits
- Improved compliance
- Reduced errors
- Enhanced outcomes

15. Conclusion

Anaesthesia is no longer optional in modern radiotherapy—it is a precision-enabling intervention. Its integration into radiotherapy practice ensures:
- Geometric accuracy
- Reproducibility
- Patient safety

A structured, SOP-driven, multidisciplinary approach is essential for optimal outcomes.

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Sunrise at Netarhat

Netarhat is known for its natural beauty of hills, forest and waterfalls. Situated 155 kms from Ranchi, It is also referred to as the "Queen of Chotanagpur" famed for its lush forests, cool breeze, and stunning sunrise/sunset views, this former British retreat is popular for nature lovers, offering a quiet escape.

ARTICLE

From Evidence to Practice: Landmark Trials That Define Modern Oncology Care

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Introduction

Modern oncology practice is built upon evidence generated from landmark clinical trials, many of which directly define today's standards of radiotherapy, systemic therapy, and multidisciplinary care. For residents preparing for examinations such as FRCR, DM, DNB, and MD, familiarity with these trials is not optional—it is a core clinical competency. However, trainees often struggle to connect individual trial names with their real-world impact on day-to-day clinical decisions. This article is designed to bridge that gap by presenting key trials alongside the specific practice changes they produced. It can be used as a rapid revision tool before viva or theory exams, a framework for case discussions, and a teaching aid during academic sessions. Residents are encouraged to revisit these summaries repeatedly, focusing not only on results but on how each trial reshaped clinical standards. Used systematically, this resource will help trainees convert trial knowledge into confident clinical reasoning.

Lodh Falls
Lodh Falls is 468 feet (143 meters) in the midst of dense forests, ranking as one of the highest in India. Also known as Budha Ghagh, it is the highest waterfall in Jharkhand

MCQs (Q1–Q10)

Theme: Breast & Head and Neck Landmark Trials

Q1. A 52-year-old woman with pT2N0 invasive ductal carcinoma undergoes breast-conserving surgery. You plan adjuvant whole-breast irradiation using hypofractionation. Which regimen was validated as non-inferior to 50 Gy in 25 fractions in the START-B trial?
A. 41.6 Gy in 13 fractions
B. 40 Gy in 15 fractions
C. 42.5 Gy in 16 fractions
D. 26 Gy in 5 fractions
E. 50 Gy in 20 fractions

Q2. A department plans to adopt a 1-week adjuvant breast radiotherapy schedule. Which dose schedule was shown to be non-inferior in the FAST-Forward trial?
A. 24 Gy in 5 fractions
B. 28.5 Gy in 5 fractions
C. 26 Gy in 5 fractions
D. 30 Gy in 5 fractions
E. 40 Gy in 5 fractions

Q3. A 38-year-old woman undergoes breast-conserving surgery and radiotherapy. A tumor bed boost is being considered.
Which major outcome was demonstrated in the EORTC Boost Trial?
A. Improved overall survival
B. Reduced ipsilateral breast tumor recurrence
C. Reduced distant metastasis
D. No effect on local recurrence
E. Reduced cardiac toxicity

Q4. In a patient with node-positive breast cancer, regional nodal irradiation is considered.
Which major outcome was demonstrated in EORTC 22922?
A. Improved cosmetic outcomes
B. Reduced cardiac toxicity
C. Improved disease-free survival
D. No difference in survival
E. Reduced acute dermatitis

Q5. A patient with locally advanced head and neck cancer is planned for concurrent chemoradiotherapy.
What was the absolute overall survival benefit at 5 years demonstrated in the MACH-NC meta-analysis?
A. 2%
B. 4%
C. 6.5%
D. 10%
E. 15%

Q6. A 56-year-old patient with stage III laryngeal cancer is planned for induction chemotherapy prior to chemoradiotherapy.
Which regimen demonstrated improved survival in the TAX 324 trial?
A. PF (cisplatin + 5-FU)
B. TPF (docetaxel + cisplatin + 5-FU)
C. Carboplatin + paclitaxel
D. Cisplatin alone
E. Methotrexate + 5-FU

Q7. Which altered fractionation approach improved local control in the DAHANCA trials?
A. Hyperfractionation
B. Accelerated fractionation
C. Hypofractionation
D. Stereotactic boost
E. Weekly fractionation

Q8. Which fractionation schedule showed superior locoregional control in RTOG 9003?
A. Standard fractionation
B. Hyperfractionation
C. Accelerated fractionation with concomitant boost
D. Hypofractionation
E. Split-course radiotherapy

Q9. Which schedule was included in the START-A trial?
A. 41.6 Gy in 13 fractions
B. 40 Gy in 15 fractions
C. 26 Gy in 5 fractions
D. 30 Gy in 10 fractions
E. 45 Gy in 25 fractions

Q10. Which agents were compared in the RTOG 1016 trial in HPV-positive oropharyngeal cancer?
A. Cisplatin vs Cetuximab
B. Cisplatin vs Carboplatin
C. Cetuximab vs Nivolumab
D. Cisplatin vs Docetaxel
E. Carboplatin vs Paclitaxel

ANSWERS WITH TRIAL REFERENCES (Q1–Q10)

Q1 Answer: B — 40 Gy in 15 fractions

Trial Reference:
START-B Trial — Haviland et al., Lancet Oncology, 2013
The START-B trial compared 40 Gy/15 fractions over 3 weeks with 50 Gy/25 fractions in early breast cancer and showed non-inferior local-regional control and reduced late normal tissue toxicity with hypofractionation. This regimen became widely adopted internationally.
Why correct:
40 Gy/15# is the validated hypofractionated schedule from START-B.
Why others are wrong:
A: 41.6 Gy/13# — used in START-A, not START-B.
C: 42.5 Gy/16# — Canadian trial (Whelan et al., NEJM 2002).
D: 26 Gy/5# — from FAST-Forward trial.
E: 50 Gy/20# — not trial-based.
Key Take-home:
START-B established 40 Gy/15# as global standard hypofractionation.

Q2 Answer: C — 26 Gy in 5 fractions

Trial Reference:
FAST-Forward Trial — Murray Brunt et al., Lancet, 2020
The FAST-Forward trial demonstrated that 26 Gy in 5 fractions over 1 week was non-inferior to 40 Gy/15# in local control and late toxicity.
Why correct:
26 Gy/5# showed acceptable toxicity and equivalent tumor control.
Why others are wrong:
A: 24 Gy/5# — not tested.
B: 28.5 Gy/5# — associated with increased toxicity.
D: 30 Gy/5# — excessive dose.
E: 40 Gy/5# — unsafe biologically.
Key Take-home:
Ultra-hypofractionation (26 Gy/5#) is now accepted standard.

Q3 Answer: B — Reduced ipsilateral breast recurrence

Trial Reference:
EORTC Boost Trial — Bartelink et al., NEJM, 2001
The trial showed that adding a 16 Gy tumor bed boost significantly reduced ipsilateral breast tumor recurrence, especially in younger women.
Why correct:
Local recurrence reduction was the primary significant outcome.
Why others are wrong:
A: No survival benefit observed.
C: Distant metastasis unaffected.
D: Incorrect—recurrence reduction demonstrated.
E: Cardiac toxicity not studied.
Key Take-home:
Boost RT improves local control, especially in young patients.

Q4 Answer: C — Improved disease-free survival

Trial Reference:
EORTC 22922 — Poortmans et al., NEJM, 2015
Regional nodal irradiation improved disease-free survival and modestly improved overall survival.
Why correct:
DFS improvement was major outcome.
Why others are wrong:
A: Cosmetic outcome not endpoint.
B: Cardiac toxicity not primary focus.
D: Incorrect—survival improved.
E: Acute dermatitis not key outcome.
Key Take-home:
Regional nodal RT improves DFS and OS.

Q5 Answer: C — 6.5%

Trial Reference:
MACH-NC Meta-analysis — Pignon et al., Lancet, 2009
Concurrent chemotherapy improved 5-year overall survival by ~6.5%.
Why correct:
This figure is the accepted pooled survival benefit.
Why others are wrong:
A & B: Underestimate effect.
D & E: Overestimate benefit.
Key Take-home:
Concurrent CRT improves OS significantly.

Q6 Answer: B — TPF

Trial Reference:
TAX 324 — Posner et al., NEJM, 2007
TPF improved survival compared with PF induction chemotherapy.
Why correct:
TPF showed superior OS.
Why others are wrong:
A: PF inferior control arm.
C–E: Not tested in TAX 324.
Key Take-home:
TPF remains standard induction regimen.

Q7 Answer: B — Accelerated fractionation

Trial Reference:
DAHANCA 6 & 7 — Overgaard et al., Lancet, 2003
Reducing overall treatment time improved local control.
Why correct:
Acceleration reduced repopulation.
Why others are wrong:
Other fractionation schedules not tested.
Key Take-home:
Shorter treatment duration improves tumor control.

Q8 Answer: C — Concomitant boost

Trial Reference:
RTOG 9003 — Fu et al., JCO, 2000
Concomitant boost showed best locoregional control.
Why correct:
Superior control vs standard RT.
Why others are wrong:
Standard fractionation inferior.
Key Take-home:
Concomitant boost improves outcomes.

Q9 Answer: A — 41.6 Gy in 13 fractions

Trial Reference:
START-A — Bentzen et al., Lancet Oncology, 2008
START-A tested 41.6 Gy/13 fractions.
Why correct:
Correct START-A dose schedule.
Why others are wrong:
Belong to different trials.
Key Take-home:
START-A validated hypofractionation biology.

Q10 Answer: A — Cisplatin vs Cetuximab

Trial Reference:
RTOG 1016 — Gillison et al., Lancet, 2019
Cisplatin showed superior survival vs cetuximab.
Why correct:
Cetuximab inferior.
Why others are wrong:
Not trial comparisons.
Key Take-home:
Cisplatin remains standard radiosensitizer.

