Review

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Review

Atypical Teratoid/Rhabdoid Tumor (ATRT): Historical Perspective, Pathology, Radiology, and Contemporary Clinical Advances

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Simple Summary

Atypical teratoid/rhabdoid tumor (ATRT) is a rare, aggressive brain tumor of infancy and early childhood. Once frequently mistaken for other tumors, ATRT is now defined by loss of the SMARCB1 (INI1) or SMARCA4 (BRG1) genes. In this review, we summarize what clinicians and researchers need to know today: typical imaging features (including marked diffusion restriction on MRI), key pathologic and molecular hallmarks, current multimodal treatment strategies, and practical considerations for radiotherapy in very young patients. We also highlight emerging directions—radiomics, subgroup-specific patterns, and novel targeted and immune therapies—that aim to improve survival while limiting late effects. Our goal is to provide a concise, clinically useful resource that connects imaging, biology, and treatment to support better care and future research for children with ATRT.

Abstract

Atypical teratoid/rhabdoid tumor (ATRT) is a rare, highly aggressive embryonal central nervous system (CNS) neoplasm of infancy and childhood. Since its recognition as a distinct entity in the late 20th century, breakthroughs in molecular pathology and neuroimaging have transformed diagnostic, prognostic, and therapeutic paradigms. This review synthesizes historical milestones, pathology and molecular biology, radiologic hallmarks, and advances of the past five years, with special emphasis on the WHO 2021 CNS tumor classification. We also highlight institutional experience at St. Louis Children's Hospital (SLCH), where systematic data collection and volumetric analyses underscore the challenges and opportunities in modern ATRT care.

Keywords: atypical teratoid/rhabdoid tumor; pediatric brain tumor; SMARCB1/INI1; diffusion MRI; radiomics; radiotherapy; proton therapy; pediatric neuro-oncology

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Introduction

Atypical teratoid/rhabdoid tumor (ATRT) is a rare but highly aggressive embryonal brain tumor that predominantly affects infants and very young children. ATRT accounts for only about 1–2% of all pediatric central nervous system (CNS) tumors, yet it constitutes a disproportionately high fraction of brain tumors in children under three years of age. Historically, many of these tumors were misdiagnosed as other small round blue cell tumors like medulloblastomas or primitive neuroectodermal tumors (PNETs). However, with advances in molecular biology, ATRT has been defined as a distinct entity characterized by the loss of the SMARCB1 (INI1) tumor suppressor gene (in the vast majority of cases) or, rarely, loss of SMARCA4 (BRG1). This genetic definition exemplifies the paradigm shift in neuro-oncology from purely morphology-based classification to an integrated molecular diagnosis, where identifying the underlying genetic lesion is essential for accurate classification. In practical terms, immunohistochemistry for loss of INI1 protein has become a key diagnostic marker distinguishing ATRT from other tumors.

Clinically, ATRT poses considerable diagnostic and therapeutic challenges due to its occurrence in very young patients and its aggressive behavior. Outcomes remain poorer than for most other pediatric brain tumors, and there is no single standardized treatment protocol given the rarity of the disease. Early diagnosis and aggressive multimodal therapy are critical to improving outcomes. Radiologically, ATRTs can be difficult to distinguish from other embryonal tumors on routine imaging. Standard MRI often cannot definitively differentiate ATRT from mimics like medulloblastoma, as both may appear as posterior fossa masses in young children. Nonetheless, evolving imaging techniques—such as advanced diffusion MRI, MR spectroscopy, and emerging radiomic analyses—are providing complementary clues that improve diagnostic confidence. For example, diffusion tensor imaging (DTI) metrics (particularly the apparent diffusion coefficient, ADC) have been shown to inversely correlate with tumor cellularity in pediatric brain tumors. In other words, highly cellular tumors tend to exhibit very low ADC values due to restricted diffusion of water in the densely packed tissue. This principle is extremely relevant in ATRT: ATRTs are often among the most cellular pediatric tumors, and they typically show markedly low ADC on MRI. Such diffusion characteristics, along with other MRI findings, can aid radiologists in distinguishing ATRT from other entities even when conventional imaging features overlap.

Materials and Methods

This review was designed as a narrative synthesis of the literature on atypical teratoid/rhabdoid tumor (ATRT), with a focus on historical context, pathology, molecular biology, neuroimaging, radiation therapy, and clinical outcomes. A comprehensive search of PubMed/MEDLINE, Embase, and Google Scholar was conducted through September 2025 using combinations of the following terms: “atypical teratoid rhabdoid tumor,” “ATRT,” “pediatric brain tumor,” “SMARCB1,” “SMARCA4,” “INI1,” “BRG1,” “diffusion MRI,” “radiomics,” “radiotherapy,” and “pediatric neuro-oncology.”

