Glioneuronal Tumors: A Pictorial Review and Practical Approach

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Abstract

Many radiologists struggle to parse between glioneuronal tumors due to significant overlap in imaging, histology, and long-term behavior. However, most glioneuronal tumors have long, primarily benign courses, and therefore, it is not uncommon for imaging to be the primary source for clinical decision-making. In cases requiring resection, imaging features can inform preoperative risk and determine the need for adjuvant therapies. While these tumors are diagnostically challenging, specific imaging features have stronger diagnostic and prognostic associations. This review aims to provide an approach to navigating the glioneuronal tumor differential for practicing radiologists and radiology trainees. Through a set of representative cases, key imaging features are discussed that inform specific glioneuronal tumor diagnoses as well as subsequent clinical management.

Keywords: glioneuronal tumors, central nervous system tumors, neuroradiology, pediatric neuroradiology

Introduction

As their name indicates, glioneuronal tumors contain glial and neuronal cellular features. They typically exhibit low-grade behavior and do not fall into the molecular subtype of other brain tumors such as gliomas, oligodendrogliomas, and astrocytomas.[1,2] Their clinical symptoms usually stem from mass effect and/or medically refractory seizures, and while they can occur across all age groups, they are often diagnosed in childhood or early adulthood.[2]

Advances in molecular pathophysiology have led the World Health Organization to update its diagnostic criteria for brain tumors, switching from an imaging or histopathological approach to genetic and molecular categorization.[1] Nevertheless, radiologists play a key role in diagnosis and prognosis. MRI is the modality of choice for tumor characterization, with occasional use of advanced techniques such as MR perfusion and spectroscopy (MRS) for indeterminate cases.[3]

Here, we present an overview of glioneuronal tumors and typical imaging findings. We also present an approach to differentiating these tumors from other types based on key imaging features that can inform diagnosis and subsequent management.

Review of Tumors

Supratentorial: Cortical

Desmoplastic Infantile Astrocytoma and Ganglioglioma

Desmoplastic infantile gangliogliomas (DIGs) and astrocytomas (DIAs) occur most often in infants under age 24 months.[4] DIGs demonstrate a slight male predominance (~60%) while DIAs have no gender predilection.[4,5] In contrast to other glioneuronal tumors, DIG/DIA presents with seizures in a minority of cases.[4,6] DIGs/DIAs are large tumors most often located within the superficial cortex in the frontal or parietal lobes (Figure 1).[4,6]

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Figure 1. Desmoplastic infantile gangliocytoma imaging features. (A) T1 postcontrast image in a 9-month-old girl demonstrates a cortical-based tumor in the frontoparietal vertex. (B) T2 FLAIR postcontrast imaging in a 1-year-old boy demonstrates a cortical-based tumor in the sylvian fissure. These tumors are mixed cystic-solid masses with cystic components (white arrows) deep to avidly enhancing solid components (black arrows). Patient B demonstrates the most common metastatic site, with leptomeningeal enhancement (arrowheads) and experienced multiple subsequent recurrences requiring multiple resections. Patient B's solid component (black arrow) also encases the insular M2 middle cerebral artery and adjacent deep veins, which is easiest to appreciate when comparing to the flow voids on the contralateral side (white arrowhead).

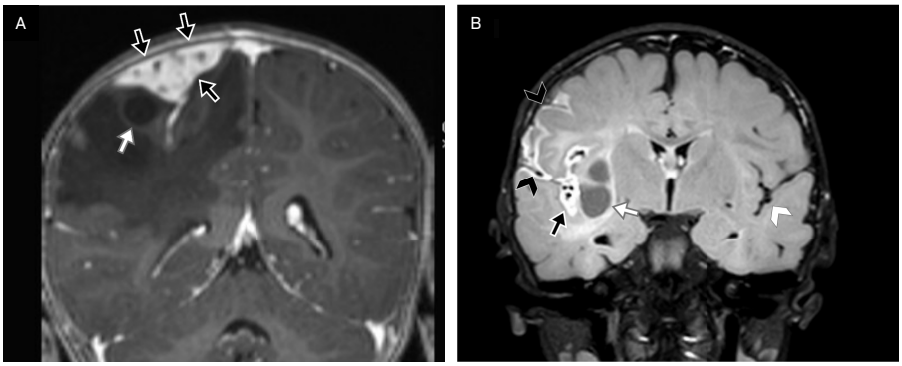
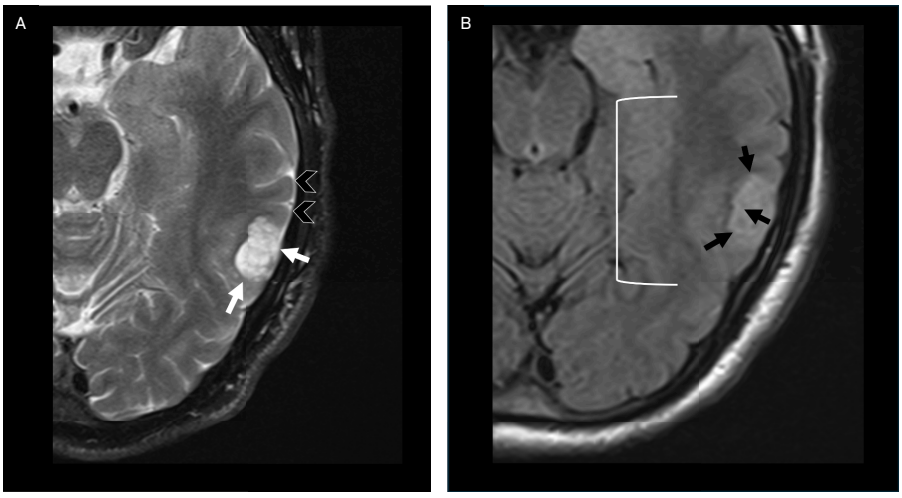


