



Molecular Classification of CNS Tumours: A Radiation Oncologist's Perspective

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Abstract

Molecular classification has transformed central nervous system (CNS) tumour diagnosis by integrating genetic and epigenetic information with histopathology and imaging. The 2021 WHO classification made molecular alterations defining criteria for several tumour entities, creating direct and emerging implications for radiation oncology. This review examines how molecular information affects diagnostic grading, risk stratification, systemic-therapy selection, radiation de-escalation, and target-volume delineation. CDKN2A/B homozygous deletion can molecularly upgrade IDH-mutant astrocytoma to WHO grade 4, while IDH mutation with 1p/19q codeletion defines oligodendroglioma. MGMT promoter methylation is primarily a predictive biomarker for alkylating chemotherapy benefit rather than an independent marker of radiosensitivity. Molecular subgroups of medulloblastoma, meningioma, and ependymoma refine prognostic assessment and may support future treatment adaptation. In contrast, current CNS radiotherapy remains predominantly anatomy-, imaging-, surgical-, and clinically driven. Molecularly guided dose reduction, margin modification, and adaptive radiotherapy remain areas for prospective validation. Precision CNS radiotherapy will therefore depend on integrating molecular biology with imaging, surgical findings, patterns of spread, patient factors, and prospective evidence.

Keywords: CNS tumours; molecular classification; radiotherapy; IDH; 1p/19q; MGMT; medulloblastoma; meningioma; ependymoma; precision radiotherapy

1. Introduction

The classification of CNS tumours has moved from a predominantly morphology-based system toward an integrated taxonomy in which histology, molecular alterations, DNA methylation, anatomical location, and clinical context are considered together. The 2021 WHO Classification of Tumours of the Central Nervous System (WHO CNS5) formalised this transition by incorporating molecular alterations into the definition of several tumour entities (Louis et al., 2021). For the radiation oncologist, this change is not simply semantic: molecular information may alter diagnosis, expected natural history, systemic-therapy options, treatment intensity, and the degree to which treatment can be individualised.

The practical question is how much molecular information should change radiotherapy. In some settings, molecular findings already have a clear treatment consequence. Examples include molecular upgrading of IDH-mutant astrocytoma by CDKN2A/B homozygous deletion and identification of oligodendroglioma by combined IDH mutation and 1p/19q codeletion. In other settings, molecular information is primarily prognostic or predictive for systemic therapy. MGMT promoter methylation in glioblastoma is an important example: it predicts benefit from alkylating chemotherapy but should not be interpreted as a stand-alone measure of radiosensitivity. In medulloblastoma, molecular subgrouping has opened the possibility of risk-adapted craniospinal irradiation (CSI), while in meningioma and ependymoma molecular classification is improving prognostic stratification but has not yet replaced conventional determinants of postoperative radiotherapy.

This review focuses on the radiation-oncology consequences of CNS molecular classification: what molecular information changes today, what may change in the near future, and where evidence remains insufficient for routine alteration of radiation dose, target volumes, or treatment technique.

2. From Histology to Integrated Molecular Diagnosis

WHO CNS5 introduced a biologically anchored classification in which certain molecular alterations are defining rather than merely descriptive. This distinction is crucial for radiation oncology because the same morphological appearance can represent biologically different diseases with different clinical trajectories and treatment strategies. Conversely, tumours that appear histologically less aggressive may be assigned a higher-grade diagnosis when a defining molecular alteration is present (Louis et al., 2021; Horbinski et al., 2022).

For diffuse gliomas, IDH mutation has become a major organising principle. Adult-type diffuse gliomas are now separated into three principal entities: astrocytoma, IDH-mutant; oligodendroglioma, IDH-mutant and 1p/19q-codeleted; and glioblastoma, IDH-wildtype. This classification removes several historical categories whose biological heterogeneity previously complicated treatment interpretation. Molecular classification therefore improves the reproducibility of clinical trials and makes treatment evidence more applicable to biologically coherent patient groups.