Peer-reviewed articles, clinical trials, institutional series, cooperative group reports, and consensus guidelines were prioritized. Case reports and preclinical studies were included selectively when they provided unique insights into imaging characteristics, subgroup biology, or translational therapeutics. References were cross-checked to ensure coverage of seminal publications and the most recent updates, including the WHO 2021 classification and cooperative-group trial results.

This article does not include unpublished patient data or new clinical analyses beyond summary descriptions of publicly available outcomes. Institutional experience at St. Louis Children’s Hospital is described qualitatively to provide context, but no new patient-level data were generated. The review follows MDPI’s guidance for narrative reviews, aiming to integrate historical developments, current state of knowledge, and emerging directions in the field.

Historical Perspective

ATRT’s history as a recognized tumor entity is relatively recent. The term “rhabdoid tumor” was first used in the late 1970s and early 1980s to describe an aggressive kidney tumor (malignant rhabdoid tumor of the kidney). Not long after, similar tumors were identified in the CNS of young children. Throughout the 1980s, many of these CNS cases were erroneously classified as medulloblastomas or PNETs due to overlapping histologic features and their common location in the posterior fossa. It wasn’t until the 1990s that pathologists recognized CNS “rhabdoid” tumors as a distinct clinicopathologic entity, and the term ATRT was introduced to denote these atypical teratoid/rhabdoid tumors. A breakthrough came around 1998–1999, when biallelic inactivation of the SMARCB1 gene (located on chromosome 22) was discovered as the defining molecular lesion in both renal and CNS rhabdoid tumors. This discovery revolutionized diagnosis: loss of the SMARCB1 gene product (INI1 protein) could be detected by immunohistochemistry, providing a quick and reliable diagnostic test for ATRT. By the early 2000s, INI1 immunohistochemistry became the gold standard for confirming ATRT, allowing clear separation of ATRTs from other pediatric brain tumors that retain INI1 expression. The importance of this molecular hallmark was formally acknowledged in classification schemes over time. Notably, the 2016 WHO classification of CNS tumors included ATRT as a distinct entity defined by SMARCB1 (or rarely SMARCA4) loss, and the most recent WHO 2021 update solidified this by requiring molecular confirmation (INI1 or BRG1 loss) for the diagnosis. The WHO 2021 classification also integrates DNA methylation profiling, recognizing that ATRT comprises three molecular subgroups (ATRT-TYR, ATRT-SHH, and ATRT-MYC) with distinct epigenetic signatures. This historical trajectory highlights how ATRT went from a misdiagnosed “variant” of other tumors to a well-defined diagnosis anchored in molecular genetics.

Developments in radiologic recognition of ATRT have paralleled the pathologic discoveries. In the 1980s and early 1990s, before ATRT was widely known as a separate entity, imaging descriptions of these tumors often reported them as “atypical” or variant medulloblastomas. By the late 1990s and early 2000s, as awareness grew, radiologists began noting characteristic imaging patterns: ATRTs were frequently larger and more heterogeneous than typical medulloblastomas, often containing areas of necrosis or hemorrhage, and they exhibited more pronounced diffusion restriction (extremely low ADC values) on MRI. In the 2010s, research efforts focused on systematically differentiating ATRTs from medulloblastomas and other embryonal tumors using advanced MRI parameters, including quantitative ADC, perfusion, and proton MR spectroscopy. In the past five years, radiology research in ATRT has incorporated radiogenomics and machine learning (radiomics) to classify tumors and explore subgroup-specific anatomic predilections (ATRT-TYR infratentorial; ATRT-MYC supratentorial/sellar; ATRT-SHH both compartments).

Pathology and Molecular Characteristics

Histologically, ATRT is defined by characteristic rhabdoid cells—large polygonal cells with eccentric nuclei, prominent nucleoli, and abundant eosinophilic cytoplasm that often contains paranuclear filamentous inclusions. Tumors frequently display mixed patterns, with epithelial-like, mesenchymal/spindle, and primitive neuroepithelial components, consistent with the “teratoid” designation. Immunohistochemistry typically demonstrates loss of INI1 (SMARCB1 deficiency) or, rarely, loss of BRG1 (SMARCA4 deficiency), with co-expression of epithelial (keratins/EMA), mesenchymal (vimentin), and neural/glial markers (GFAP, synaptophysin).