Figure 2. Dysembryoplastic neuroepithelial tumor. Axial T2 (A) demonstrates a T2-hyperintense multicystic lesion in the left temporal lobe (white arrows). On axial T2 FLAIR (B), the lesion demonstrates a hyperintense ring (black arrows), suggestive of the "FLAIR rim" sign. This tumor does not demonstrate features associated with higher grade tumors and worse outcomes. Specifically, there are no satellite lesions, which would most commonly be found along the medial aspect of the tumor (white bracket), and the adjacent cortex has no focal dysplasia.



Cystic components are found in most cases (>90%) and often located deep to solid components (Figure 1).[6] The peripheral solid components enhance vividly.[4,5] While these tumors are considered benign, they can demonstrate metastatic behavior; one case series has documented metastasis at presentation in 11.7% of cases (Figure 1B).[7]

The main differentials are pleomorphic xanthoastrocytoma (PXA), ganglioglioma, dysembryoplastic neuroepithelial tumor (DNET), and atypical teratoid/rhabdoid tumor (AT/RT). DIAs/DIGs typically present at younger ages and have stronger T2 hyperintensity compared with PXAs and gangliogliomas.[4] In addition, DIAs and DIGs enhance more strongly than DNETs.[4,6] AT/RTs are more likely to calcify and hemorrhage than DIAs and DIGs; however, calcification and hemorrhage can be seen in DIA/DIG cases with acute clinical symptoms.[4,5] Total resection is uncommon (27-30% of cases), and adjuvant therapy is recommended only in cases of documented tumor progression.[6]

Imaging offers a few key predictors of patient outcomes.[6] The leptomeninges are the most common site of metastasis (Figure 1), but intracranial and spinal metastases have also been reported.[6] Midline location is associated with more aggressive histological behavior, increased chance of recurrence, and increased intraoperative risk.[4,6] Specifically, involvement of the hypothalamus should be reported, as it is associated with increased mortality secondary to postoperative respiratory compromise.[4] Finally, the solid components of DIAs/DIGs usually encase vessels, which can help with diagnosis but can also limit surgical resection.[6]

Dysembryoplastic Neuroepithelial Tumors

Slow-growing and cortical-based, DNETs most commonly affect patients <20 years and are found in the temporal and frontal lobes.[8]

DNETs demonstrate a characteristic multicystic, "bubbly" lesion with high T2 and low T1 signal (Figure 2).[8] Multicystic morphology is associated more with DNETs than with other cortically based lesions such as oligodendrogliomas and gangliogliomas.[9] Most DNETs extend into the subcortical white matter, occasionally in a tapered fashion.[9] The FLAIR rim sign has been suggested as a specific finding for DNET (82% specificity in one case series) (Figure 2B).[10]

Some DNETs will demonstrate peripheral contrast enhancement, may contain macroscopic calcification (~20%), and rarely contain intratumoral hemorrhage.[11] They frequently present with multiple satellite lesions, preferentially occurring along the medial

aspect of the primary lesion.[8] Additionally, focal cortical dysplasia can be associated with higher-grade DNETs.[12]

Surgical resection provides effective seizure control. Persistent seizures have been seen in 46% of patients with satellite lesions but only 18% of patients without satellite lesions.[13]

Polymorphous Low-Grade Neuroepithelial Tumors of the Young

Polymorphous low-grade neuroepithelial tumors of the young (PLNTY) were previously considered a subtype of DNET, differentiated via mutations within the MAPK pathway.[14] They are primarily seen in children and young adults,[15] occurring only rarely in older adults.[16] Typically, PLNTYs are cortically based, heterogeneous cystic or mixed solid cystic masses (Figure 3) classically with central calcification[17] and mild, heterogeneous enhancement.[18] A "transmantle"-like signal abnormality (linear increased T2 signal in the deep white matter extending peripherally) can be seen, with some mixed evidence around its postresection persistence for predicting poorer seizure control.[18] Beyond seizure-associated morbidity, the tumor follows a benign course with minimal mass effect. There is low recurrence after total resection[17]; however, a recent meta-analysis reported that the extent of resection was not significantly associated with postoperative seizure control.[19]

Gangliogliomas

Gangliogliomas are the most common epilepsy-associated neoplasm, typically found in the temporal and frontal lobes.[20,21] They can occur infratentorially, including in the spinal column. They occur in both pediatric and adult populations, with most pediatric cases diagnosed in those over the age of 10 years[21] and adult cases at a median age of 32 years.[22]