MCQs (Q11–Q20)
Theme: Lung Cancer Landmark Trials

Q11. A 62-year-old man with unresectable Stage III NSCLC completes concurrent chemoradiotherapy without progression.
Which consolidation therapy demonstrated significant improvement in progression-free and overall survival in this setting?
A. Pembrolizumab
B. Durvalumab
C. Nivolumab
D. Atezolizumab
E. Erlotinib

Q12. A department is reviewing dose escalation strategies in Stage III NSCLC. Which dose escalation strategy was associated with worse survival outcomes in RTOG 0617?
A. 60 Gy vs 70 Gy
B. 50 Gy vs 60 Gy
C. 66 Gy vs 74 Gy
D. 60 Gy vs 80 Gy
E. 45 Gy vs 60 Gy

Q13. Which major outcome was demonstrated in the CHART trial (Continuous Hyperfractionated Accelerated Radiotherapy) in NSCLC?
A. Improved distant metastasis control
B. Improved overall survival
C. Reduced toxicity
D. Improved nodal staging
E. Improved chemotherapy delivery

Q14. A patient with metastatic NSCLC and PD-L1 expression ≥50% is being evaluated for first-line therapy.
Which trial established pembrolizumab monotherapy as standard in this population?
A. KEYNOTE-189
B. KEYNOTE-024
C. CheckMate-017
D. PACIFIC
E. IMpower150

Q15. In previously treated metastatic squamous NSCLC, which trial demonstrated superiority of nivolumab over docetaxel?
A. KEYNOTE-042
B. CheckMate-017
C. CheckMate-057
D. PACIFIC
E. KEYNOTE-024

Q16. Which trial demonstrated the benefit of pembrolizumab combined with chemotherapy in metastatic non-squamous NSCLC?
A. KEYNOTE-024
B. KEYNOTE-189
C. CheckMate-017
D. PACIFIC
E. IMpower110

Q17. Which major radiotherapy strategy was evaluated in the RTOG 0617 trial besides dose escalation?
A. IMRT vs Proton therapy
B. IMRT vs 3DCRT
C. Sequential vs concurrent chemotherapy
D. SRS vs SBRT
E. Proton vs Carbon ion

Q18. Which trial demonstrated that atezolizumab improves survival in metastatic NSCLC after chemotherapy?
A. IMpower150
B. IMpower110
C. OAK trial
D. CheckMate-017
E. PACIFIC

Q19. Which trial evaluated EGFR-mutant Stage III NSCLC patients receiving osimertinib after chemoradiotherapy?
A. PACIFIC
B. LAURA
C. ADAURA
D. FLAURA
E. AURA3

Q20. Which survival benefit was demonstrated in the PACIFIC trial with durvalumab consolidation?
A. Improved local control only
B. Improved progression-free survival only
C. Improved overall survival only
D. Improved both PFS and OS
E. No survival benefit

ANSWERS WITH TRIAL REFERENCES (Q11–Q20)

Q11 Answer: B — Durvalumab

Trial Reference:
PACIFIC Trial — Antonia et al., NEJM 2017; Updated OS NEJM 2018
The PACIFIC trial demonstrated that durvalumab consolidation after concurrent chemoradiotherapy significantly improved both progression-free survival (PFS) and overall survival (OS) in unresectable Stage III NSCLC.
Why correct:
Durvalumab improved median PFS (16.8 vs 5.6 months) and OS significantly.
Why others are wrong:
A: Pembrolizumab studied mainly in metastatic setting.
B: Nivolumab not studied in this setting.
C: Atezolizumab not PACIFIC agent.
D: Erlotinib not standard post-CRT.
Key Take-home:
Durvalumab consolidation is standard of care after definitive CRT.

Q12 Answer: A — 60 Gy vs 70 Gy

Trial Reference:
RTOG 0617 — Bradley et al., Lancet Oncology 2015
This trial compared 60 Gy vs 74 Gy (not 70 Gy—exam trap) and showed worse survival with high-dose RT.
Why correct:
High-dose arm showed inferior OS (median 20.3 vs 28.7 months).
Why others are wrong:
B–E: Not actual RTOG 0617 comparisons.
Key Take-home:
Dose escalation beyond 60 Gy is harmful in Stage III NSCLC.

Q13 Answer: B — Improved overall survival

Trial Reference:
CHART Trial — Saunders et al., Lancet 1997
CHART demonstrated improved survival using accelerated hyperfractionation in NSCLC.
Why correct:
Shortened overall treatment time improved survival.
Why others are wrong:
A: Distant control not main outcome.
C: Toxicity increased slightly.
D & E: Not study endpoints.
Key Take-home:
Acceleration improves survival in NSCLC.

Q14 Answer: B — KEYNOTE-024

Trial Reference:
KEYNOTE-024 — Reck et al., NEJM 2016
Pembrolizumab monotherapy improved OS vs chemotherapy in PD-L1 ≥50%.
Why correct:
Established pembrolizumab as first-line therapy.
Why others are wrong:
A: Combination chemo trial.
C: Nivolumab study.
D: Stage III trial.
E: Atezolizumab trial.
Key Take-home:
PD-L1 ≥50% → pembrolizumab monotherapy.

Q15 Answer: B — CheckMate-017

Trial Reference:
CheckMate-017 — Brahmer et al., NEJM 2015
Demonstrated improved survival with nivolumab vs docetaxel in squamous NSCLC.
Why correct:
OS benefit demonstrated.
Why others are wrong:
C: Non-squamous NSCLC trial.
Others unrelated.
Key Take-home:
Nivolumab superior to docetaxel in squamous NSCLC.

Q16 Answer: B — KEYNOTE-189

Trial Reference:
KEYNOTE-189 — Gandhi et al., NEJM 2018
Pembrolizumab + chemotherapy improved survival in non-squamous NSCLC.
Why correct:
Major survival benefit.
Why others wrong:
Other trials test different settings.
Key Take-home:
Combination immunotherapy improved outcomes.

Q17 Answer: B — IMRT vs 3DCRT

Trial Reference:
RTOG 0617 Secondary Analysis
IMRT reduced pneumonitis and cardiac dose.
Why correct:
Planning modality comparison included.
Why others wrong:
Not trial components.
Key Take-home:
IMRT associated with reduced toxicity.

Q18 Answer: C — OAK Trial

Trial Reference:
OAK Trial — Rittmeyer et al., Lancet 2017
Atezolizumab improved survival vs docetaxel.
Why correct:
OS benefit confirmed.
Why others wrong:
Different trial settings.
Key Take-home:
Atezolizumab improves OS post-chemotherapy.

Q19 Answer: B — LAURA

Trial Reference:
LAURA Trial — Wu et al., NEJM 2024
Osimertinib improved PFS after CRT in EGFR-mutant Stage III NSCLC.
Why correct:
Major recent practice-changing study.
Why others wrong:
Different disease stages.
Key Take-home:
Osimertinib emerging standard in EGFR Stage III.

Q20 Answer: D — Improved both PFS and OS

Trial Reference:
PACIFIC Trial — Antonia et al., NEJM
Both survival endpoints improved.
Why correct:
Core PACIFIC finding.
Why others wrong:
Incomplete outcome statements.
Key Take-home:
PACIFIC reshaped Stage III NSCLC practice.

MCQs (Q21–Q30)
Theme: GI Oncology Landmark Trials

Q21. A 58-year-old male with locally advanced distal esophageal adenocarcinoma is planned for neoadjuvant therapy prior to surgery.
Which regimen was used in the CROSS trial?
A. 50.4 Gy with cisplatin + 5-FU
B. 41.4 Gy with weekly carboplatin + paclitaxel
C. 45 Gy with capecitabine
D. 50 Gy with FOLFOX
E. Chemotherapy alone

Q22. Which major outcome was demonstrated in the CROSS trial?
A. Improved pathological complete response without survival benefit
B. Improved overall survival with neoadjuvant chemoradiotherapy
C. No difference compared to surgery alone
D. Reduced surgical complications
E. Improved distant metastasis only

Q23. A 63-year-old patient with resectable gastric cancer is planned for perioperative chemotherapy.
Which regimen was used in the MAGIC trial?
A. FLOT
B. ECF (epirubicin, cisplatin, fluorouracil)
C. FOLFOX
D. CAPOX
E. Cisplatin alone

Q24. Which trial established FLOT chemotherapy as superior to ECF/ECX in resectable gastric cancer?
A. MAGIC
B. INT-0116
C. FLOT4
D. CLASSIC
E. ACTS-GC

Q25. A patient with gastric adenocarcinoma undergoes surgery with D0 resection.
Which trial demonstrated survival benefit with postoperative chemoradiotherapy?
A. MAGIC
B. INT-0116
C. FLOT4
D. CLASSIC
E. ARTIST

Q26. Which chemotherapy regimen was used in the INT-0116 trial?
A. Cisplatin + paclitaxel
B. 5-FU + leucovorin
C. Capecitabine alone
D. FLOT
E. FOLFOX

Q27. In locally advanced rectal cancer, which trial compared short-course RT followed by chemotherapy with standard long-course chemoradiation?
A. OPRA
B. RAPIDO
C. CROSS
D. PRODIGE 7
E. FOWARC

Q28. Which major outcome was demonstrated in the RAPIDO trial?
A. Reduced distant metastases
B. Increased local recurrence
C. Reduced toxicity
D. No difference in outcomes
E. Improved surgical margin rates only

Q29. Which trial evaluated total neoadjuvant therapy (TNT) strategies and organ preservation in rectal cancer?
A. RAPIDO
B. OPRA
C. PRODIGE 7
D. CROSS
E. FLOT4

Q30. Which trial evaluated the role of HIPEC in colorectal peritoneal metastases and showed no overall survival benefit?
A. PRODIGE 7
B. RAPIDO
C. OPRA
D. MAGIC
E. CROSS

ANSWERS WITH TRIAL REFERENCES (Q21–Q30)

Q21 Answer: B — 41.4 Gy with weekly carboplatin + paclitaxel

Trial Reference:
CROSS Trial — van Hagen et al., NEJM 2012
The CROSS trial used 41.4 Gy in 23 fractions with weekly carboplatin and paclitaxel, followed by surgery.
Why correct:
This regimen defines modern neoadjuvant CRT for esophageal cancer.
Why others are wrong:
A: Higher dose regimen not used in CROSS.
C: Capecitabine-only regimens not tested.
D: FOLFOX not CROSS regimen.
E: Chemotherapy alone inferior.
Key Take-home:
CROSS established weekly carbo-paclitaxel with 41.4 Gy as standard.