From a radiotherapy perspective, the integrated diagnosis should be available before final treatment decisions whenever feasible. Molecular results should be interpreted alongside age, performance status, extent of resection, postoperative MRI, residual enhancement, T2/FLAIR abnormality, dissemination, and anticipated patterns of failure. Molecular information is consequently an additional layer of treatment information rather than a replacement for anatomy or clinical judgement.

3. Molecular Upgrading and Its Radiation-Oncology Consequences

One of the clearest examples of molecular information directly changing tumour grade is IDH-mutant astrocytoma with CDKN2A/B homozygous deletion. In WHO CNS5, homozygous deletion of CDKN2A and/or CDKN2B is sufficient to classify an IDH-mutant astrocytoma as WHO grade 4, even in the absence of classical histological features of necrosis or microvascular proliferation (Louis et al., 2021). This prevents an apparently lower-grade tumour from being managed solely according to morphology when its molecular profile indicates more aggressive biology.

The radiation implication is primarily a change in treatment pathway rather than a molecularly prescribed radiation dose. A patient previously considered for prolonged observation or lower-intensity treatment may instead require management appropriate to a grade-4 diffuse astrocytic tumour. Molecular upgrading can therefore prevent undertreatment. However, the molecular label does not by itself establish the optimal dose, fractionation, target margin, or treatment technique; those decisions remain dependent on the total clinical and anatomical picture.

In the appropriate clinical and anatomical setting, TERT promoter mutation, EGFR amplification, or combined whole-chromosome +7/-10 can support a molecular diagnosis of glioblastoma in an IDH-wildtype diffuse astrocytic tumour even when classic histological features are absent (Brat et al., 2018; Weller et al., 2021). This illustrates the broader principle of molecular upgrading: classification may change before the gross radiological phenotype becomes obviously different.

For radiation oncologists, this creates a practical workflow requirement. Molecular pathology should ideally be available before treatment intent and postoperative RT planning are finalised. When pathology and molecular results are discordant, multidisciplinary review with neuropathology, neuroradiology, neurosurgery,

medical oncology, and radiation oncology is preferable to making a treatment decision from an isolated biomarker.

4. Molecular Risk Stratification and Radiation De-escalation

Medulloblastoma: molecular subgrouping has substantially improved risk stratification. Major groups include WNT-activated, SHH-activated, and non-WNT/non-SHH disease, with further molecular subdivisions (Northcott et al., 2011). WNT-activated tumours in appropriately defined favourable-risk populations generally have excellent outcomes and have therefore been a major focus of treatment de-escalation.

Prospective molecularly stratified studies such as SIOP PNET5-MB have evaluated reduced-dose CSI in selected standard-risk WNT-activated medulloblastoma (Rutkowski et al., 2021). The radiation-oncology significance is important: molecular biology can identify a group in whom reducing CSI dose may be clinically meaningful because long-term neurocognitive, endocrine, vascular, and other treatment-related toxicities matter greatly in patients with long expected survival. However, favourable molecular biology should not be equated with permission to omit CSI outside appropriately designed clinical trials. Molecular classification currently supports selective de-escalation research rather than universal radiation omission.

Oligodendroglioma: the combination of an IDH mutation and whole-arm 1p/19q codeletion defines the tumour entity (Louis et al., 2021). This molecular definition identifies a biologically coherent tumour group with a relatively favourable natural history and substantial sensitivity to alkylating chemotherapy. Treatment implications are therefore closely linked to molecular diagnosis rather than historical oligodendroglial morphology alone.

In selected patients with grade 2 oligodendroglioma who have undergone gross total resection and have no high-risk clinical features, observation can be appropriate. For higher-risk grade 2 disease, adjuvant RT with

systemic therapy may be considered, while grade 3 disease generally requires combined-modality treatment (Cairncross et al., 2013; van den Bent et al., 2013). Contemporary ASTRO guidance similarly incorporates molecular diagnosis, age, extent of resection, and high-risk features into RT selection (ASTRO, 2022). Thus, molecular classification enables risk-adapted use of RT rather than making RT mandatory for every molecularly defined tumour.