On imaging, gangliogliomas are typically superficial, well-circumscribed mixed cystic and solid tumors, but a diffuse infiltrative appearance is possible. Enhancement is variable and associated with the solid components (Figure 4); infratentorial gangliogliomas are more likely to demonstrate "paintbrush" enhancement (dorsal predominant) compared with supratentorial tumors.[22] Brainstem gangliogliomas are associated with older patient age (median of 30 years), peritumoral edema, higher solid proportion, and lower rates of calcification.[23]

With their variable imaging appearance, the differential is wide, including PXA, PLNTY, and oligodendrogliomas. In general, PXAs enhance more vividly than gangliogliomas and very rarely calcify, while up to 50% of gangliogliomas calcify.[23] Finally, gangliogliomas have been reported to have higher normalized cerebral blood volume and lower normalized apparent diffusion coefficient compared with oligodendrogliomas.[24]

Tumor locations associated with worse outcomes include the brainstem, cerebellopontine angle, spinal cord, and midline tumors.[22] Brainstem involvement is often the defining factor for total vs subtotal resection.[21] There is mixed evidence for the ability of resection extent to predict long-term outcomes.[22,25,26] Finally, there have been rare case reports of malignant degeneration; in these cases, leptomeningeal involvement was common and MRS showed anaplastic features (increased choline, lipid, and lactate).[22]

Cerebral Gangliocytoma

Cerebral gangliocytomas (CGs) are rare (<3% of CNS tumors), occurring most frequently in children and young adults.[2] These tumors typically arise in the cerebral cortex, most often in the temporal lobe (~75%) (**Figure S1**), though occasional exophytic and rare extra-axial presentations have been described.

Sometimes exhibiting variable imaging features, CGs can be difficult to distinguish from other tumors, particularly gangliogliomas. Meningioma is in the differential, given that CGs can be extra-axial and demonstrate a dural tail.[2,27] MRS may be a helpful diagnostic adjunct, as CGs demonstrate a highly elevated choline signal and reduced N-acetylaspartate, a pattern more typical of high-grade gliomas than other glioneuronal tumors such as ganglioglioma.[27] Diagnosis is ultimately based on histopathology.

Supratentorial: Intraventricular

Myxoid Glioneuronal Tumors

Previously known as "DNET of the septum pellucidum,"[1,28] myxoid glioneuronal tumors (MGTs) are now defined by a gain-of-function mutation in the PDGFRA gene.[29] Given their similar histology and patient populations, MGTs and DNETs are difficult to distinguish from each other; therefore, imaging and genetic testing play an important diagnostic role.

The pathognomonic finding of MGT is location within the septum pellucidum (Figure 5), but they have also been reported in the midbrain.[29] Other imaging characteristics are similar to DNETs, with a multicystic morphology with well-defined margins and variable enhancement.[30] Unlike DNETs, no macrocalcifications have been reported in histologically proven MGTs.[29]

Central Neurocytoma

Originally classified as oligodendrogliomas, central neurocytomas (CNs) are now recognized as a distinct entity.[31,32] These tumors present at a median age of 30-31 years and typically demonstrate no gender predilection.[31,32]

Classically, CNs are found in the frontal horn of the lateral ventricles in close association with the foramen of Monro.[31,32] Most are unilateral at presentation, with bilateral involvement more common in larger tumors.[32] These tumors consist of mixed calcific, cystic, and solid tissue components, resulting in heterogeneous imaging characteristics (Figure 6). CNs may contain coarse "popcorn" calcifications on CT,[31,32] while cystic components produce a "Swiss-cheese" or "soap bubble" appearance on T2 sequences.[31,33] The solid tissue component

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Figure 3. Polymorphous low-grade neuroepithelial tumor of the young. Axial T2 (A) MRI demonstrates a T2-hyperintense cortically based lesion in the right temporal lobe (white arrow) with central heterogeneous signal. This central signal demonstrates susceptibility artifact (black arrow) on susceptibility-weighted imaging (B), consistent with calcification.