Molecularly, ATRT is a SWI/SNF (BAF) chromatin-remodeling complex–deficient cancer. Loss of SMARCB1/SMARCA4 disrupts chromatin regulation and creates dependencies on epigenetic regulators such as PRC2/EZH2. DNA methylation profiling delineates three reproducible molecular subgroups—ATRT-TYR, ATRT-SHH, and ATRT-MYC—with distinct age distributions, anatomic predilections, and emerging prognostic and therapeutic implications. The WHO 2021 CNS classification defines ATRT as a SMARCB1- or SMARCA4-deficient embryonal tumor, emphasizing integrated histology, immunohistochemistry, and molecular diagnostics to capture morphologic variants lacking classic rhabdoid features.

Radiological Features

Imaging is central to ATRT evaluation. On non-contrast CT, ATRTs are typically hyperdense and uncommonly calcified. MRI reveals large, heterogeneous masses with mixed T1/T2 signal intensities, irregular enhancement, frequent hemorrhage and necrosis, and an invasive growth pattern into adjacent brain structures or ventricles. A hallmark is marked diffusion restriction: ATRTs are strikingly hyperintense on DWI with very low ADC values, often lower than medulloblastoma, reflecting extreme hypercellularity. Perfusion-weighted MRI commonly shows heterogeneous, variably elevated rCBV, while proton MR spectroscopy typically demonstrates elevated choline, reduced NAA, and prominent lipid/lactate peaks—with a notable absence of taurine that helps distinguish ATRT from medulloblastoma. Given the high risk of CSF dissemination, craniospinal MRI and CSF cytology are essential at diagnosis and follow-up. **Figures 1-6** – To demonstrate the radiology, here we include images of an example case: a 12yo F presenting with strabismus and headache; figures include (1) an illustration of her brain tumor, (2-5) brain MR images at presentation (2-4) and after initial debulking surgery (5), and (6) body FDG PET/CT showing recurrent metastatic disease in the abdomen 16mo after initial presentation.