Q22 Answer: B — Improved overall survival

Trial Reference:
CROSS Trial — van Hagen et al., NEJM 2012
Median OS improved from 24 months to 49 months with neoadjuvant CRT.
Why correct:
Survival improvement is defining outcome.
Why others wrong:
A: OS benefit clearly demonstrated.
C: Incorrect—CRT superior.
D & E: Secondary observations only.
Key Take-home:
CROSS transformed esophageal cancer management.

Q23 Answer: B — ECF

Trial Reference:
MAGIC Trial — Cunningham et al., NEJM 2006
The MAGIC trial established perioperative ECF chemotherapy.
Why correct:
ECF was original MAGIC regimen.
Why others wrong:
A: FLOT used in FLOT4 trial.
C–E: Not MAGIC regimens.
Key Take-home:
MAGIC established perioperative chemotherapy paradigm.

Q24 Answer: C — FLOT4

Trial Reference:
FLOT4 Trial — Al-Batran et al., Lancet 2019
FLOT showed improved survival vs ECF/ECX.
Why correct:
FLOT superior to ECF.
Why others wrong:
A: MAGIC older regimen.
B: INT-0116 postoperative CRT.
D–E: Adjuvant chemo trials.
Key Take-home:
FLOT is current perioperative standard.

Q25 Answer: B — INT-0116

Trial Reference:
INT-0116 — Macdonald et al., NEJM 2001
Postoperative CRT improved survival.
Why correct:
Defined adjuvant CRT role.
Why others wrong:
Other trials test different settings.
Key Take-home:
Adjuvant CRT improves survival after limited lymphadenectomy.

Q26 Answer: B — 5-FU + leucovorin

Trial Reference:
INT-0116 — Macdonald et al., NEJM 2001
Chemotherapy backbone was 5-FU + leucovorin.
Why correct:
Correct regimen used in trial.
Why others wrong:
Other regimens used elsewhere.
Key Take-home:
Classic INT-0116 chemo backbone.

Q27 Answer: B — RAPIDO

Trial Reference:
RAPIDO Trial — Bahadoer et al., Lancet Oncology 2021
Compared short-course RT + chemotherapy vs standard CRT.
Why correct:
Core RAPIDO design.
Why others wrong:
Other trials different objectives.
Key Take-home:
RAPIDO introduced TNT paradigm.

Q28 Answer: A — Reduced distant metastases

Trial Reference:
RAPIDO Trial — Bahadoer et al., Lancet Oncology 2021
Reduced distant metastasis risk significantly.
Why correct:
Primary endpoint improvement.
Why others wrong:
Incorrect trial findings.
Key Take-home:
RAPIDO reduced systemic failure risk.

Q29 Answer: B — OPRA

Trial Reference:
OPRA Trial — Garcia-Aguilar et al., JCO 2022
Evaluated TNT sequencing and organ preservation.
Why correct:
Organ preservation strategy studied.
Why others wrong:
Other trials different aims.
Key Take-home:
OPRA supports watch-and-wait approach.

Q30 Answer: A — PRODIGE 7

Trial Reference:
PRODIGE 7 — Quenet et al., Lancet Oncology 2021
HIPEC did not improve survival.
Why correct:
No OS benefit shown.
Why others wrong:
Different disease settings.
Key Take-home:
HIPEC not routinely recommended.

MCQs (Q31–Q40)
Theme: GU & Gynecologic Oncology Landmark Trials

Q31. A 68-year-old man presents with newly diagnosed metastatic hormone-sensitive prostate cancer with low metastatic burden. He is receiving ADT. Which subgroup showed overall survival benefit with addition of prostate radiotherapy in the STAMPEDE RT arm?
A. High metastatic burden only
B. Low metastatic burden only
C. Both low and high burden
D. Node-positive M0 disease
E. Bone-only metastases

Q32. Which radiotherapy dose schedule was used for prostate irradiation in the STAMPEDE RT arm?
A. 74 Gy in 37 fractions
B. 55 Gy in 20 fractions
C. 60 Gy in 30 fractions
D. 66 Gy in 33 fractions
E. 45 Gy in 25 fractions

Q33. Which major outcome distinguished the CHAARTED trial from earlier androgen deprivation studies?
A. Demonstrated benefit of prostate RT
B. Demonstrated survival benefit of early docetaxel
C. Demonstrated superiority of abiraterone
D. Established SBRT role
E. Demonstrated benefit of pelvic nodal RT

Q34. In the CHAARTED trial, which subgroup showed the greatest survival benefit from early docetaxel?
A. Low-volume metastatic disease
B. High-volume metastatic disease
C. Node-positive only disease
D. Visceral metastasis only
E. PSA >100 only

Q35. Which defining inclusion criteria distinguished the LATITUDE trial population?
A. Any metastatic disease
B. High-risk metastatic hormone-sensitive prostate cancer
C. Castration-resistant disease
D. Node-positive disease only
E. Biochemical recurrence only

Q36. Which drug combination significantly improved survival in the LATITUDE trial?
A. ADT + Docetaxel
B. ADT + Abiraterone
C. ADT + Enzalutamide
D. ADT + Apalutamide
E. ADT + Cabazitaxel

Q37. A 64-year-old woman undergoes surgery for Stage IB Grade 3 endometrial carcinoma. Which trial established vaginal brachytherapy as comparable to pelvic EBRT with less toxicity?
A. PORTEC-1
B. PORTEC-2
C. PORTEC-3
D. GOG-99
E. EMBRACE

Q38. Which major outcome was demonstrated in the PORTEC-3 trial?
A. No benefit of chemotherapy
B. Improved overall survival with combined chemoradiation
C. Increased pelvic recurrence only
D. Reduced distant metastases only
E. No survival difference

Q39. Which dosimetric parameter was a key predictor of local control in the EMBRACE I trial for cervical cancer?
A. HR-CTV D90
B. Rectal D2cc
C. Bladder V50
D. Point A dose
E. Total treatment time only

Q40. Which major risk factors were identified in GOG-99 to define high-intermediate risk endometrial cancer?
A. Tumor size alone
B. Age, depth of invasion, grade
C. LVSI only
D. Cervical involvement
E. Lymph node positivity

ANSWERS WITH TRIAL REFERENCES (Q31–Q40)

Q31 Answer: B — Low metastatic burden only

Trial Reference:
STAMPEDE Trial (RT arm) — Parker et al., Lancet, 2018
The STAMPEDE RT arm showed that prostate radiotherapy improved overall survival only in patients with low metastatic burden, defined using CHAARTED criteria. No benefit was seen in high-burden disease.
Why correct:
Low-volume metastatic disease demonstrated statistically significant OS benefit.
Why others are wrong:
A: High-volume disease showed no OS benefit.
C: Combined benefit not observed.
D: M0 disease not included.
E: Bone-only not defining subgroup.
Key Take-home:
RT to primary improves survival only in low-volume metastatic disease.

Q32 Answer: B — 55 Gy in 20 fractions

Trial Reference:
STAMPEDE RT Protocol — Parker et al., Lancet, 2018
Two RT schedules were allowed: 55 Gy in 20 fractions or 36 Gy in 6 fractions weekly.
Why correct:
55 Gy/20# is most widely used schedule from the trial.
Why others wrong:
A: Radical prostate schedule, not STAMPEDE.
C–E: Not trial schedules.
Key Take-home:
55 Gy/20# is key STAMPEDE RT dose.

Q33 Answer: B — Early docetaxel improves survival

Trial Reference:
CHAARTED Trial — Sweeney et al., NEJM, 2015
The CHAARTED trial demonstrated significant overall survival benefit when docetaxel was added early to ADT.

Why correct:
Established early chemotherapy paradigm.
Why others wrong:
C: LATITUDE evaluated abiraterone.
Others unrelated.
Key Take-home:
Docetaxel early improves OS.

Q34 Answer: B — High-volume disease

Trial Reference:
CHAARTED Trial — Sweeney et al., NEJM, 2015
Greatest survival benefit seen in high-volume disease (visceral mets or ≥4 bone mets).
Why correct:
High-volume subgroup drove OS benefit.
Why others wrong:
Low-volume benefit smaller initially.
Key Take-home:
Docetaxel most beneficial in high-volume disease.

Q35 Answer: B — High-risk metastatic disease

Trial Reference:
LATITUDE Trial — Fizazi et al., NEJM, 2017
Included patients with high-risk metastatic disease, defined by ≥2 risk factors.
Why correct:
Distinctive inclusion criterion.
Why others wrong:
Other disease categories excluded.
Key Take-home:
LATITUDE targeted high-risk metastatic patients.

Q36 Answer: B — ADT + Abiraterone

Trial Reference:
LATITUDE Trial — Fizazi et al., NEJM, 2017
Addition of abiraterone significantly improved OS.
Why correct:
Major survival improvement demonstrated.
Why others wrong:
Other drugs tested in separate trials.
Key Take-home:
Abiraterone improves survival.

Q37 Answer: B — PORTEC-2

Trial Reference:
PORTEC-2 Trial — Nout et al., Lancet, 2010
Vaginal brachytherapy provided equivalent control with less toxicity.
Why correct:
Key finding of PORTEC-2.
Why others wrong:
Other trials tested different endpoints.
Key Take-home:
VBT preferred for intermediate-risk disease.

Q38 Answer: B — Improved overall survival

Trial Reference:
PORTEC-3 Trial — de Boer et al., Lancet Oncology, 2018
Combined chemoradiation improved survival in high-risk patients.
Why correct:
OS improvement demonstrated.
Why others wrong:
Incomplete statements.
Key Take-home:
Chemoradiation beneficial in high-risk disease.

Q39 Answer: A — HR-CTV D90

Trial Reference:
EMBRACE I Trial — Pötter et al., Lancet Oncology, 2021
HR-CTV D90 strongly predicted local control.
Why correct:
Primary dosimetric predictor.
Why others wrong:
Organs-at-risk parameters not predictors.
Key Take-home:
HR-CTV D90 critical in brachytherapy.

Q40 Answer: B — Age, depth, grade

Trial Reference:
GOG-99 — Keys et al., JCO 2004
Defined high-intermediate risk group.
Why correct:
Core defining variables.
Why others wrong:
Incomplete risk definitions.
Key Take-home:
Age + grade + invasion defines risk.