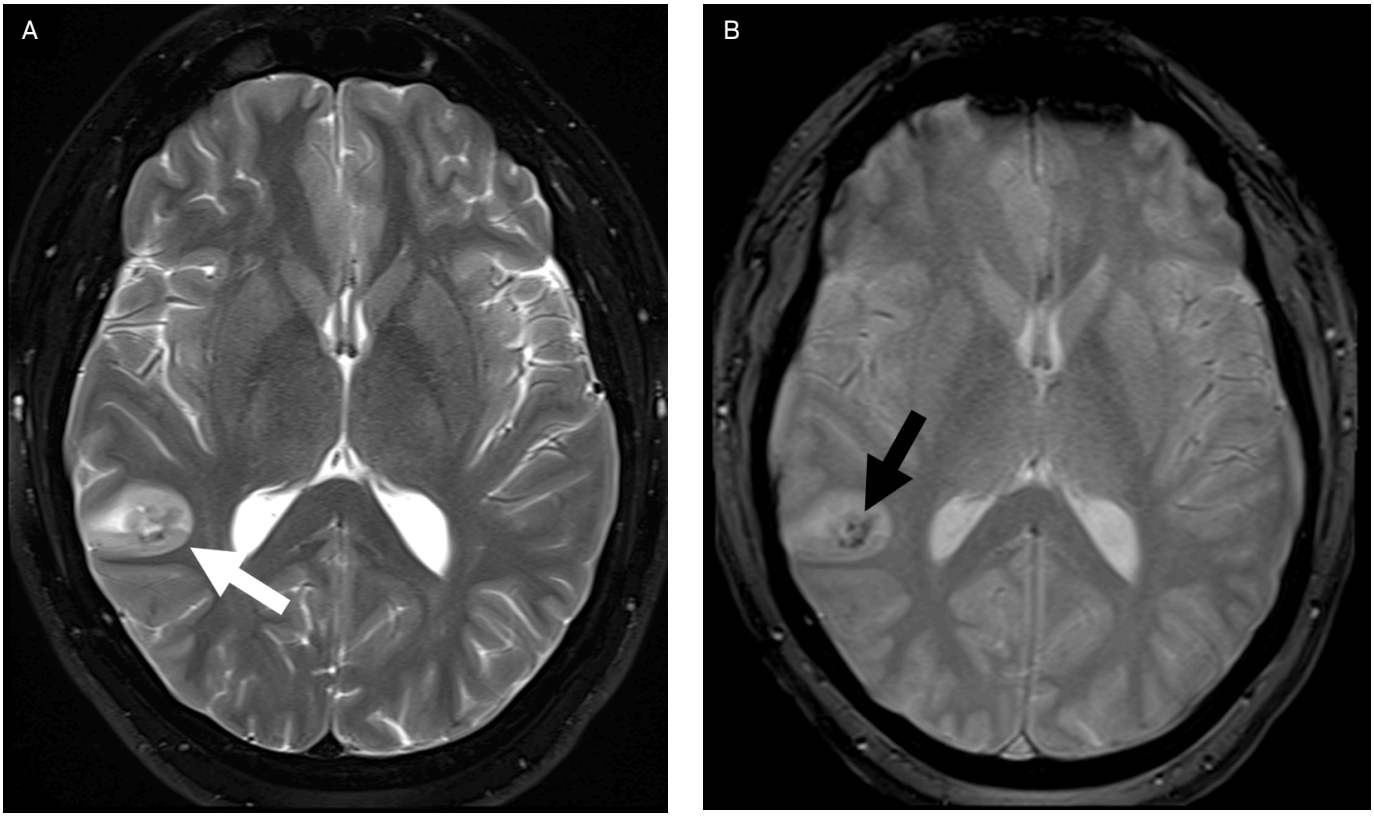


Figure 4. Variable imaging presentation of gangliogliomas. Ganglioglioma appearance can vary on imaging, owing to factors such as composition (cystic vs mixed cystic solid), borders (well-circumscribed vs infiltrative), and postcontrast enhancement. Two patients with pathology-proven gangliogliomas demonstrate this spectrum. Imaging of patient (A) shows a well-circumscribed, primarily cystic, tumor in the classic temporal lobe location with peripheral enhancement (white arrowheads) on T1 postcontrast imaging. Imaging of the second patient (B, C) demonstrates an infiltrative, mostly solid tumor with no enhancement (B, white oval), which is better visualized on T2 FLAIR (C, black arrowheads). Neither tumor had calcifications, which can occur in up to half of gangliogliomas. Given this variability, ganglioglioma is important to keep on the differential for cortical-based epilepsy-associated neoplasms.