5. MGMT Promoter Methylation: Predictive Rather Than Simply “Radiosensitive”

MGMT promoter methylation is one of the most clinically established biomarkers in glioblastoma. Epigenetic silencing of MGMT reduces repair of DNA damage caused by alkylating agents and is associated with greater benefit from temozolomide (TMZ) (Hegi et al., 2005). The important distinction for radiation oncology is that MGMT promoter methylation is principally a predictive biomarker for alkylating chemotherapy benefit, not a validated independent biomarker that should determine radiation dose.

This distinction is clinically relevant when treating older or frail patients. The choice between combined chemoradiation, abbreviated RT, and chemotherapy-oriented strategies may incorporate MGMT status together with age, performance status, comorbidity, tumour burden, and patient preference (Malmström et al., 2012; Wick et al., 2012). In a patient with a methylated tumour, anticipated benefit from TMZ may support its inclusion when otherwise appropriate. Conversely, absence of methylation may reduce the expected incremental benefit of TMZ, particularly when treatment tolerance is a major concern.

The terminology used in clinical discussion should therefore be precise. “MGMT-methylated” should not automatically be translated as “radiosensitive,” and “MGMT-unmethylated” should not be interpreted as proof of radioresistance. The clinical evidence primarily concerns interaction with alkylating

chemotherapy. Whether MGMT status reliably identifies differential RT benefit remains a separate question requiring appropriate comparative evidence.

6. Molecular Classification and Target-Volume Delineation

Among the most important questions for radiation oncologists is whether molecular classification should change target volumes. At present, the answer is cautious. CNS radiotherapy remains predominantly based on the surgical cavity, residual tumour, MRI-defined disease, expected routes of microscopic spread, dissemination pattern, and established patterns of failure. Molecular information may refine risk but does not yet provide a prospectively validated method for replacing conventional anatomical target definition.

For IDH-mutant grade 2 and 3 diffuse glioma, contemporary ESTRO-EANO guidance recommends MRI-informed delineation, including the resection cavity and residual tumour. T2/FLAIR abnormalities considered tumour are incorporated into the GTV, with differentiation from oedema particularly relevant in grade 3 disease. The guideline recommends a 10-mm GTV-to-CTV expansion for grade 2 disease and 15 mm for grade 3 disease, with appropriate editing to anatomical barriers (Baumert et al., 2025). These recommendations apply across IDH-mutant diffuse glioma categories and do not prescribe different margins solely according to 1p/19q status.

The implication is that molecular classification has so far influenced target delineation indirectly. It identifies the biological entity and clinical risk context, but it does not automatically define where microscopic disease is located. A molecularly favourable tumour can still recur locally if target volumes are inadequate, while a molecularly aggressive tumour does not necessarily require an arbitrarily enlarged CTV.

Future target delineation may become more biologically informed. Radiomics, amino-acid PET, perfusion and diffusion MRI, spatial

genomics, DNA methylation, and circulating tumour DNA could potentially identify regions with different probabilities of microscopic involvement or recurrence. However, a biologically reduced margin should not be adopted simply because a molecular subgroup has a favourable prognosis. Margin reduction requires prospective evidence that local control is not compromised.

7. Meningioma: Molecular Risk Beyond Histological Grade

Meningioma illustrates a different aspect of molecular classification. Histological WHO grade remains clinically important, but molecular alterations and DNA methylation-based classification can provide additional information regarding recurrence risk and biological behaviour (Sahm et al., 2017; Nassiri et al., 2021). TERT promoter mutation and CDKN2A/B homozygous deletion are particularly relevant because they can support classification as WHO grade 3 in the appropriate setting.