MCQs (Q41–Q50)
Theme: CNS + Immunotherapy + Precision Oncology Landmark Trials

Q41. A 58-year-old patient with newly diagnosed glioblastoma undergoes maximal safe resection.
Which regimen demonstrated improved survival in the EORTC-NCIC trial (Stupp trial)?
A. Radiotherapy alone (60 Gy)
B. Radiotherapy + concurrent and adjuvant temozolomide
C. Radiotherapy + lomustine
D. Radiotherapy + bevacizumab
E. Temozolomide alone

Q42. Which molecular subgroup derived the greatest survival benefit from temozolomide in the Stupp trial subgroup analysis?
A. EGFR amplification
B. MGMT promoter methylation
C. IDH mutation
D. TP53 mutation
E. ATRX mutation

Q43. Which major outcome was demonstrated in CheckMate-143, comparing nivolumab with bevacizumab in recurrent glioblastoma?
A. Improved overall survival
B. Improved progression-free survival
C. No overall survival benefit
D. Reduced toxicity
E. Improved neurological outcomes

Q44. Which radiotherapy strategy was evaluated in NRG-BN001 for glioblastoma?
A. Reduced dose radiotherapy
B. Dose escalation using IMRT
C. Proton therapy vs photon therapy
D. Hypofractionated radiotherapy
E. SRS boost alone

Q45. A patient with multiple brain metastases is evaluated for radiosurgery.
Which trial demonstrated improved survival when SRS was added to WBRT for patients with single brain metastasis?
A. RTOG 9508
B. EORTC 22952–26001
C. JROSG 99-1
D. NRG BN001
E. CheckMate-143

Q46. Which major finding was demonstrated in the EORTC 22952–26001 trial after SRS or surgery for brain metastases?
A. WBRT improved overall survival
B. WBRT reduced intracranial recurrence
C. WBRT reduced distant metastasis
D. WBRT improved neurocognition
E. WBRT reduced extracranial disease

Q47. In recurrent/metastatic head and neck cancer, which trial established pembrolizumab-based therapy as standard first-line treatment?
A. KEYNOTE-024
B. KEYNOTE-048
C. CheckMate-141
D. MACH-NC
E. TAX 324

Q48. Which major genomic assay-based trial guided adjuvant chemotherapy decisions in ER-positive, HER2-negative breast cancer?
A. MINDACT
B. TAILORx
C. KEYNOTE-189
D. NSABP B-06
E. HERA

Q49. Which trial validated the 70-gene signature (MammaPrint) in breast cancer risk stratification?
A. TAILORx
B. MINDACT
C. HERA
D. NSABP B-28
E. ATLAS

Q50. Which subgroup showed no survival benefit with addition of bevacizumab in newly diagnosed glioblastoma trials?
A. MGMT methylated
B. MGMT unmethylated
C. All molecular subgroups
D. IDH mutant
E. EGFR amplified

ANSWERS WITH TRIAL REFERENCES (Q41–Q50)

Q41 Answer: B — Radiotherapy + concurrent and adjuvant temozolomide

Trial Reference:
EORTC-NCIC Trial (Stupp Trial) — Stupp et al., NEJM 2005
The landmark Stupp trial showed that adding concurrent and adjuvant temozolomide to 60 Gy RT significantly improved median survival (14.6 vs 12.1 months) and 2-year survival (26% vs 10%).
Why correct:
Concurrent + adjuvant temozolomide became standard of care.
Why others wrong:
A: RT alone inferior survival.
C: Lomustine not tested in this trial.
D: Bevacizumab failed to improve OS in upfront setting.
E: TMZ alone inadequate therapy.
Key Take-home:
RT + TMZ defines modern GBM standard.

Q42 Answer: B — MGMT promoter methylation

Trial Reference:
Hegi et al., NEJM 2005 — Stupp Trial Molecular Analysis
MGMT methylated tumors showed marked survival benefit from temozolomide.
Why correct:
MGMT methylation predicts TMZ sensitivity.
Why others wrong:
Other mutations not predictive in this trial.
Key Take-home:
MGMT methylation is strongest predictive biomarker.

Q43 Answer: C — No overall survival benefit

Trial Reference:
CheckMate-143 — Reardon et al., Lancet Oncology 2017
Nivolumab failed to improve OS compared to bevacizumab.
Why correct:
No survival advantage demonstrated.
Why others wrong:
No improvement in survival endpoints.

Key Take-home:
Immunotherapy not superior in recurrent GBM.

Q44 Answer: B — Dose escalation using IMRT

Trial Reference:
NRG-BN001 — Tsien et al., JCO 2019 (Phase I/II)
Evaluated safe dose escalation with IMRT.
Why correct:
Focus was dose escalation feasibility.
Why others wrong:
Other techniques not central.
Key Take-home:
Dose escalation under investigation.

Q45 Answer: A — RTOG 9508

Trial Reference:
RTOG 9508 — Andrews et al., Lancet 2004
SRS + WBRT improved survival in single metastasis patients.
Why correct:
Key survival improvement demonstrated.
Why others wrong:
Other trials evaluated recurrence endpoints.
Key Take-home:
SRS improves survival in single metastasis.

Q46 Answer: B — WBRT reduced intracranial recurrence

Trial Reference:
EORTC 22952–26001 — Kocher et al., Lancet Oncology 2011
WBRT reduced intracranial recurrence but did not improve survival.
Why correct:
Local and distant brain relapse reduced.
Why others wrong:
No OS benefit demonstrated.
Key Take-home:
WBRT improves control but not survival.

Q47 Answer: B — KEYNOTE-048

Trial Reference:
KEYNOTE-048 — Burtness et al., Lancet 2019
Pembrolizumab improved OS in recurrent/metastatic HNSCC.
Why correct:
Established pembrolizumab first-line.
Why others wrong:
Different disease settings.
Key Take-home:
Pembrolizumab standard in R/M HNSCC.

Q48 Answer: B — TAILORx

Trial Reference:
TAILORx — Sparano et al., NEJM 2018
Guided chemotherapy decisions based on recurrence score.
Why correct:
Established genomic-guided therapy.
Why others wrong:
Different genomic platforms.
Key Take-home:
Genomic assays personalize therapy.

Q49 Answer: B — MINDACT

Trial Reference:
MINDACT — Cardoso et al., NEJM 2016
Validated 70-gene signature (MammaPrint).
Why correct:
Central genomic validation study.
Why others wrong:
Other trials use different assays.
Key Take-home:
MammaPrint validated genomic risk stratification.

Q50 Answer: C — All molecular subgroups

Trial Reference:
AVAglio & RTOG 0825 — Chinot et al., NEJM 2014
Bevacizumab failed to improve overall survival in all subgroups.
Why correct:
No OS benefit across molecular groups.
Why others wrong:
No subgroup showed survival advantage.
Key Take-home:
Bevacizumab improves PFS but not OS.

Trial vs Practice Change in Clinical and Radiation Oncology

Breast oncology

START-B
Trial message: 40 Gy in 15 fractions over 3 weeks was comparable to 50 Gy in 25 fractions, with less late toxicity.
Practice change: Hypofractionated whole-breast RT became standard for most early breast cancers.
FAST-Forward
Trial message: 26 Gy in 5 fractions over 1 week was non-inferior to 40 Gy in 15 fractions for local control and acceptable toxicity.
Practice change: Ultra-hypofractionated adjuvant breast RT entered routine practice in selected patients.
EORTC Boost Trial
Trial message: Tumor bed boost reduced ipsilateral breast recurrence, especially in younger women.
Practice change: Boost is strongly considered in young age, high grade, close margins, and other higher-risk settings.
EORTC 22922 / MA.20
Trial message: Regional nodal irradiation improved disease-free outcomes, with modest survival gains in selected node-positive/high-risk node-negative patients.
Practice change: Broader use of supraclavicular/internal mammary nodal irradiation in appropriately selected cases.
TAILORx
Trial message: Many ER-positive, HER2-negative, node-negative patients with intermediate recurrence scores did not benefit from chemotherapy.
Practice change: Genomic assays began guiding omission of chemotherapy.
MINDACT

Trial message: Genomic low risk can identify patients who may safely avoid chemotherapy despite clinical high risk.
Practice change: MammaPrint gained a role in chemo decision-making in selected early breast cancer.

Head and neck oncology

MACH-NC
Trial message: Concurrent chemotherapy provides the greatest survival benefit compared with induction or adjuvant approaches.
Practice change: Cisplatin-based concurrent chemoradiation became standard for many locally advanced HNSCC cases.
TAX 324
Trial message: TPF was superior to PF as induction chemotherapy.
Practice change: When induction is chosen, TPF became the preferred regimen.
DAHANCA 6/7
Trial message: Shortening treatment time improved locoregional control.
Practice change: Repopulation became a central practical consideration; accelerated schedules gained importance.
RTOG 9003
Trial message: Altered fractionation, especially concomitant boost, improved locoregional control.
Practice change: Altered fractionation became an evidence-based option when chemotherapy is unsuitable or in selected organ-preservation settings.
RTOG 1016 / De-ESCALaTE HPV
Trial message: Cetuximab was inferior to cisplatin in HPV-positive oropharyngeal cancer.
Practice change: Cisplatin remained standard radiosensitizer; cetuximab should not be used as a de-intensification substitute.
KEYNOTE-048
Trial message: Pembrolizumab, alone or with chemotherapy, improved OS in recurrent/metastatic HNSCC depending on PD-L1 status.
Practice change: PD-L1-guided immunotherapy became first-line standard in recurrent/metastatic disease.

Lung oncology

PACIFIC
Trial message: Durvalumab after concurrent CRT significantly improved PFS and OS in unresectable Stage III NSCLC.
Practice change: Consolidation durvalumab became standard after definitive concurrent CRT.
RTOG 0617
Trial message: Dose escalation to 74 Gy worsened survival compared with 60 Gy.
Practice change: 60 Gy remained standard definitive dose in Stage III NSCLC; "more dose is better" was rejected.
CHART
Trial message: Accelerated hyperfractionation improved survival in NSCLC.
Practice change: Reinforced importance of overall treatment time and radiobiology in thoracic RT.
KEYNOTE-024
Trial message: Pembrolizumab improved survival over chemotherapy in metastatic NSCLC with PD-L1 ≥50%.
Practice change: Single-agent immunotherapy became first-line standard in high PD-L1 disease.
KEYNOTE-189
Trial message: Pembrolizumab plus chemotherapy improved OS in metastatic non-squamous NSCLC.
Practice change: Chemo-immunotherapy became standard for many patients irrespective of very high PD-L1 dependence.
CheckMate-017 / 057
Trial message: Nivolumab outperformed docetaxel in previously treated squamous and non-squamous NSCLC.
Practice change: Immunotherapy displaced docetaxel in second-line metastatic NSCLC.
LAURA
Trial message: Osimertinib markedly improved PFS after CRT in EGFR-mutant unresectable Stage III NSCLC.
Practice change: Molecular stratification entered post-CRT consolidation strategy in EGFR-mutant Stage III disease.