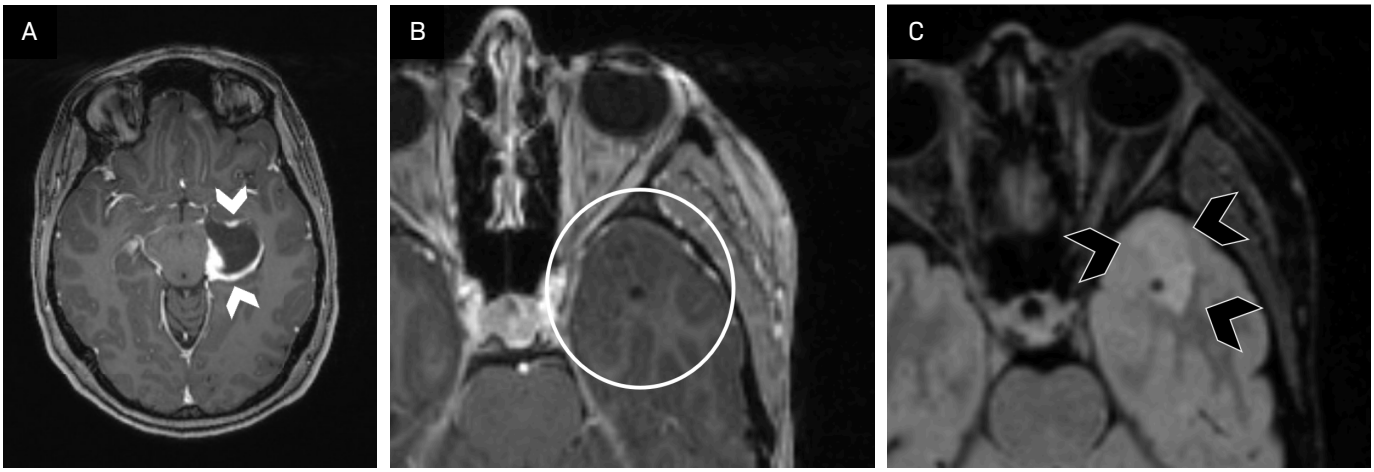


Figure 5. Myxoid glioneuronal tumor (formerly dysembryoplastic neuroepithelial tumor [DNET] of the septum pellucidum). Axial T2 FLAIR imaging (A) demonstrates a mass in the septum pellucidum (arrow) with imaging features similar to DNET: well-circumscribed with multiple hyperintense internal cystic structures. The mass is nonenhancing on axial postcontrast T1 image (B, arrowhead), consistent with the variable enhancement patterns of DNET. At biopsy, the mass was confirmed to contain histological characteristics of DNET of the septum pellucidum, which subsequently was classified as myxoid glioneuronal tumor. Genetic testing for PDGFRA was not performed for this patient owing to lack of testing availability but would be considered the gold standard for myxoid glioneuronal tumor diagnosis.

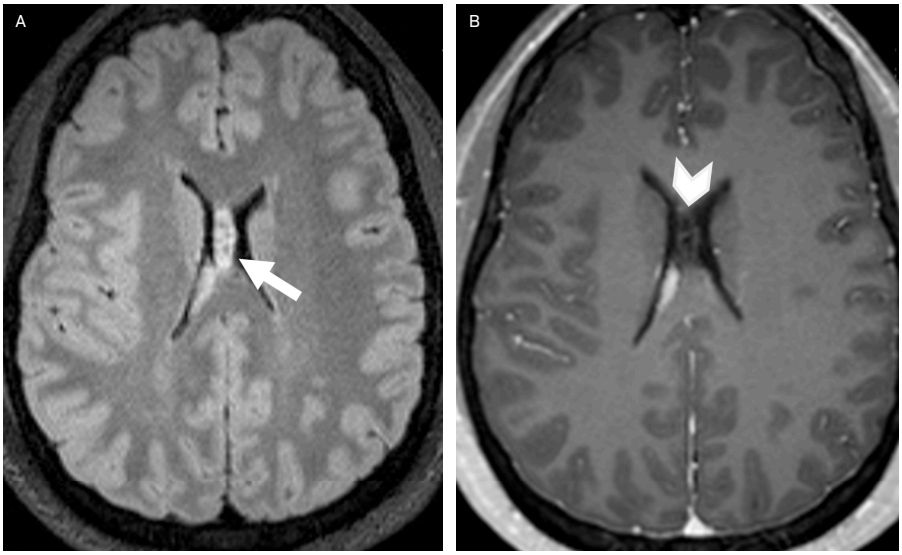
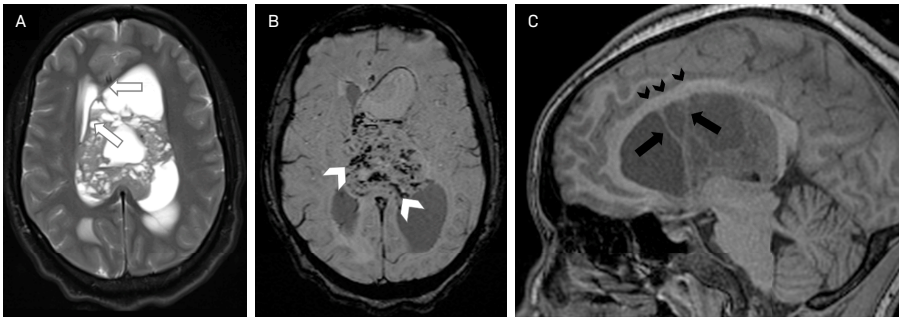


Figure 6. Central neurocytoma. Axial T2 (A) and susceptibility-weighted imaging (B) images show a mixed cystic solid mass with abutment and bowing of the septum pellucidum (white arrows). Susceptibility artifact (white arrowheads) from calcification and vascularity helps differentiate it from myxoid glioneuronal tumor. The "scalloping sign" is seen on sagittal T1 precontrast imaging (C) with peripheral cysts creating radial lines from their walls (black arrows) and a wavy appearance of the ventricular wall (black arrowheads).



dictates the degree of diffusion restriction and postcontrast enhancement, with the adjacent choroid plexus used for baseline enhancement comparison.[31,33,34]

While the imaging appearance of CNs can vary, specific characteristics can assist with diagnosis and prognosis. These include a broad-based septum pellucidum attachment (Figure 6), peripheral cystic components, periventricular edema, and the "scalloping sign," a wavy ventricular wall with adjacent rows of cysts.[12,15,16] On MRS, CNs typically demonstrate elevated choline and reduced creatinine and N-acetylaspartate[31,33]; elevated glycine is considered a sensitive but nonspecific finding for CN.[32]

Total resection is the gold standard treatment for CNs; however, this is limited by the degree of vascularity and potential neurologic complications, the rates of which range from 30% to 70%.[33] Adjuvant radiation is controversial in cases of subtotal resection but may be considered for tumors that demonstrate atypical and/or aggressive histologic features (~20% of CNs).[31,32,35] Imaging findings associated with aggressive histologic features include enhancement, larger size, and the presence of irregular vessels.[36] Radiation is also considered for recurrent disease, which typically occurs 3-4 years post-treatment.[31,35]

