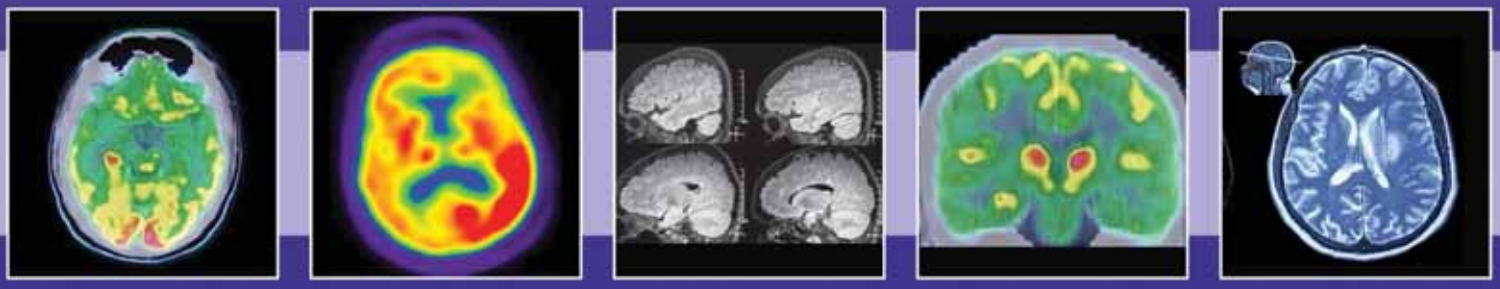
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Pitfalls in The Diagnosis of Neurological Disorders



Ambar Chakravarty

Foreword
Robert C Griggs

JAYPEE

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CHAPTER 12.2

Pitfalls in the Diagnosis of Brain Tumors: A Neuroradiologist's Perspectives

Sukalyan Purkayastha, Soumik Das

INTRODUCTION

Brain tumors encompass a wide variety of benign as well as malignant pathologies that arise from the different cellular components of the neuroparenchyma, the meninges, or the skull. The classification system of brain tumors has evolved progressively over the last few decades as we have incorporated and integrated microscopic features and immunohistochemical (IHC) expression of lineage-associated proteins into the current World Health Organization (WHO) classification system of brain tumors. Imaging markers for specific histologic subtypes of some types of tumors have been established. Also, newer imaging modalities such as MR perfusion imaging, diffusion-weighted magnetic resonance imaging (MRI), single-photon emission tomography, positron emission tomography, and MR spectroscopy (MRS) have helped in narrowing down the diagnosis in doubtful cases. We shall be discussing the most common types of brain tumors and their differential diagnoses, the pitfalls, and how to avoid them.

ASTROCYTOMAS

Pilocytic Astrocytoma

Pilocytic astrocytomas[1] that arise in the posterior fossa may mimic medulloblastoma. These typically do not show diffusion restriction whereas medulloblastomas restrict on diffusion-weighted imaging (DWI).

Posterior fossa ependymoma is a tumor that extrudes out through the foramen of Magendie and lateral recesses and can resemble pilocytic astrocytomas.

The imaging appearance of hemangioblastoma can closely resemble pilocytic astrocytoma, but pilocytic astrocytomas are tumors of children whereas hemangioblastomas which are tumors of middle-aged adults. Hemangioblastomas have significant peritumoral edema and markedly elevated relative cerebral blood volume (rCBV).

Pilomyxoid astrocytoma is a differential diagnosis of hypothalamic pilocytic astrocytoma. Hemorrhage is relatively common in pilomyxoid astrocytoma but rare in hypothalamic pilocytic astrocytoma.

Demyelinating disease and postviral inflammation can mimic visual pathway pilocytic astrocytomas. Optic neuritis can cause enlargement and enhancement of the optic nerves and chiasm.

The differential diagnosis of hemispheric pilocytic astrocytomas with a "nodule plus cyst" appearance is ganglioglioma (GG). GGs are mostly cortically based and frequently calcify. Pleomorphic xanthoastrocytomas (PXAs) can present with a solid "nodule plus cyst" but are tumors of young adults, and not children.[2] PXAs often incite meningeal reaction ("dural tail" sign).

Subependymal Giant Cell Astrocytoma

One of the major differential diagnoses of subependymal giant cell astrocytoma (SEGA) in patients with tuberous sclerosis is a subependymal nodule (SEN). SENs remain stable over time, but SEGAs show slow progressive growth and eventually need surgical treatment. SEGAs typically appear close to the foramen of Monro, but SENs may lie anywhere along the ventricular margin, especially close to the caudothalamic groove.

Other close differential diagnoses are central neurocytoma (a "bubbly" tumor arising in the body of the lateral ventricle) and subependymoma (a tumor of middle-aged and elderly patients).[3]

Pleomorphic Xanthoastrocytoma

The most important differential diagnosis of PXA is GG, a cortically based epileptogenic tumor.

Another less common tumor having a "cyst + nodule" appearance mimicking PXA is hemispheric pilocytic astrocytoma.[2]

Dysembryoplastic neuroepithelial tumor (DNET) is another cortically based epileptogenic tumor but characteristically has a multicystic "bubbly" appearance.