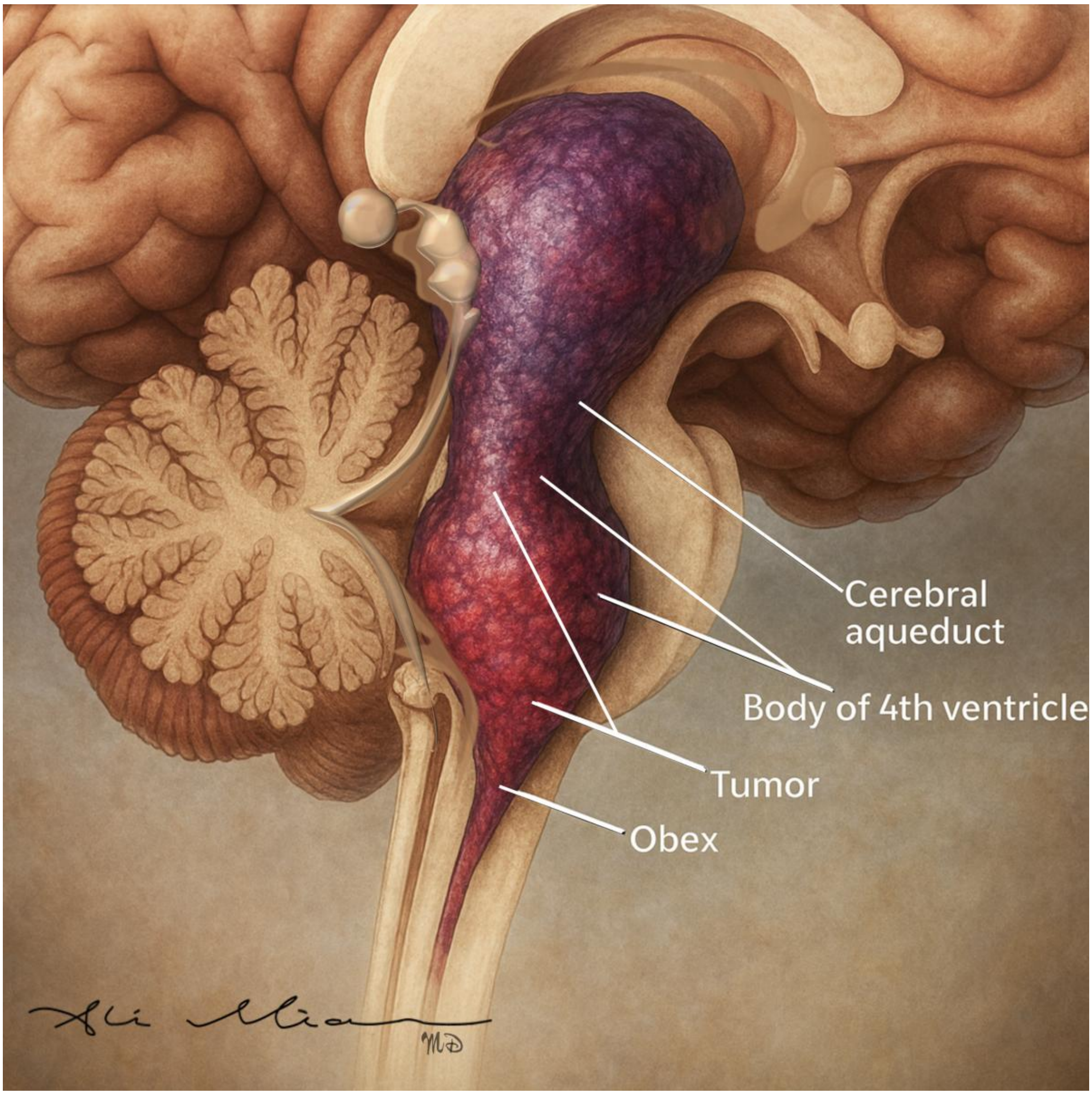

Figure 1. Schematic illustration of a brain mass centered in the 3rd and 4th ventricle. *Ali Mian, MD.*

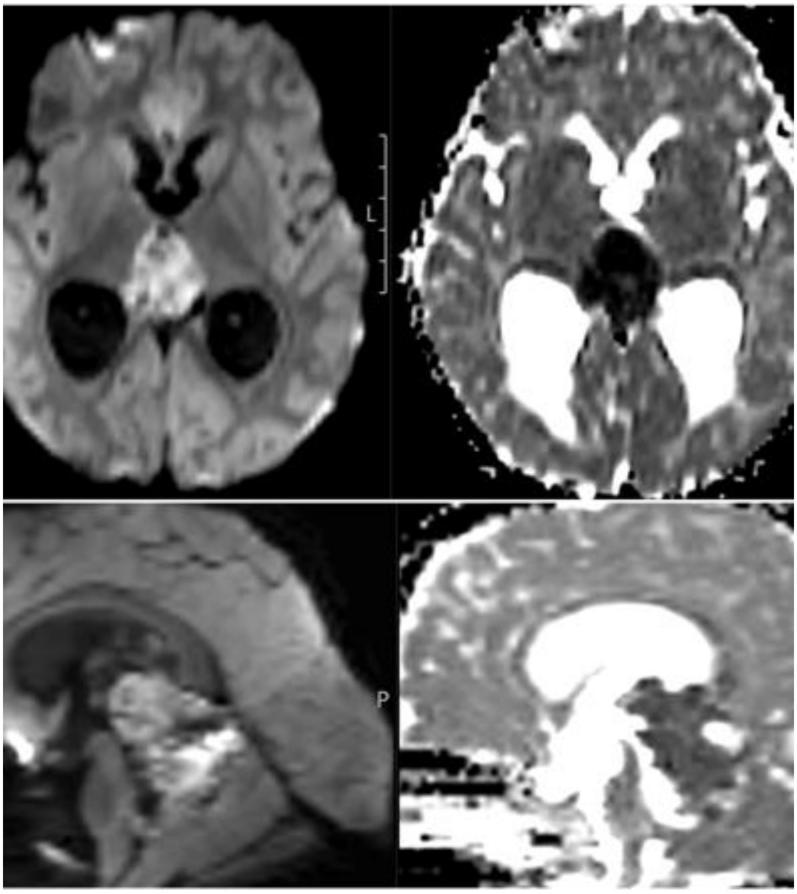

Figure 2. 12yo F Brain MR: Coronal T1 pre-and post contrast shows an enhancing mass centered in the 3rd and 4th ventricle with resultant supra-tentorial obstructive hydrocephalus.