Esophagus and upper GI

CROSS
Trial message: Neoadjuvant chemoradiotherapy with weekly carboplatin-paclitaxel plus 41.4 Gy improved R0 resection, pCR, and OS over surgery alone.
Practice change: CROSS-type neoadjuvant CRT became a major standard for resectable locally advanced esophageal/GEJ cancer.
MAGIC
Trial message: Perioperative ECF improved survival over surgery alone in gastric/GEJ adenocarcinoma.
Practice change: Perioperative chemotherapy became standard in resectable gastric cancer.
FLOT4
Trial message: FLOT was superior to ECF/ECX.
Practice change: FLOT replaced older perioperative chemotherapy regimens in fit patients.
INT-0116
Trial message: Postoperative chemoradiotherapy improved survival after surgery for gastric cancer, especially when surgery was less than optimal D2.
Practice change: Adjuvant CRT became important particularly after inadequate nodal dissection or selected high-risk situations.

Rectal and colorectal oncology

German CAO/ARO/AIO-94
Trial message: Preoperative CRT had lower toxicity and better local control than postoperative CRT.
Practice change: Preoperative treatment became standard for locally advanced rectal cancer.
RAPIDO
Trial message: Short-course RT followed by systemic chemotherapy reduced disease-related treatment failure, largely via lower distant metastases.
Practice change: TNT became mainstream for high-risk rectal cancer.
PRODIGE 23
Trial message: Induction FOLFIRINOX before CRT improved disease-free and metastasis-free outcomes.
Practice change: TNT intensified further in fit, high-risk patients.
OPRA

Trial message: TNT with response-adapted nonoperative management improved organ preservation possibilities. Practice change: Watch-and-wait became more structured in complete clinical responders.

PRODIGE 7

Trial message: HIPEC did not improve OS in colorectal peritoneal metastases in that setting.

Practice change: Routine oxaliplatin-based HIPEC was questioned and often avoided outside selected contexts.

Prostate oncology

CHAARTED

Trial message: Early docetaxel added to ADT improved OS, particularly in high-volume metastatic hormone-sensitive disease.

Practice change: Upfront intensification became standard in mHSPC.

LATITUDE

Trial message: Abiraterone plus ADT improved OS in high-risk metastatic hormone-sensitive prostate cancer.

Practice change: AR pathway intensification became standard first-line treatment.

STAMPEDE (multiple arms)

Trial message: Repeatedly showed survival gains with systemic intensification; RT to primary improved OS in low-volume metastatic disease.

Practice change: STAMPEDE reshaped prostate cancer broadly—docetaxel, abiraterone, ARPI intensification, and prostate RT in low-volume M1 disease all entered routine practice.

HORRAD

Trial message: No clear OS benefit overall from prostate RT in metastatic disease, though low-volume signals existed.

Practice change: Helped frame later interpretation of STAMPEDE; primary RT considered mainly for low-burden disease.

HYPO-RT-PC / PACE / PROFIT

Trial message: Hypofractionated and in some settings ultra-hypofractionated RT can provide comparable control.

Practice change: Moderate hypofractionation became standard in localized prostate cancer; SBRT expanded in selected settings.

Gynecologic oncology

PORTEC-1
Trial message: Pelvic RT reduced locoregional recurrence in intermediate-risk endometrial cancer, without OS benefit.
Practice change: Adjuvant RT was used selectively for local control rather than routinely for everyone.
GOG-99
Trial message: Defined high-intermediate-risk endometrial cancer using age, grade, and depth of invasion.
Practice change: Risk-adapted adjuvant treatment became standard.
PORTEC-2
Trial message: Vaginal brachytherapy provided equivalent vaginal control to pelvic RT with less toxicity in high-intermediate-risk disease.
Practice change: VBT became preferred adjuvant treatment for many high-intermediate-risk endometrial cancer patients.
PORTEC-3
Trial message: Chemoradiation improved outcomes in high-risk endometrial cancer, particularly stage III and serous/high-risk subsets.
Practice change: Combined modality treatment gained traction in high-risk endometrial carcinoma.
EMBRACE I
Trial message: MRI-guided adaptive brachytherapy with dosimetric optimization achieved high local control with acceptable morbidity in cervical cancer.
Practice change: Image-guided adaptive brachytherapy became the modern cervical brachytherapy standard; HR-CTV D90 became central.

CNS oncology

EORTC-NCIC / Stupp trial
Trial message: Concurrent and adjuvant temozolomide with RT improved survival in glioblastoma.
Practice change: RT plus TMZ became standard upfront therapy for GBM.
Hegi MGMT analysis
Trial message: MGMT promoter methylation predicts greater benefit from temozolomide.
Practice change: MGMT became essential for prognostication and treatment tailoring discussions.
RTOG 9508
Trial message: WBRT plus SRS improved survival in patients with a single brain metastasis.
Practice change: SRS gained firm evidence-based use in selected limited brain metastases.
EORTC 22952–26001

Trial message: Adjuvant WBRT after surgery/SRS reduced intracranial relapse but did not improve OS. Practice change: Routine WBRT after local therapy became less favored; surveillance strategies expanded.
JROSG 99-1
Trial message: SRS alone preserved cognition better but increased intracranial recurrence versus SRS + WBRT.
Practice change: SRS alone became acceptable for selected limited brain metastases with close MRI follow-up.
CheckMate-143
Trial message: Nivolumab did not improve OS over bevacizumab in recurrent GBM.
Practice change: Immunotherapy did not become standard in unselected recurrent GBM.

Precision oncology and systemic therapy across tumors

HERA
Trial message: Adjuvant trastuzumab improved outcomes in HER2-positive breast cancer.
Practice change: One year of trastuzumab became standard adjuvant therapy.
CLEOPATRA
Trial message: Pertuzumab plus trastuzumab and docetaxel improved survival in metastatic HER2-positive breast cancer.
Practice change: Dual HER2 blockade became first-line metastatic standard.
EMILIA
Trial message: T-DM1 improved outcomes over lapatinib-capecitabine in previously treated HER2-positive disease.
Practice change: Antibody-drug conjugates moved to the center of HER2 treatment sequencing.
CheckMate-214
Trial message: Nivolumab-ipilimumab improved OS in intermediate/poor-risk metastatic RCC.
Practice change: Immune combination became frontline standard in appropriate RCC risk groups.

Congratulations

51ST ICRO QUIZ WINNERS

Dr Omal Shereef
JR2 at Mahavir Cancer Sansthan Patna

1

Dr Avilash Banerjee
JR3 at Yashoda Super Speciality Hospital Hyderabad

2

Parasnath Hill

Parasnath Hill, located in Jharkhand's Giridih district, is the highest mountain in the state at 1,350 meters. Known as Sammed Sikharji, it is the holiest Jain pilgrimage site, where 20 of the 24 Tirthankaras attained salvation.

FROM THE OFFICE OF AROI

Applications Invited for: Fellowships/ Grants/ Best Papers

S.No.	Name of Fellowship	No's	For	Age Group	Fellowship Grant (in INR)	Basis	Member of AROI For #/yrs.	Min Papers	Regularly Attending AROI conferences	Already availed fellowship in the past
1. Overseas										
1.1	AROI Fellowship	1	Radiation Oncologist	>50	1.5 Lakhs	MD/DNB	20	5	Yes	Then weightage to be given To those who have not Availed any Fellowship (or Any other Candidate is not available)
1.2	AROI Fellowship	2	Radiation Oncologist	41-50	1.5 Lakhs	MD/DNB	10	5	Yes	
1.3	AROI Fellowship	3	Radiation Oncologist	35-40	1 Lakh	MD/DNB	5	3	Yes	
1.4	AROI Fellowship	4	Radiation Oncologist	30-35	1 Lakh	MD/DNB	3	3	Yes	
2. Within India at higher centres										
2.1	AROI Fellowship	1	Radiation Oncologist	30-35	30,000	MD/DNB	3	1	Yes	
2.2	AROI Fellowship	1	Medical Physicist	< 40	30,000	DRP/MSc (MP)	2	BASED ON THE RESUME AND INTERVIEW AT THE CONFERENCE & PREFERENCE GIVEN TO PAPER PRESENTERS	Yes	
2.3	AROI Fellowship	1	RT Technologist	<45	10,000	AERB Certified	Yes		No	
2.4	Neil Joseph Fellowship	6	2nd & 3rd year PG student		25,000	Student MD/DNB	Yes	RESUME AND INTERVIEW		

3. AROI Best Paper Awards

3.1	Best Proffered Paper for Senior Members	1	Radiation Oncologist	>40 - ≤50	Only certificate	Post MD/DNB >10 Yrs of experience	10-15 yrs of LM	
3.2	Best Proffered Paper for Senior Members	1	Radiation Oncologist	-≤40	Only certificate	Post MD/DNB Min 5yrs of experience	5-10 yrs of LM	
3.3	Dr. K. T. Bhownik Young Doctor Award	1	Radiation Oncologist Post MD/DNB	<40	Plaque + 30,000 For fellowship	Post MD/DNB	Min 3 yrs of LM	
3.4	Dr. M. S. Gujral Gold Medal	1	MD/DNB Students	<30	20,000+Medal		Yes	
3.5	Dr. M. C. Pant Gold Medal	1	MD /DNB Students	<30	15,000+Medal		Yes	
3.6	Gold Medal Medical Physics	1	Physicist/Radiation oncologist with physicist	<40	10,000 + Medal	DRP/MSc in Med. Physics	Yes	

FROM THE OFFICE OF AROI

Applications Invited for: Fellowships/ Grants/ Best Papers

Procedure for Fellowship Application:

1. Fellowship applicants have to email their CV with publication details, AROI LM number, a copy of their date of birth certificate, Self-certified proclamation that they are working full time in Radiation Oncology along with AROICON registration number.
2. All the documents mentioned above needs to carry during interview.
3. No Objection certificate (NOC) from their Head of Department to travel for 4weeks for fellowship, if get selected.
4. ICRO membership is mandatory for fellowships more than 35 years of age category.
5. Fellowship amount will be given to candidates after 15% deduction.
6. Fellowship must be completed before **31st October 2027**.
7. All the applications for the fellowships to be emailed to the office of Secretary General AROI by **5 PM, 31st August 2026. Applications received after deadline will not be entertained.**

Procedure for Best Papers/ Grants Application:

1. All the applications for best paper awards to be emailed along with the abstract & full paper in word copy, CV, DOB certificate & AROI LM number to the Secretary General AROI by **5 PM, 31st August 2026.**
2. PG Students shall send recommendation for presenting Best paper through Head of the Department.
3. NOC for publication in JCRT (if selected) is required. PG students should approach for best paper through their HOD/guide.
4. All the documents mentioned above needs to carry during the presentation.
5. AROICON Registration is mandatory before applying for any fellowship / Best paper.
6. AROI membership is mandatory before applying for any fellowship / Best paper.