Supratentorial: Subcortical Tumors

Multinodular and Vacuolating Neuronal Tumors

Primarily occurring in adults, multinodular and vacuolating tumors (MVNTs) present at mean and median ages ranging from 38 to 45 years.[37,38] They are benign tumors with very slow growth and do not typically require resection.[38]

Their classic MRI appearance is a cluster of nonenhancing T2 and FLAIR hyperintense nodules centered at the cortical-subcortical junction, most commonly in the temporal lobe (Figure 7), without restriction.[37,38] The differential is wide and includes other glioneuronal tumors and non-neoplastic processes such as prominent perivascular spaces or focal cortical dysplasia. The most appropriate follow-up MRI protocol for MVNTs continues to be debated.[38]

Papillary Glioneuronal Tumors

Extremely rare entities, papillary glioneuronal tumors (PGNTs) primarily affect adults in their second decade.[39] There is no gender-based predilection.

The imaging features of these tumors are similar to those of other glioneuronal tumors, with a mixed solid cystic appearance. The 2 features suggestive of PGNT are location—80% are within or adjacent to the lateral ventricles—and septated cystic components, though these are not specific.[40] PGNTs are generally diagnosed histologically, genetically differentiated by the fusion of SLC44A1-PRKCA proteins.[41] Treatment involves resection, which has a good prognosis.[2]

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Figure 7. Multinodular and vacuolating neuronal tumor (MVNT). Axial T2 fat-saturated image demonstrates a cluster of juxtacortical hyperintense cysts (arrow) in the right parietal lobe, classic findings for MVNT. This lesion will typically not demonstrate enhancement on postcontrast images nor will it have diffusion restriction.

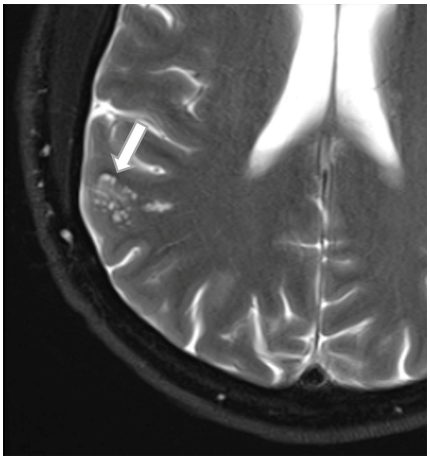
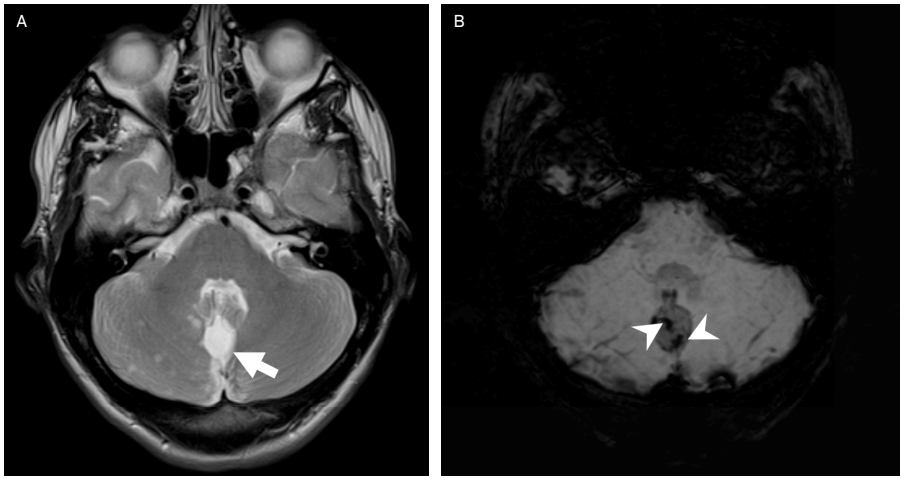


Figure 8. Rosette-forming glioneuronal tumor (RGNT). Axial T2 imaging (A) demonstrates a small (<3.0 cm) hyperintense lesion in the posterior fossa abutting the 4th ventricle (arrow). Susceptibility-weighted imaging (B) has susceptibility artifact (arrowheads) consistent with microhemorrhages. Susceptibility artifact from calcification would be exceedingly rare in RGNT and more consistent with a glioneuronal tumor or pilocytic astrocytoma.



Posterior Fossa

Rosette-Forming Glioneuronal Tumors

Rosette-forming glioneuronal tumors (RGNTs) are defined histologically[42] and typically present in adults at a mean of 23 years, with a slight female predilection (1.36:1).[43] The classic location is adjacent to the 4th ventricle, with local parenchymal extension (Figure 8).[2] Less common sites include the cerebellum and spine.[43] Given their mixed solid-cystic composition, their imaging appearance is variable; thus, tissue sampling is required for diagnosis.