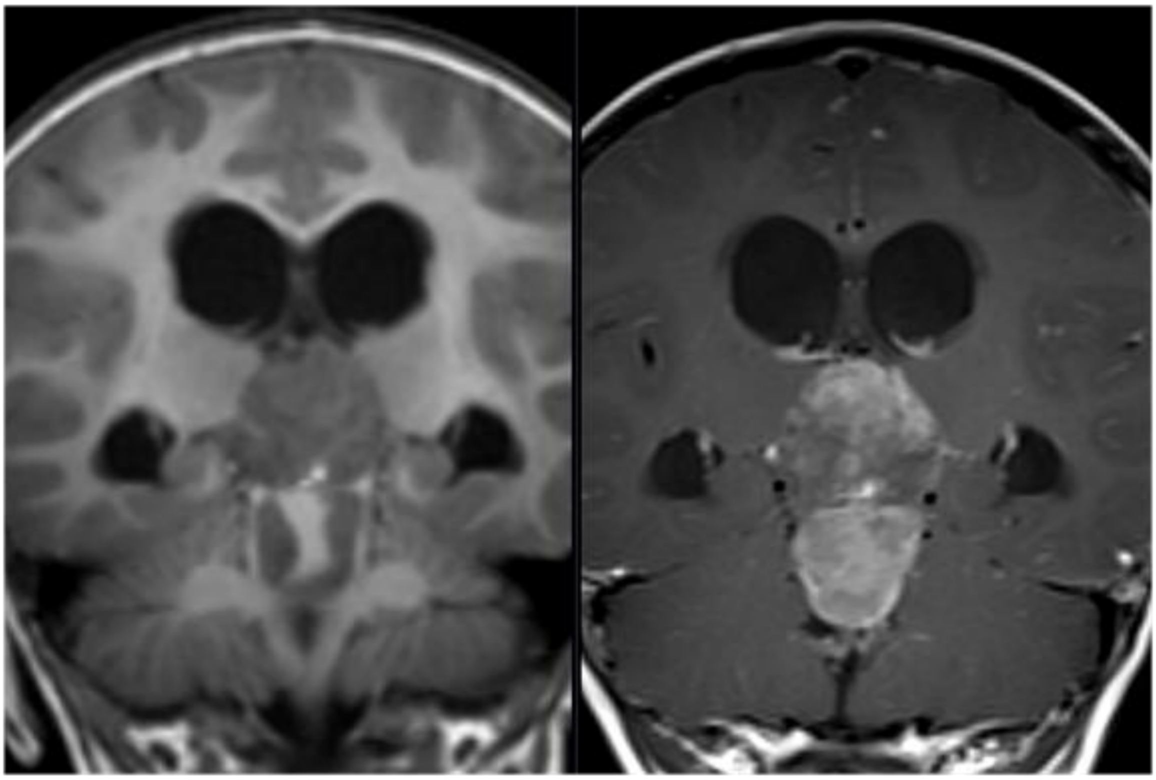

Figure 3. Axial and sagittal DWI with ADC maps show restricted diffusion within the lesion, indicating high cellularity of the tumor tissue.

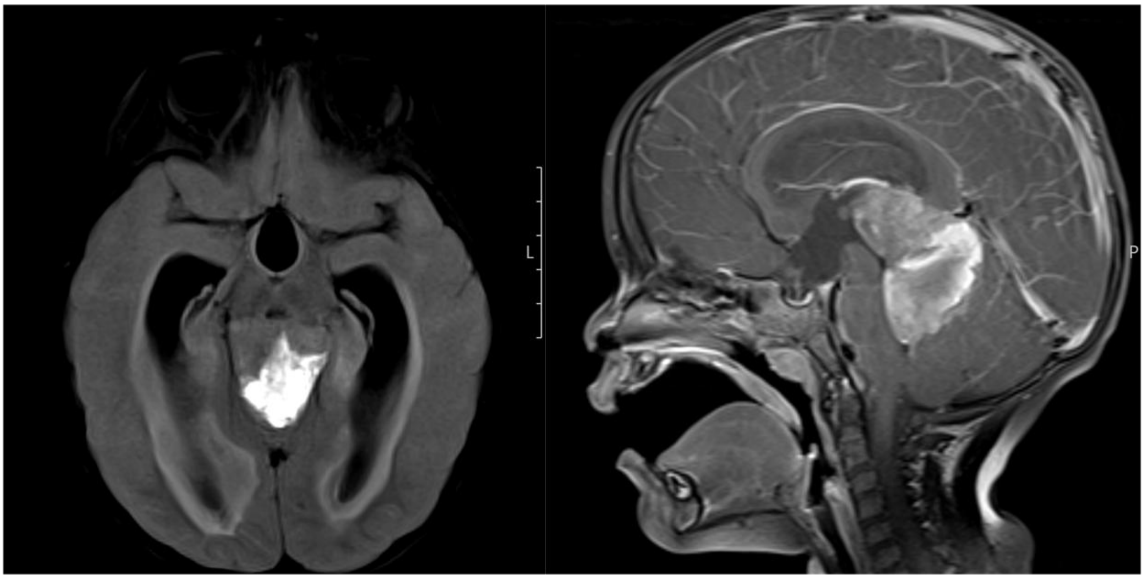

Figure 4. Axial T2 FLAIR and Sagittal post contrast T1 shows an enhancing mass centered in the 3rd and 4th ventricle with resultant supra-tentorial obstructive hydrocephalus.