BAP1 loss is associated with an aggressive meningioma phenotype, particularly in rhabdoid tumours. NF2 alterations are common and biologically informative, but an NF2 alteration alone is not an indication for postoperative RT. This illustrates the difference between a biomarker that describes tumour biology and a biomarker that has demonstrated predictive value for a particular treatment.

DNA methylation-based classification is potentially important because it may refine recurrence-risk assessment beyond conventional histological grading. Its eventual value for radiation oncology would be greatest if it could prospectively identify patients whose risk is sufficiently high to justify adjuvant RT after otherwise borderline indications, or conversely identify patients who can safely avoid RT. At present, this remains an evolving area, and conventional factors such as WHO grade, extent of resection, tumour location, recurrence, and neurological risk remain central to postoperative decision-making.

8. Ependymoma: Molecular Subgroups and Postoperative Radiotherapy

Ependymoma has undergone major molecular reclassification. Major entities include posterior fossa PFA and PFB tumours and supratentorial ZFTA fusion-positive and YAP1 fusion-positive tumours, with additional molecularly defined spinal entities (Pajtler et al., 2017; Louis et al., 2021). This classification is clinically meaningful because molecular groups demonstrate different biological behaviour and patterns of outcome.

PFA tumours generally behave more aggressively than PFB tumours, while YAP1 fusion-positive supratentorial tumours have been associated with more favourable outcomes. ZFTA fusion-positive tumours represent an important supratentorial group. These distinctions may eventually allow postoperative RT to be intensified or de-escalated according to biological risk, but current practice remains strongly influenced by extent of resection, residual disease, dissemination, tumour location, age, and conventional clinical risk.

For the radiation oncologist, molecular subgrouping should therefore be incorporated into multidisciplinary risk assessment but should not be used in isolation to determine postoperative RT. The key unanswered question is whether molecular subgroup-specific radiation strategies improve the therapeutic ratio compared with current risk-adapted treatment. Prospective collaborative trials are particularly important because individual molecular subgroups may be too uncommon for single-institution studies to provide definitive evidence.

9. Molecular Information in the Radiation-Oncology Workflow

The clinical value of molecular classification depends not only on the availability of testing but also on when and how the result enters the treatment pathway. A practical workflow begins with adequate tissue acquisition, integrated histological diagnosis, review of molecular results in the multidisciplinary setting, and correlation

with postoperative imaging and surgical findings before finalising the radiation plan.

For diffuse gliomas, the clinically useful dataset may include IDH status, 1p/19q status where relevant, CDKN2A/B status in IDH-mutant astrocytoma, and MGMT promoter methylation in glioblastoma. Depending on tumour type, additional testing may include TERT promoter, EGFR, chromosome 7/10 status, BRAF, H3 alterations, methylation profiling, or fusion testing. The exact panel should remain tumour-specific rather than indiscriminately broad.

The radiation oncologist should ask four practical questions when reviewing a molecular report: (1) Does the result change the diagnosis or WHO grade? (2) Does it change expected prognosis or recurrence pattern? (3) Does it predict benefit from systemic therapy? and (4) Is there prospective evidence that it should change radiation dose, target volume, timing, or technique? The fourth question is particularly important because molecular plausibility should not be mistaken for clinical validation.

A useful conceptual model is therefore: tissue acquisition → integrated diagnosis → molecular risk classification → correlation with MRI and surgical findings → multidisciplinary treatment selection → RT planning → longitudinal reassessment. This prevents molecular results from being interpreted in isolation and preserves the central role of anatomy and clinical context in radiotherapy.

10. Pitfalls and Challenges

Molecular precision does not automatically mean clinical precision. Tumour heterogeneity can result in sampling error, particularly when a small biopsy is used to represent a spatially heterogeneous tumour. Recurrent tumours may also acquire additional alterations, making the molecular profile at recurrence different from that at initial diagnosis. Timing and adequacy of tissue sampling should therefore be considered when interpreting results.