Email address and details -

a) Dr. V Srinivasan, Secretary General AROI: secretaryaroi@gmail.com
b) Dr. S. N. Senapati, President AROI: snsenapati2007@gmail.com

FROM THE OFFICE OF AROI

BIDS INVITED FOR AROI NATIONAL CONFERENCE, AROI – ICRO / AROI – ESTRO COURSES & OTHER COURSES-

Bids are invited to hold-

1. **National AROI Conference of 2028**

2. **AROI- ICRO Teaching Program 2028-**
   a) AROI-ICRO SUN PG Teaching Courses (3 courses)

3. **AROI Teaching Courses endorsed by ESTRO for 2028**
   a) AROI Gynae Teaching Course endorsed by ESTRO
   b) AROI Advanced Technologies Teaching course endorsed by ESTRO

4. **AROI - ESTRO Teaching Course for 2028**
   a) Head & Neck Teaching course

5. **Best of ASTRO – 2027 & 2028**

6. **YROC 2028**

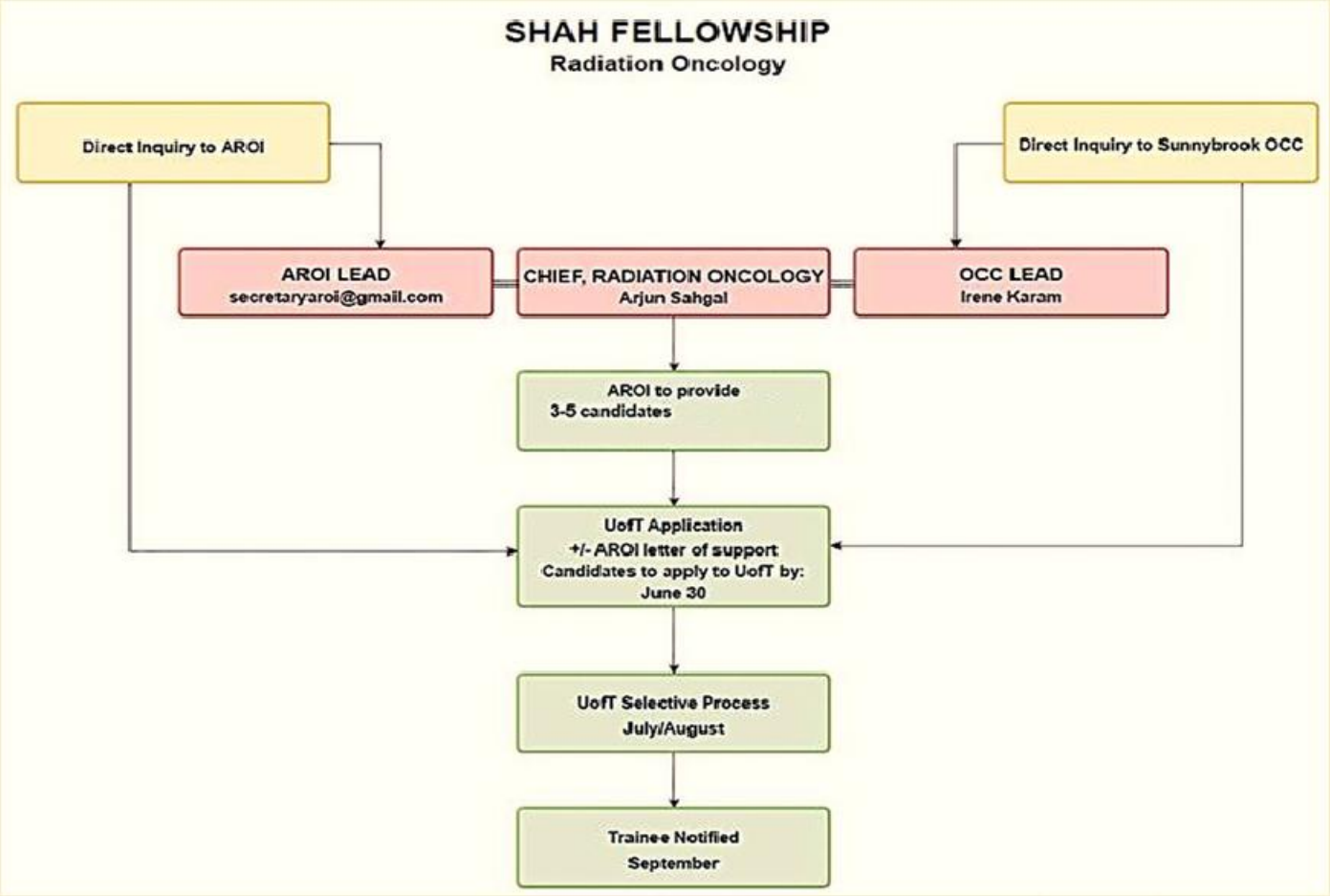
How to Apply

- Application forwarded by Head of the Department/ Head of the Institute
- Should be through Zonal / State Chapter of AROI
- Application should reach to Dr. V. Srinivasan, Secretary General AROI, by 31st August 2026.
  E-mail: secretaryaroi@gmail.com

Dr. S N Senapati
President AROI

Dr. V Srinivasan
Secretary General AROI

APPLICATION FOR SHAH FELLOWSHIP AT UNIVERSITY OF TORONTO–



* **UofT**- University of Toronto.

Selection criteria:

a) Post MD/DNB students within 3 years of their qualification will be eligible
b) Five candidates will be selected by AROI during 46th AROICON 2026 at Hyderabad.
c) The Fellowship will provide the requisite funding to the selected Fellow (1/5) for one year, after securing the admittance to University of Toronto as per the University norms.
d) The selected Fellow will have to return to India after completion of the Fellowship and letter to this effect as well as recommendation letter for the same will be provided by the Employer as well as AROI before the beginning of the Fellowship.

RAJAROICON 2025

10th and 11th January 2026

RAJ-AROICON 2025, the annual conference of the Rajasthan Chapter of the Association of Radiation Oncology of India, was held on 10th and 11th January 2026 at GBH Cancer Hospital, Udaipur. The conference was attended by over 200 delegates and faculty members, making it a vibrant academic gathering of radiation oncologists, academicians, and trainees. The inaugural event was graced by Dr. Kirti Kumar Jain, CMD, GBH Group of Hospitals; Dr. S. N. Senapati, President AROI; Dr. V. Srinivasan, Secretary General AROI; Dr. R. S. Gothwal, President RAJ AROI, and Dr. Ramesh Purohit, Secretary RAJ AROI. The conference was hosted by GBH Cancer Hospital with Dr. Mamta Lodha, as Organising Chairperson, along with Dr. Manan Sarupria, Organising Secretary and Dr. Vibhor Patodi, Co-Organising Secretary, playing a key role in coordination and execution of the event. Theme of the conference was "Innovating Cancer Care: Precision, Compassion and Collaboration". The event featured high-quality scientific sessions, interactive discussions, and active delegate participation, reinforcing the commitment to academic excellence and collaborative cancer care. Over 100 abstracts were received for paper and poster presentations showing growing interest from the residents. An oncology quiz competition was organized for the residents with participation of a total of 11 teams having three candidates in each team. Two senior radiation oncologists were felicitated with Life time achievement awards, Dr SK Dangayach and Dr RK Tanwar.

12TH YOUNG RADIATION ONCOLOGISTS CONFERENCE (YROC)

24th and 25th of January 2026

The 12th Young Radiation Oncologists Conference (YROC), 1st national level program of AROI for 2026 calendar year was successfully conducted by Kidwai Memorial Institute of Oncology in association with Association of Radiation Oncologists of Karnataka (AROK) on the 24th and 25th of January 2026 at NIMHANS convention center, Bengaluru. The Conference was based on the theme "Beyond the Beam: Pushing Boundaries, Transforming Outcomes" to have certain focused discussion. The program was formally inaugurated by the Chief Guest Dr. Bhagavan B C Honorable Vice-Chancellor RGUHS, Karnataka, Dr. Naveen T Director (Addi. Charge) Kidwai Memorial Institute of Oncology, Dr. Vikas Jagtap Junior Vice President AROI and YROF Convenor, Dr. V. Srinivasan Secretary General AROI , Dr. Siddanna R Palled President AROK, Dr. Sunil R. A. Secretary AROK, Dr. Shamsundar S D YROC Organizing Secretary. During the inaugural session, the dignitaries emphasized the significance of gaining in-depth knowledge in exploring the role of radiation therapy in newer diseases like nonmalignant conditions. They also stressed the necessity of topic oriented focused discussion to equip the Radiation Oncology residents to manage these complex oncology cases and also involving the oncology community in better screening programs to detect the cases in early stage only. The 12th YROC witnessed a record of more than 475 registrations including delegates and faculties. More than 225 abstracts of scientific research were presented by Radiation Oncology post graduate students and Delegates as either Oral or Poster presentations. The conference was divided into 6 scientific sessions, namely- Hypo-Fractionation, Reirradiation, Toxicity and Survivorship, Omics in Radiation Oncology, Radiation in Non-Malignancy and Advanced Technologies. Each session consisted of lectures and Panel Discussion, so that the scientific learning would be more interactive. More than 70 distinguished faculties across Globally and India took part in the scientific program. As AROI is celebrating its Golden Jubilee year, Dr. Ramesh Bilimagga Past President AROI gave a talk on Glorious 50 years of AROI, to create awareness among the delegates as to how AROI has evolved over last 50 years. The event concluded with a quiz competition. The program concluded with a valedictory function and distribution of cash prize and certificates to the winners of oral and poster presentations for both PG and Post PG category, Quiz Winners and runners.