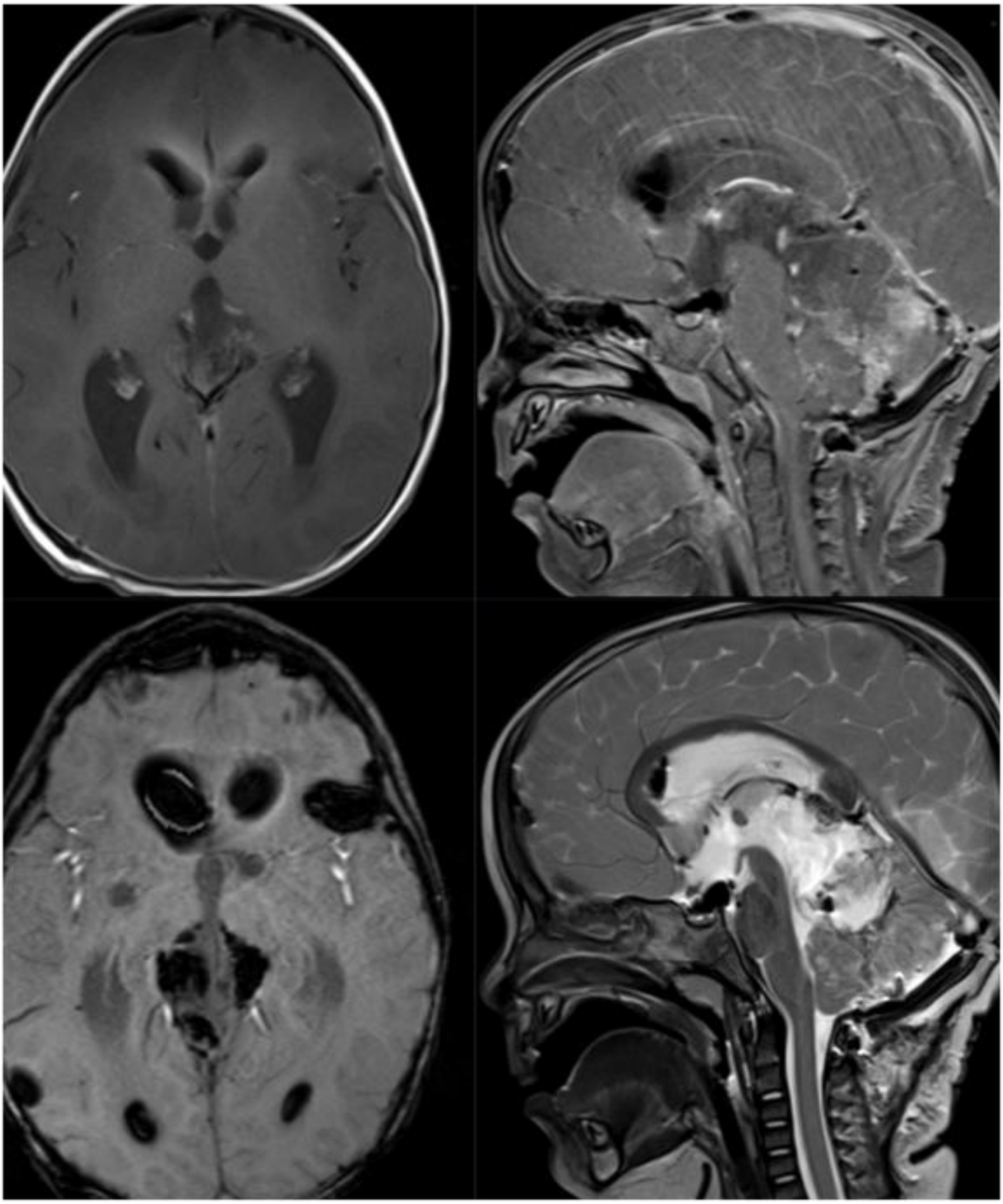

Figure 5. Axial SWI and Sagittal post contrast T1 shows interval resection of the mass previously occupying the third and fourth ventricle with resultant supra-tentorial obstructive hydrocephalus.