Assay-related variability is another challenge. Different laboratories, platforms, cut-offs, and analytical methods may produce different results for some biomarkers, including MGMT promoter methylation. Clinical interpretation should take into account the assay used and the evidence supporting its methodology.

A third challenge is the distinction between prognostic and predictive biomarkers. A prognostic biomarker is associated with outcome regardless of treatment; a predictive biomarker indicates differential benefit from a particular intervention. A molecular alteration associated with poor survival does not necessarily imply radioresistance, and a favourable molecular subgroup does not automatically justify RT de-escalation. The evidence needed to support treatment adaptation is substantially stronger than evidence establishing prognostic association.

Finally, molecular taxonomy is evolving faster than prospective radiotherapy evidence. New entities can be defined before sufficient numbers of patients have accumulated to conduct randomized RT-specific trials. Radiation recommendations must therefore remain anchored to evidence, while molecular findings are incorporated in a transparent and appropriately cautious manner.

11. Future Directions: Toward Precision Radiotherapy

The next stage of CNS radiotherapy is likely to involve integration of molecular profiling with advanced imaging, radiomics, artificial intelligence, and longitudinal biomarkers. The objective should not simply be to intensify treatment for biologically aggressive disease. Equally important is identifying patients in whom radiation dose, volume, or treatment burden can safely be reduced without compromising tumour control.

One potential application is molecularly guided dose de-escalation. WNT-activated medulloblastoma provides a clinically relevant example in which molecularly defined favourable biology can be incorporated into prospective reduced-dose CSI strategies. Similar approaches may eventually emerge in other tumours if molecular subgroups demonstrate sufficiently low recurrence risk and prospective studies establish acceptable disease control.

A second area is biological target-volume optimisation. Current CTV margins are largely derived from anatomical patterns of microscopic spread and recurrence analyses. Future models could combine molecular subtype, spatial imaging, radiomics, functional imaging, and genomic information to generate patient-specific maps of recurrence probability. Such approaches could potentially distinguish regions requiring full-dose treatment from regions in which dose could be reduced, although this remains investigational.

A third opportunity is longitudinal adaptation. Serial MRI, advanced imaging, circulating tumour DNA, and other biomarkers may eventually identify treatment response or emerging resistant clones during therapy. Adaptive radiotherapy could then move beyond geometric adaptation to biologically informed adaptation. This will require robust biomarkers, reproducible assays, validated decision thresholds, and prospective demonstration of clinical benefit.

Artificial intelligence may provide the computational framework for integrating these multidimensional datasets. Automated

segmentation, radiomics, molecular prediction from imaging, outcome modelling, and adaptive planning are rapidly developing. Clinically useful systems will require external validation, interpretability, workflow integration, and prospective evaluation rather than simply high retrospective prediction accuracy.

12. Conclusion

Molecular classification has fundamentally changed CNS tumour diagnosis and is increasingly relevant to radiation oncology. CDKN2A/B homozygous deletion can molecularly upgrade IDH-mutant astrocytoma; IDH mutation with 1p/19q codeletion defines oligodendroglioma; MGMT promoter methylation predicts benefit from alkylating chemotherapy; and molecular subgroups of medulloblastoma, meningioma, and ependymoma refine biological risk.

The immediate radiation-oncology impact is greatest in diagnosis, treatment selection, systemic-therapy integration, and risk stratification. In contrast, molecularly determined radiation dose and target-volume modification remain less mature. Current radiotherapy should therefore continue to integrate molecular biology with MRI, surgical findings, patterns of spread, patient factors, and established clinical evidence. The future of CNS radiotherapy is not simply “molecular radiotherapy,” but integrated precision treatment in which molecular information adds biological context to accurate anatomical and clinical decision-making.

13. Summary of Key Molecular Markers in Radiation Oncology

Table 1. Clinically relevant molecular markers and their present or emerging implications for radiotherapy.