The main differential considerations are pilocytic astrocytoma and DNET. Most reported RGNTs are <3.0 cm,[43] with a diameter <3.5 cm being just over 3 times more likely for RGNT.[44] Calcifications in RGNTs are exceedingly rare, with only 2 cases reported,[43] while pilocytic astrocytomas and DNETs have reported calcification incidences of 17%[45] and 20%,[11] respectively. Microhemorrhage is common in RGNTs (Figure 8), and while pilocytic astrocytomas can hemorrhage, it is exceedingly rare for DNETs, reported in 3%.[46]

While RGNTs are considered benign,[2] mass effect can be significant and require resection.[47] Pineal involvement is possible and should be reported, as it changes surgical approach.[48] Overall recurrence rates between total and subtotal resection of these tumors are similar, at about 10%[44]; however, the recurrence/progression timeline for subtotal resection vs total resection is reported at an average of 2.9 and 6.2 years, respectively.[44] Additionally, there is evidence that a higher proportion of solid tissue within the tumor predicts higher recurrence risk and worse histological features.[43] Rarely, RGNTs can develop satellite lesions and CSF involvement, which can lead to drop metastases; thus, postcontrast series should be evaluated for leptomeningeal involvement.[42,43] Finally, MRS usually shows elevated choline, reduced N-acetylaspartate, and absent lactate and lipid peaks[2]; deviations from this pattern should raise suspicion for malignant transformation.

Dysplastic Cerebellar Gangliocytomas

Also known as Lhermitte-Duclos disease, dysplastic cerebellar gangliocytomas are found primarily in patients with Cowden syndrome.[49] Arising in the cerebellum, they are benign, with no reported malignant transformation.[49] Surgical resection provides definitive treatment in acute cases; however, these tumors often do not require intervention.[50]

MRI demonstrates a unilateral cerebellar parenchymal mass with T2 hyperintensity, no enhancement, and variable diffusion restriction. Widened cerebellar folia create the pathognomonic "tiger-stripe" pattern,[49,50] which is primarily seen in adults.[51] SWI and contrast-enhanced sequences can demonstrate prominent vascular structures within the folia (**Figure S2**),[52] while MRS may reveal reduced choline and an inverted lactate peak.[52,53] The differential is limited, but subacute infarct or cerebritis can be considered in patients with acute deterioration.

Discussion

Differentiating between glioneuronal tumors is challenging, owing to similarities in demographics, clinical presentation, and imaging appearance, as well as their overall low incidence, resulting in limited reports in the literature. In this paper, we propose a diagnostic approach to these tumors, present representative cases, and describe prognostic imaging features based on our review of the current literature.

Owing to their shared cellular origin, most glioneuronal tumors have a mixed cystic-solid composition (Figure 9).

Figure 9. Summary of classic glioneuronal tumor imaging findings.