12TH YOUNG RADIATION ONCOLOGISTS CONFERENCE (YROC)

24th and 25th of January 2026

UPCOMING EVENTS TO ATTEND

AROI calendar 2026-

1. **46th AROICON 2026 -**

At HICC & Novotel Hyderabad by Telangana State Chapter- 3rd- 6th Dec 2026

2. **ICRO SUN PG 2026 -**

- On Thoracic malignancies at Cancer Institute Adyar, Chennai- Dr Priya Iyer- 4th & 5th Jul 26.
- On Stereotaxy at Cancer hospital, IGMC Shimla- Dr Manish Gupta- 5th & 6th Sep 26.

3. **AROI-ESTRO & AROI TEACHING COURSES ENDORSED BY ESTRO 2026 -**

- 4th AROI ESTRO H & N COURSE- Dr Shalini Singh at SGPGIMS Lucknow- 4th -6th Jun 26
- 13th AROI Advanced Technologies Teaching course endorsed by ESTRO- Dr Pardeep Garg from GGSMCH, Faridkot. Venue will be Chandigarh. – Jan 2027 (dates to be fixed)

4. **8th BEST OF ASTRO 2026 -**

Dr Kanika Sharma Sood at Dharamshila Narayana Superspeciality Hospital, Delhi- 1st & 2nd of August 2026.

5. **ICRO-INTAS RADIOBIOLOGY COURSE 2026 -**

Dr Manoj Gupta at SGRR Institute of Medical and Health Sciences, Dehradun (UK) –Nov 2026 (tentative schedule).

STEREO-ONC 2026

23rd May 2026

50 YEARS
Excellence in Radiation Oncology

IBSCON 2026

16th Annual Conference of Indian Brachytherapy Society

20-22, Aug 2026

Re-imagining Brachytherapy In
Comprehensive Cancer Care
Precision : Personalisation : Compassion

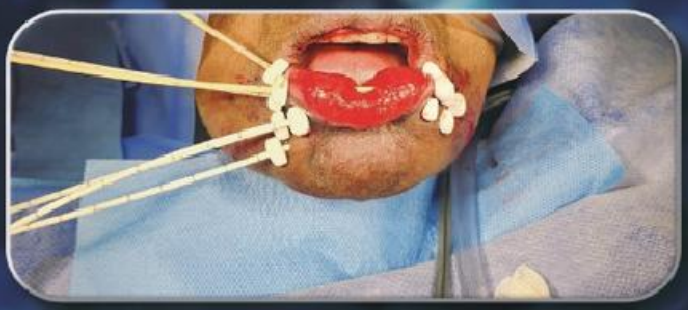
Ida B Scudder Cancer Center
Christian Medical College Vellore
Serving the nation since 1900

WELCOME TO IBSCON 2026

With great pleasure, we welcome you to be a part of the 16th Annual Conference of the Indian Brachytherapy Society – IBSCON 2026, to be held at Christian Medical College, Vellore, from August 20–22, 2026.

CMC Vellore, with its 125-year legacy of compassionate, patient-centred healthcare and clinical excellence, is honoured to host this national academic event. Our Department of Radiation Oncology—now in its 87th year—remains deeply committed to advancing high-quality cancer care that combines innovation, precision, and care.

The theme for this year's conference, "Re-imagining Brachytherapy in Comprehensive Cancer Care – Precision: Personalization: Compassion," captures our vision of integrating technological advancements with the enduring values of patient comfort and individualized treatment.



IBSCON 2026

20 - 22 August 2026

50 YEARS
Excellence in Radiation Oncology

IBSCON 2026 aims to bring together experts in various fields of brachytherapy, physicists, researchers, and trainees from India and around the world. The scientific programme is being thoughtfully crafted to highlight emerging developments across brachytherapy, share pioneering research, and promote practical insights that can be applied directly to patient care. We hope that this platform will not only enrich scientific understanding but also strengthen collaborations and inspire confidence in practising evidence-based brachytherapy.

We warmly invite you to join us in Vellore for three days of learning, meaningful exchange, and collegiality. We look forward to welcoming you to IBSCON 2026 and hope you will take back rich academic insights, renewed connections, and lasting memories.

Dr. Patricia Sebastian
Organizing Secretary, IBSCON 2026
Department of Radiation Oncology
Christian Medical College, Vellore

Keynote speaker

Dr José Luis Guinot Rodríguez

Clinical Head of the Brachytherapy Unit, Radiation Oncology Service, Valencian Institute of Oncology Foundation (IVO), Valencia, Spain

Registration details:

	Early Bird	Regular	Late
	Before 31st April	1st May - 30th June	1st July - 15th August
Resident	₹ 2000	₹ 3500	₹ 4500
Indian - IBS member	₹ 3500	₹ 5000	₹ 6000
Indian - IBS non member	₹ 4500	₹ 6000	₹ 7000
Vendor	₹ 5500	₹ 7000	₹ 8000
International	$ 100	$ 125	$ 150

This brochure is subject to minor changes. Please visit the website for regular updates

Spot registration :

- Registrar : ₹ 6500
- Indian : ₹ 8000
- Vendor : ₹ 9000
- International : $ 200

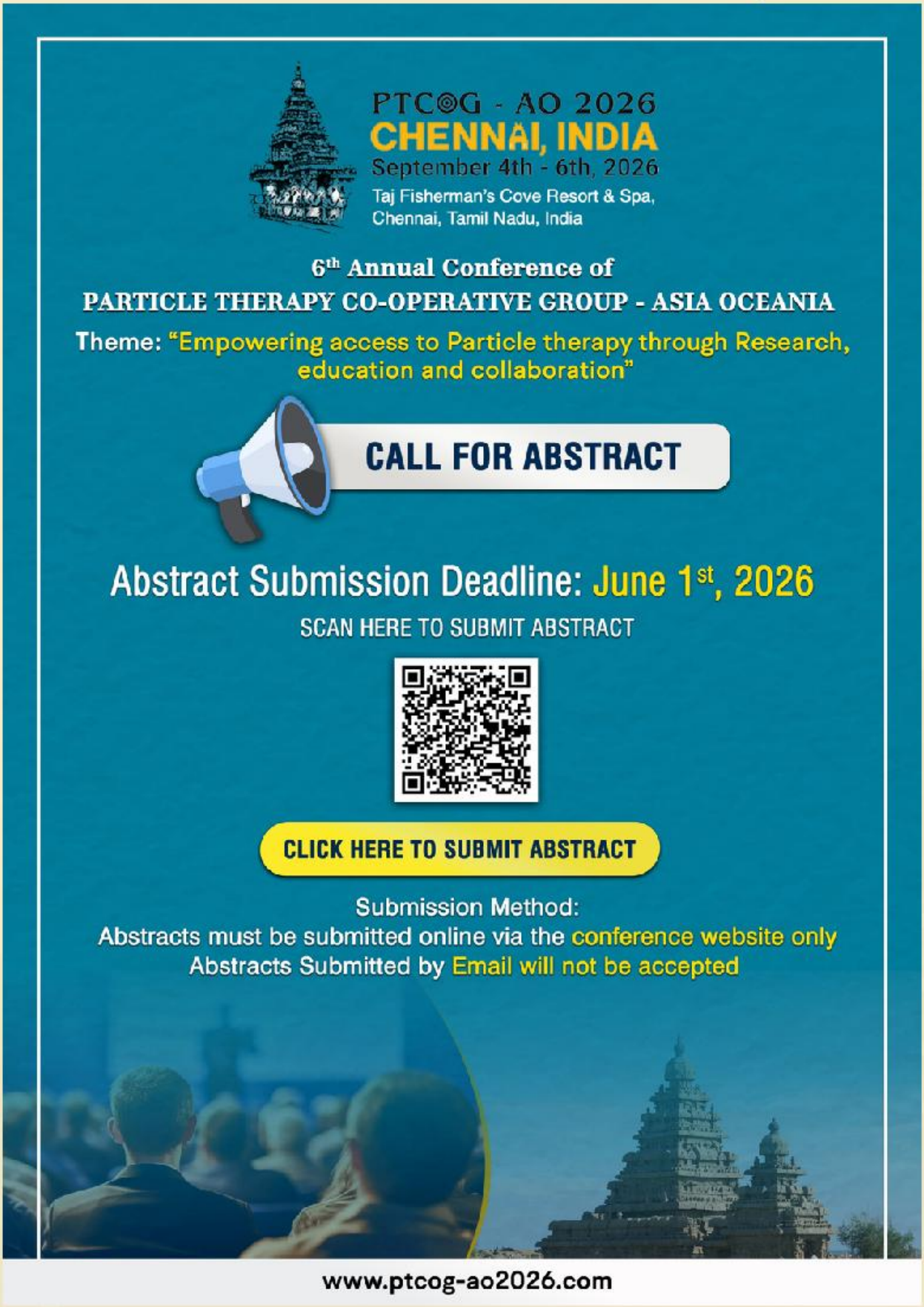
Workshop fee : ₹ 2000

Registration link:

https://www.cognitoforms.com/DistanceEducationCMCVellore/IndianBrachytherapySocietyConference2026?qr

ESTRO MEETS ASIA 2026

28 - 30 August 2026


PTCOG - AO 2026
CHENNAI, INDIA
September 4th - 6th, 2026
Taj Fisherman's Cove Resort & Spa,
Chennai, Tamil Nadu, India
6th Annual Conference of
PARTICLE THERAPY CO-OPERATIVE GROUP - ASIA OCEANIA
Theme: "Empowering access to Particle therapy through Research, education and collaboration"
CALL FOR ABSTRACT
Abstract Submission Deadline: June 1st, 2026
SCAN HERE TO SUBMIT ABSTRACT
CLICK HERE TO SUBMIT ABSTRACT
Submission Method:
Abstracts must be submitted online via the conference website only
Abstracts Submitted by Email will not be accepted
www.ptcog-ao2026.com

37TH UPAROICON 2026

31st October and 01st November 2026

ONCOLOGIC PANEL 2026

"of the residents, by the residents, for the residents."