Tumour / setting	Key molecular feature	Clinical meaning	Radiotherapy relevance
Astrocytoma, IDH-mutant	CDKN2A/B homozygous deletion	Molecular WHO grade 4	Supports grade-4 treatment pathway; does not independently define dose
Glioblastoma, IDH-wildtype	TERT; EGFR; +7/-10 in appropriate setting	May establish molecular glioblastoma	Supports definitive chemoradiation pathway
Oligodendroglioma	IDH + 1p/19q codeletion	Defines tumour entity	Enables risk-adapted observation/RT and systemic therapy
Glioblastoma	MGMT promoter methylation	Predicts greater alkylator benefit	Helps contextualise TMZ; not a stand-alone RT marker
Medulloblastoma	WNT activation	Favourable biology in selected clinical setting	Reduced-dose CSI may be studied in selected patients/trials
Meningioma	TERT/CDKN2A/B; BAP1; methylation class	Refines grade/risk	May inform future adjuvant RT stratification
Ependymoma	PFA/PFB; ZFTA/YAP1	Distinct biological groups	Refines risk; subgroup-specific RT remains evolving

14. Conceptual Figure

Figure 1. Molecular classification to precision radiotherapy.

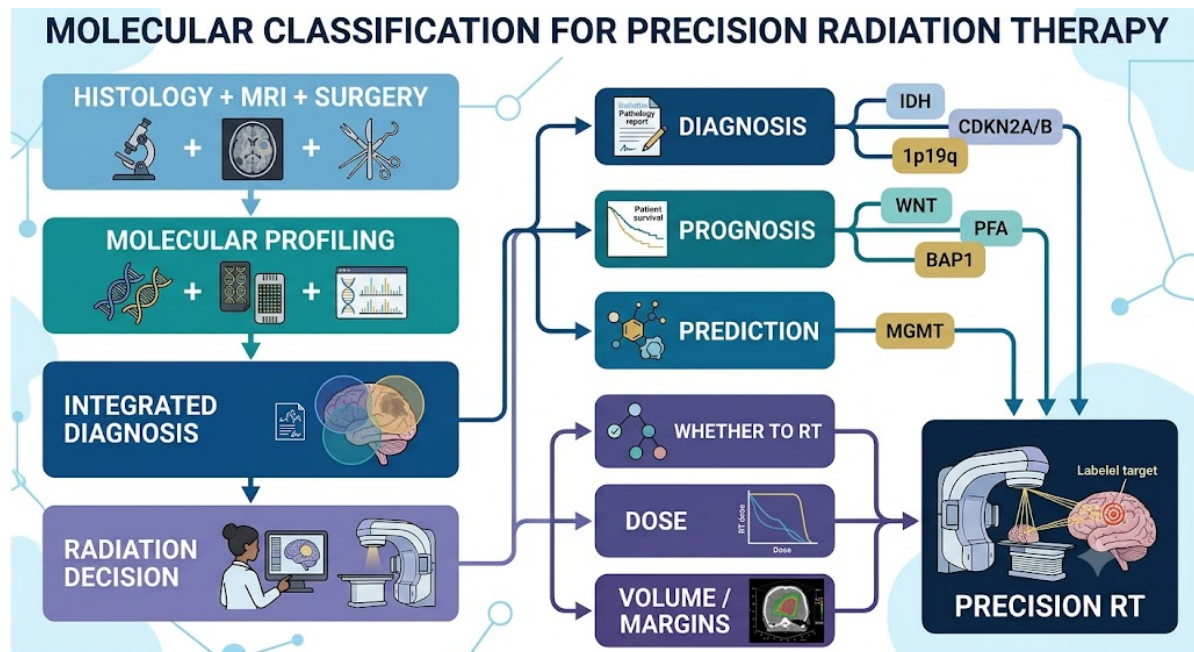


Figure 1: Molecular information is integrated with histology, imaging and surgical findings to refine diagnosis, risk and treatment selection. Most current molecular applications are diagnostic or prognostic; molecularly determined dose and margin adaptation remain investigational.

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