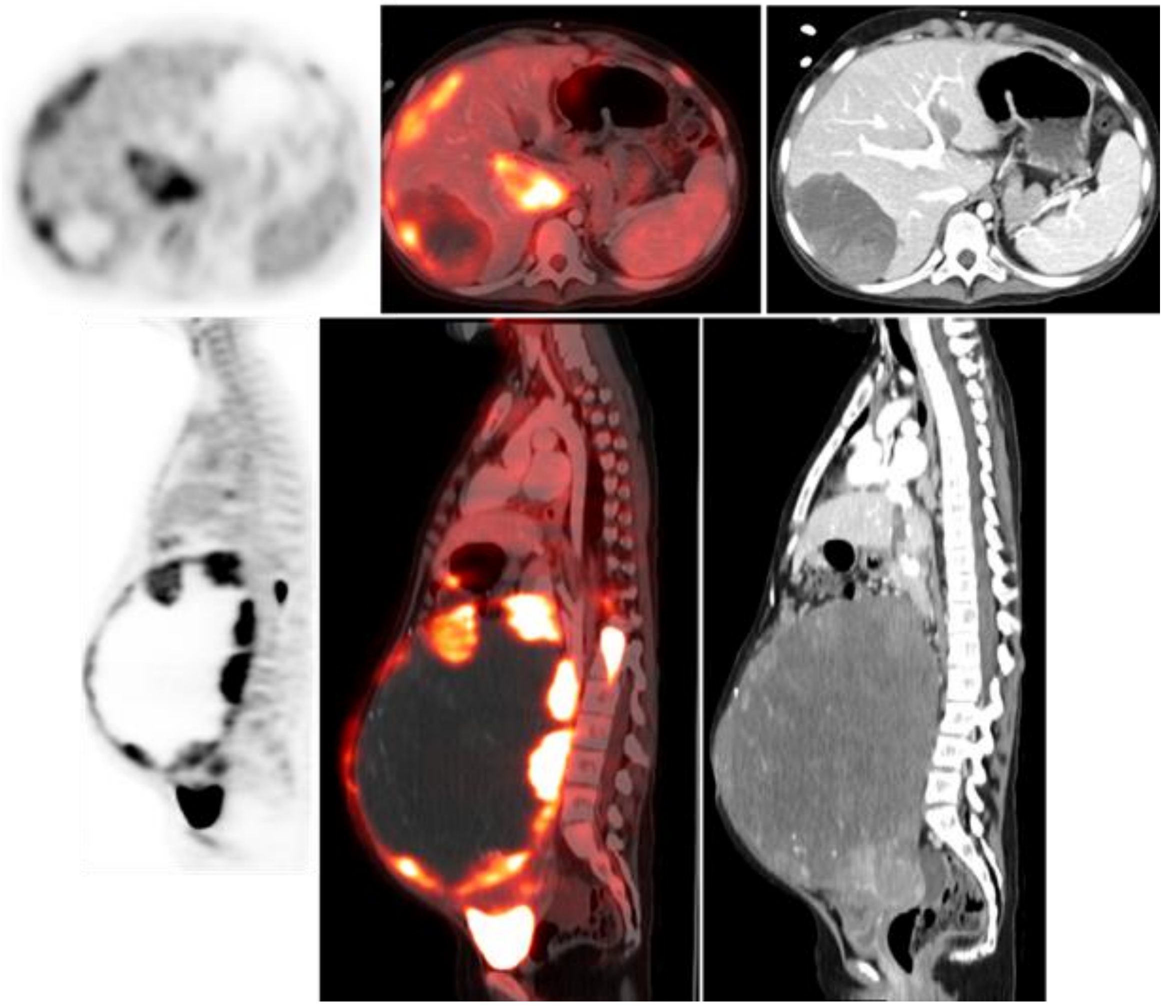

Figure 6. Axial and Sagittal FDG PET/CT showing a large intra-abdominal mass with solid nodular hypermetabolic components: a Rhabdoid tumor metastasis to the abdomen which occurred after primary brain ATRT resection. Unfortunately, the patient ultimately succumbed to the abdominal metastases.

Diagnostic challenges remain because appearance overlaps with other pediatric tumors (medulloblastoma, ETMR, ependymoma, choroid plexus tumors). However, red-flag features that favor ATRT include extremely low ADC, hemorrhagic/necrotic heterogeneity in very young children, lack of calcification, absence of an MRS taurine peak, and subgroup-consistent anatomic patterns (e.g., sellar/suprasellar lesions in ATRT-MYC). Emerging radiomics and machine-learning models that integrate morphology, diffusion, perfusion, and spectroscopy show promise for noninvasive classification and risk stratification, though clinical adoption is nascent. Nuclear medicine can complement MRI in select scenarios: FDG-PET often shows intense hypermetabolism useful for distinguishing recurrence from treatment change, amino-acid PET may improve lesion conspicuity (limited ATRT-specific data), and investigational tracers (e.g., B7-H3, GD2) foreshadow theranostic applications.

Clinical Management and Outcomes

Management is multimodal: maximal safe resection, intensive multi-agent chemotherapy (often including intrathecal therapy and high-dose chemotherapy with autologous stem-cell rescue), and radiotherapy when feasible. The COG ACNS0333 regimen established improved survival compared with historical controls, particularly when early focal radiotherapy was incorporated. Nonetheless, long-term survival remains modest compared with other pediatric CNS tumors, with outcomes influenced by age at diagnosis (<1 year worse), extent of resection, dissemination status, and possibly molecular subgroup. Survivorship challenges include neurocognitive, endocrine, and ototoxic sequelae, underscoring the need for precision approaches that balance control with late-effect mitigation.