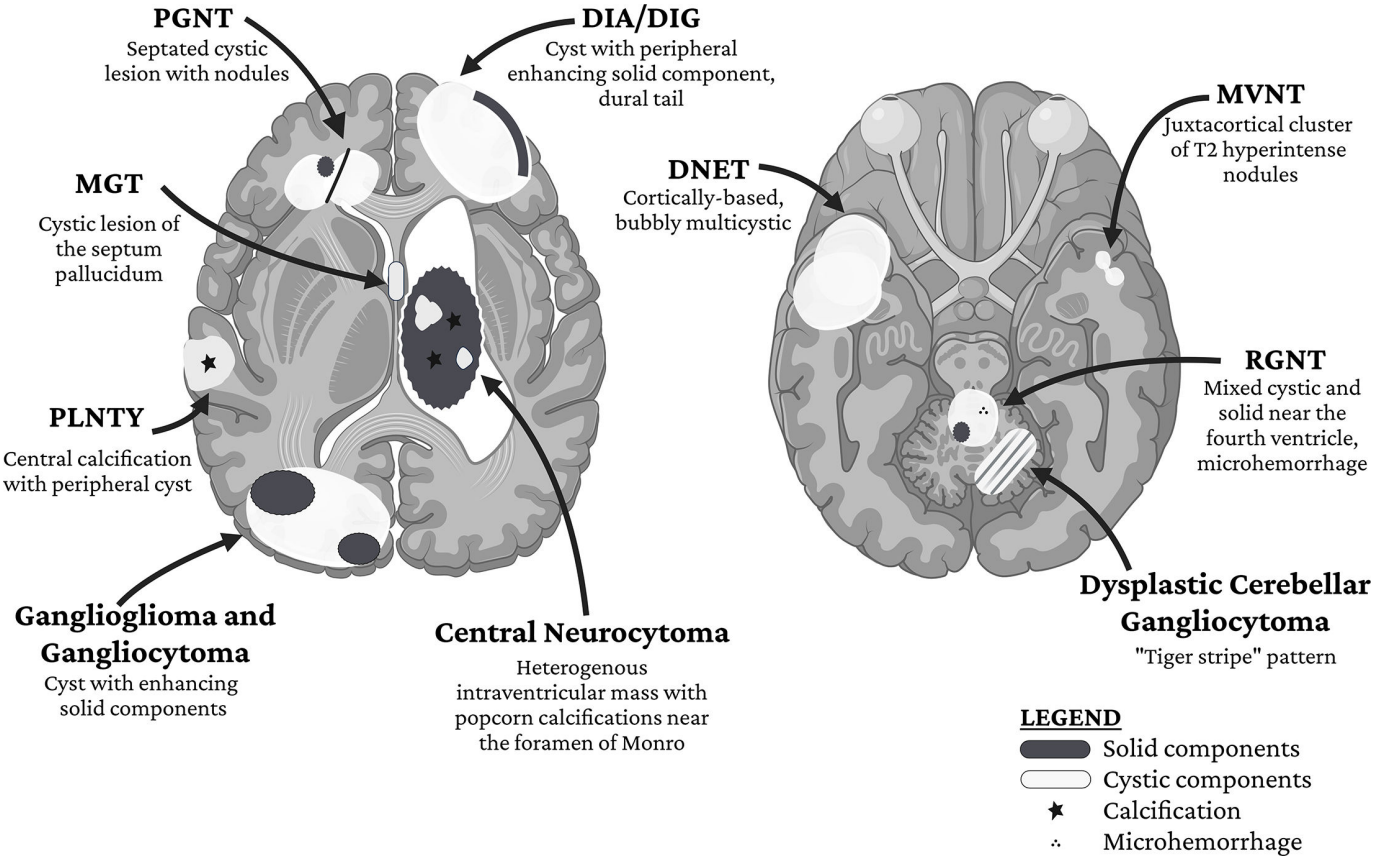
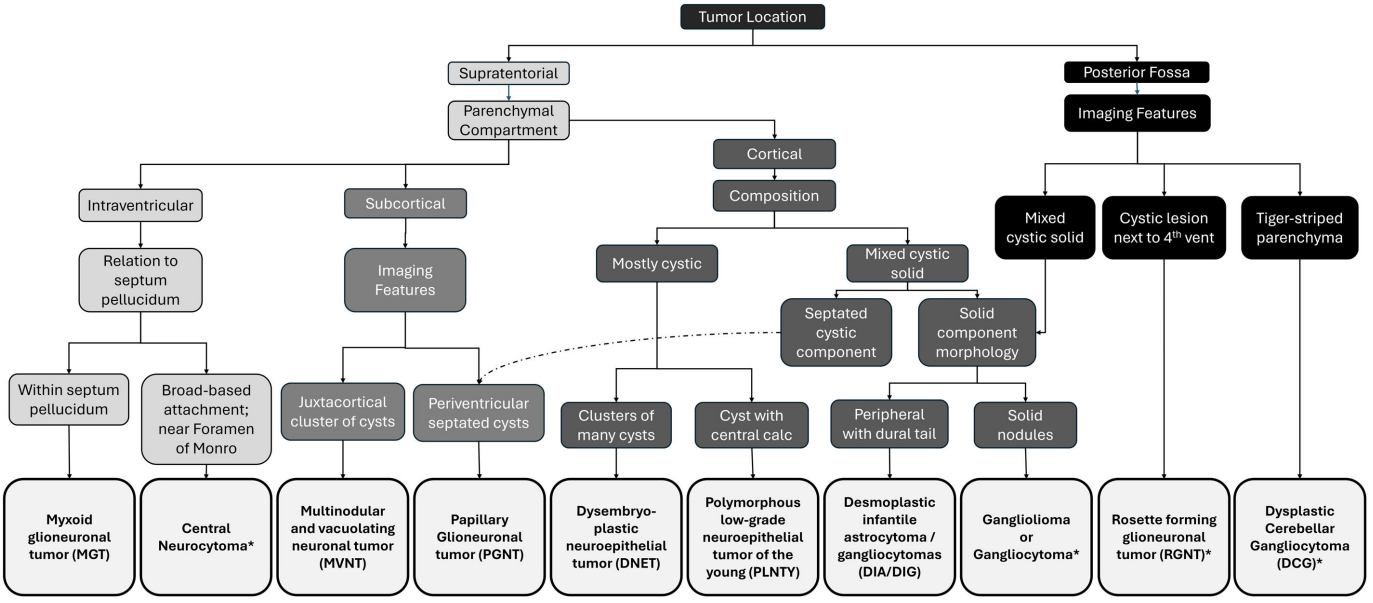


Figure 10. Approach to glioneuronal tumor differentiation. Glioneuronal tumors can overlap considerably in their imaging presentation. This flowchart shows the most prevalent presentations. Note that gangliogliomas are particularly variable in their imaging appearance and should be considered in the setting of an atypical cortical-based tumor. * indicates potential role for MR spectroscopy or MR perfusion.



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Location and parenchymal compartment are most useful for initially narrowing the differential, followed by the morphology and arrangement of the specific tumor components (eg, peripheral vs septated cysts, calcification vs no calcification) (Figure 10). MR perfusion and spectroscopy may be helpful in some cases. Ultimately, many glioneuronal tumors require histopathology for definitive diagnosis, but some patients may choose to forgo biopsy, relying instead entirely on image-based diagnosis.

Radiologists have a great impact on the management of glioneuronal tumors, as the primary treatment consists of excision with continued surveillance. Preoperative reports should include features that could alter operative approach and risk, such as involvement of midline structures and their relationship to major vessels. Furthermore, specific imaging features are associated with more aggressive histopathology in certain glioneuronal tumors; these can inform care management discussions. While the evidence is mixed for several glioneuronal tumors on the relationship between resection extent and long-term outcomes, including seizure control, the presence of metastatic foci or recurrent tumor on imaging affects the decision whether to pursue adjuvant systemic therapy.

In conclusion, while differentiating between glioneuronal tumors is challenging, narrowing the differential diagnosis is possible with careful assessment of tumor location and relational morphology. Radiologists can provide useful evidence for clinical management, including surgical approach, operative risk, and the need for adjuvant treatment.

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