Dr Piyush Kumar, SRMS Bareilly

The Onco Logic Panels 2026, held on 22nd February 2026 at SRMS Institute of Medical Sciences (SRMSIMS), Bareilly, was an innovative oncology conference themed "of the residents, by the residents, for the residents." The event adopted an interactive panel-based format , positioning residents as central participants in academic discussions.
The program began with structured panel discussions covering contemporary oncologic issues. Initial sessions focused on molecular markers in brain tumors, emphasizing their diagnostic and prognostic value, followed by debates on neoadjuvant strategies in locally advanced head and neck cancers and adjuvant radiotherapy in early oral cancers, highlighting the importance of risk-adapted treatment decisions. A later session on axillary management in breast cancer discussed treatment de-escalation and the evolving role of sentinel lymph node biopsy and radiotherapy.

A key highlight was the SRMS Oration by Dr. N. R. Datta, who discussed advances in radiation oncology, stressing innovation, precision medicine, and collaboration. The event also featured the launch of a PlayWorkbook for Radiation Oncology residents, designed to enhance learning through interactive educational tools such as puzzles and crosswords.

Baidyanath Dham

Baba Baidyanath Temple (Baidyanath Dham) in Deoghar, Jharkhand, is one of the 12 sacred Jyotirlingas in India and a revered Shakti Peetha, where Sati's heart is believed to have fallen

ONCOLOGIC PANEL 2026

"of the residents, by the residents, for the residents."

Afternoon sessions addressed dose escalation in esophageal cancer , molecular classification in endometrial cancer, total neoadjuvant therapy in rectal cancer, and management strategies in metastatic disease, emphasizing evidence-based, patient-centered care. Panels were rigorously evaluated on scientific content, clarity, and reasoning, with faculty feedback enhancing learning outcomes.

In conclusion, Onco Logic Panels 2026 successfully demonstrated the effectiveness of a resident-driven academic model, promoting critical thinking, peer learning, and evidence-based discussion. The structured evaluation system and strong faculty mentorship made the conference a valuable model for future oncology educational programs.

NINTH TEACHING COURSE IN RADIOBIOLOGY AND MEDICAL PHYSICS

Sri Aurobindo Institute of Medical Science, Indore

The Ninth Teaching Course in Radiobiology and Medical Physics was successfully conducted on 4th and 5th April 2026 at Sri Aurobindo Medical College, Indore. The course saw active participation from 75 students representing diverse regions of the country, including Jammu & Kashmir, New Delhi, Rajasthan, Karnataka, Maharashtra, Gujarat, Madhya Pradesh, and Chhattisgarh.

The academic program was thoughtfully designed to cover all essential aspects of Radiobiology and Medical Physics through a series of comprehensive and well-structured lectures. Dr. Virendra Bhandari delivered engaging sessions on Radiobiology and Beam Modifying Devices, providing valuable insights into both fundamental concepts and clinical applications.

The Medical Physics component was led by an experienced and dedicated team comprising Dr. Deepika Malik, Dr. Ashar Lodi, Dr. Prityusha Bagdare, Mr. Mahendran, Mr. Devendra Chawala, and Ms. Gomathi R., who delivered lectures across a wide spectrum of topics with clarity and precision.

The course was highly appreciated by the participants, who found the lectures informative, interactive, and easy to understand. The quality of teaching and the practical orientation of the sessions were particularly well received.

Overall, the program proved to be an enriching academic experience. It is expected that the knowledge and skills gained during this course will greatly benefit the participants in their future academic and professional endeavors.

Dr Virendra Bhandari,
Professor & Head , Radiation Oncology

NINTH TEACHING COURSE IN RADIOBIOLOGY AND MEDICAL PHYSICS

Sri Aurobindo Institute of Medical Science, Indore

WORLD CANCER DAY OBSERVED AT JNMC, AMU, ALIGARH

Awareness Drive, Academic Engagement & Survivor Interaction

The Department of Radiation Oncology, JNMC, AMU, Aligarh, marked World Cancer Day 2026 with a series of impactful academic and awareness activities conducted over 27–28 January 2026, under the theme "United by Unique."The programme was organized under the aegis of the UPAROI Society, reflecting a collaborative commitment toward cancer awareness and education. The two-day event witnessed enthusiastic participation from doctors, scholars, and students across diverse disciplines. A wide range of creative and academic competitions were organized to engage participants and promote cancer awareness among young minds. These included:

- Quiz Competition
- Scientific E-Poster Competition
- Slogan Writing
- Poster Making
- Image Reimagination
- Storytelling
- Reel Making
- Cancer Ribbon Reimagined

Approximately 130 participants took part in these activities, highlighting strong academic engagement and growing interest in cancer awareness initiatives among students and healthcare trainees.

On 5 February 2026, the department conducted a special programme featuring a Cancer Survivor Interaction Session, which emerged as one of the most meaningful highlights of the celebration. The session provided participants with an opportunity to listen to and interact with cancer survivors, allowing them to understand firsthand the journeys of resilience, courage, and recovery. The programme concluded with a valedictory ceremony, during which winners of the competitions were felicitated and acknowledged for their contributions.

An important milestone was also announced during the event — the signing of a Memorandum of Understanding (MoU) by Aligarh Muslim University (AMU) under the Public–Private Partnership (PPP) model. This MoU will facilitate the establishment of a state-of-the-art Linear Accelerator (LINAC) Radiotherapy facility and Nuclear Imaging setup within the Department of Radiation Oncology, JNMC, AMU. This development represents a significant step toward strengthening advanced cancer care infrastructure and improving patient access to modern oncological services.

WORLD CANCER DAY OBSERVED AT JNMC, AMU, ALIGARH

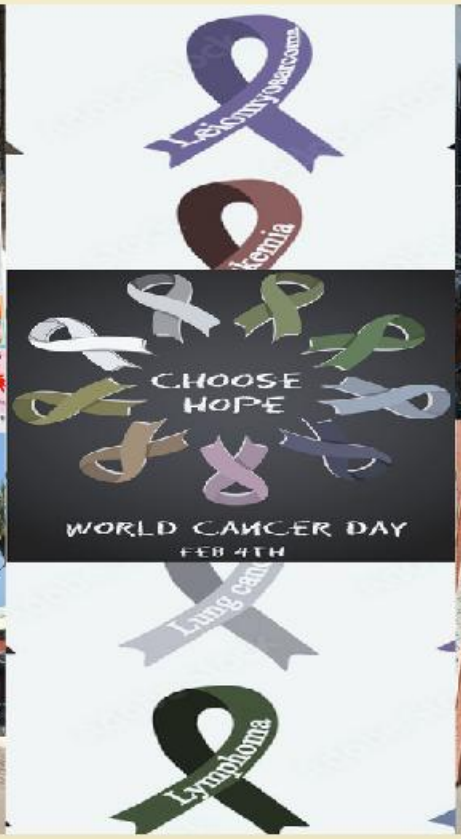
The Department expressed its commitment to continuing such educational and community-oriented initiatives in the future, with the aim of fostering awareness, encouraging academic participation and strengthening patient-centered cancer care.

Reported by:

Dr Md Shadab Alam
Secretary, UPAROI Society
Department of Radiation Oncology
JNMC, AMU, Aligarh

Rajrappa Mandir
The 6000 years old Rajrappa Mandir also known as the' Chhinnmastika Temple is one of the shaktipeeths amongst the 51 in India.


WORLD CANCER DAY 2026
"United By Unique"
DEPARTMENT OF RADIATION ONCOLOGY
EVENT DETAILS
Scan / Click
Open to All UG/PG/Residents/PhD
Participation Certificates for All
Faculty of Medicine, JNMC is organizing a
MEGA EVENT
(Under the Aegis of UPAROI Society)
REGISTRATION DETAILS
ENTRY FEE:
₹ 200/- Only
Last Date:
24 January 2026
REGISTER HERE
Scan or Click
JAN 27 2026
Story Writing
Reel Making
Poster Design
Slogan Writing
Image Reimagination
JAN 28 2026
CANCER QUIZ
Cancer Quiz Competition
Scientific Poster Presentation
FEB 05 2026
Pop-up Quiz
Valedictory Function
Interaction with cancer survivors and skit
Lunch
CANCER AWARENESS COMPETITIONS
① STORY WRITING
② REEL
③ POSTER
④ SLOGAN
⑤ IMAGE REIMAGINATION
@ Exam Hall, JNMC
Time: 10:00 AM onwards
Medical
CANCER QUIZ COMPETITIONS
Non-Medical
SCIENTIFIC POSTER PRESENTATION
@ LT - 1, JNMC
Time: 10:00 AM onwards
CANCER DAY EVENTS
Followed by Valedictory Function
Skit
Interaction with Cancer Survivors
POP-UP QUIZ
All Winners will be Felicitated with a Memento
@ JNMC Auditorium
Time: 10:00 AM onwards


CHOOSE HOPE
WORLD CANCER DAY
FEB 4TH
Lung cancer
Lymphoma


WORLD CANCER DAY 2026

EVENTS

JAN 27th 2026

- ✓ Slogan Writing
- ✓ Poster
- ✓ Image Reimagination
- ✓ Story Telling
- ✓ Reel
- ✓ Cancer Ribbon Reimagined

JAN 28th 2026

- ✓ Quiz Competition
- ✓ Scientific E-Poster

Quiz Competition



Scientific E-Poster Competition





Slogan Writing



Poster




Image Reimagination



Story Telling


WORLD CANCER DAY, 2026
RESERVED
RESERVED
RESERVED

Sunset of Patratu valley

Patratu Valley, located about 40 km from Ranchi in Jharkhand's Ramgarh district, is a popular scenic destination known for its dramatic hairpin bends, lush greenery, and the massive Patratu Dam