Institutional Experience (SLCH Cohort)

At St. Louis Children's Hospital, a prospective ATRT database captures demographics, molecular subgroup, imaging, surgery, chemotherapy, and radiation parameters using standardized templates. Age-aware diffusion interpretation, grounded in developmental DTI norms, is applied to contextualize tumor ADC in infants and young children. Preliminary outcomes align with contemporary cooperative-group reports, reinforcing that timely focal radiotherapy and maximal safe resection are associated with improved survival, while omission or substantial delay of radiotherapy in very young patients correlates with early relapse. Ongoing benchmarking against international registries aims to refine risk-adapted strategies.

Radiation Therapy in ATRT

Radiation is a critical—but challenging—component of ATRT therapy. In localized disease, focal irradiation to the tumor bed (typically 54–59.4 Gy) is standard; in disseminated disease or positive CSF cytology, craniospinal irradiation (23.4–36 Gy) with a boost is considered. Modern techniques (proton therapy, IMRT/VMAT, IGRT) improve conformality and reduce dose to normal tissues, which is vital for infants and toddlers. Early incorporation of radiotherapy has improved local control and survival, yet risks of neurocognitive, endocrine, and ototoxic late effects persist, particularly under age three. Recent trends emphasize precision radiotherapy tailored to disease biology and patient vulnerability, with growing adoption of proton therapy, exploration of reduced-dose CSI in trials, and investigation of RT-immunotherapy combinations.

Recent Advances and Future Directions

Key advances include refinement of methylation-based subgroups and recognition of SMARCA4-deficient ATRT; multiparametric MRI/radiomics with subgroup-location correlations; integration of WHO 2021 molecular criteria; and translational pipelines evaluating epigenetic (EZH2) inhibitors, immune-targeted therapies (B7-H3, GD2, CAR-T), and patient-derived tumoroids for drug screening. International collaboration and data-sharing are accelerating risk-adapted, biology-driven care, with the goal of improving both survival and quality of survival.

Conclusion

ATRT illustrates the convergence of molecular pathology, advanced imaging, and multimodal therapy in modern pediatric neuro-oncology. Defined by SMARCB1/SMARCA4 deficiency and stratified into TYR, SHH, and MYC subgroups, ATRT exhibits imaging hallmarks—especially extreme diffusion restriction and taurine-negative spectra—that aid differentiation from embryonal mimics. While outcomes have improved with intensive therapy and earlier radiotherapy, precision medicine approaches that integrate subgroup biology, imaging biomarkers, and novel therapeutics are poised to drive the next gains in survival while mitigating late effects.

Table 1.

Tracer / Modality	Age / Setting	Tumor Site / Pattern	Key PET Finding	Clinical Utility / Note	Reference
18F-FDG PET/CT	Adolescent; primary leptomeningeal ATRT	Diffuse leptomeningeal (no parenchymal mass)	Hypermetabolic leptomeningeal uptake	Supported diagnosis and extent of LMD	Kayo et al., 2021 (Case report)

11C-Methionine PET; 18F-FDG PET; 201Tl SPECT; 123I-MIBG SPECT	16-year-old; frontal lobe ATRT	Frontal lobe mass	MET and FDG uptake described; multi-tracer evaluation	Metabolic characterization across tracers	Sasajima et al., 2002 (Case report)
18F-FDG PET (case reports, mixed)	Pediatric/adult; supratentorial & sellar	Hemispheric or sellar masses	Often high FDG; occasional atypical/low uptake described	Differentiate recurrence vs treatment change (context-dependent)	Narrative from case literature
Amino-acid PET (MET/FET)	Pediatric brain tumors (limited ATRT-specific data)	Embryonal tumors incl. ATRT (mixed)	Higher tumor-to-background than FDG (general peds neuro-onc)	Target delineation and response assessment potential	Cistaro et al., 2021 (review)
PET/MRI (hybrid)	Pediatric neuro-oncology	Varied	Combines metabolic and structural info	Promising adjunct; limited ATRT-specific series	Kang et al., 2022 (review)
B7-H3–targeted molecular imaging (investigational)	Pediatric brain tumors incl. ATRT	Molecular target (CD276)	High B7-H3 expression in ATRT supports theranostics	Potential future PET imaging/therapy pairing	Haydar et al., 2020; Theruvath et al., 2020

Abbreviations

ATRT – Atypical Teratoid/Rhabdoid Tumor
ADC – Apparent Diffusion Coefficient
CNS – Central Nervous System
DTI – Diffusion Tensor Imaging
MRI – Magnetic Resonance Imaging
RT – Radiotherapy
CSI – Craniospinal Irradiation
FDG – Fluorodeoxyglucose
PET – Positron Emission Tomography

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