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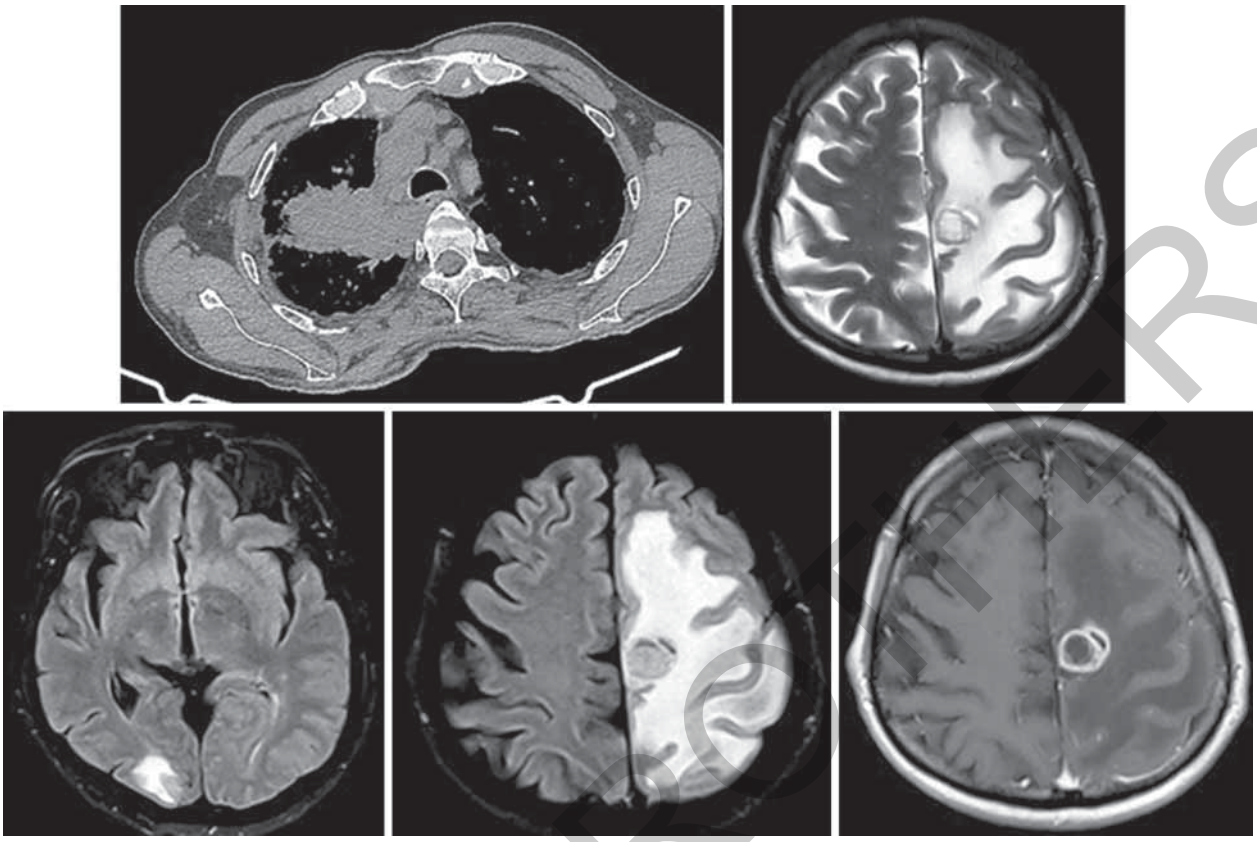


FIG. 1: Axial CT scan of thorax reveals a mass lesion in the right lung. Axial T2W and fluid-attenuated inversion recovery (FLAIR) images of brain reveal multiple neuroparenchymal lesions. The larger lesion in the left frontal region is associated with disproportionate edema with partial inversion of the central necrotic component on FLAIR sequence. Heterogeneous predominantly peripheral enhancement is noted on postcontrast T1W study.

Oligodendroglioma (OG) may present as a slow-growing cortical–subcortical white matter junction lesion that shows adjacent calvarial remodeling but lacks the typical "cyst + nodule" appearance.

Glioblastoma Multiforme

The major differential diagnosis of glioblastoma multiforme (GBM) is metastasis. Metastases are usually multiple and often occur at the gray–white matter junction. Large metastases (**Fig. 1**) are discrete and have disproportionate perilesional edema, not diffusely infiltrating like GBM.

Other glioblastoma mimics include primary central nervous system (CNS) lymphoma, anaplastic astrocytoma, and anaplastic OG. Anaplastic astrocytomas are generally nonenhancing although some may enhance and in such cases, it may be difficult to differentiate them from GBM (**Fig. 2**) on the basis of imaging alone.

Primary CNS lymphoma usually involves the corpus callosum but is usually nonnecrotic in the absence of human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS). Gliomatosis cerebri (GC) is a type of diffuse glioma which by definition involves three or more lobes with frequent bihemispheric, basal ganglionic, and/or infratentorial extension.

Abscess is a non-neoplastic differential of GBM. Abscesses typically restrict on DWI and show cytosolic and succinate amino acids on MRS.

"Tumefactive" demyelination may show rim enhancement. An incomplete rim with the open segment pointing toward the sulcus and cortex is typical.

Diffuse Midline Glioma, H3K27M Mutant

The differential diagnosis for unilateral thalamic H3K27M-mutant glioma is the same as other IDH-wild-type WHO grade III and II astrocytomas.[4-6]

Bithalamic tumors may resemble the artery of Percheron infarct, internal cerebral vein thrombosis, acute necrotizing encephalopathy, and Wernicke encephalopathy.

NONASTROCYTIC GLIAL NEOPLASMS

Oligodendrogliomas

Diffuse astrocytoma is a major differential of OG. Diffusely infiltrating astrocytomas predominantly involve the white matter and are nonenhancing. Necrosis and hemorrhage are less common in OG than anaplastic astrocytomas but are not definitive.[7]

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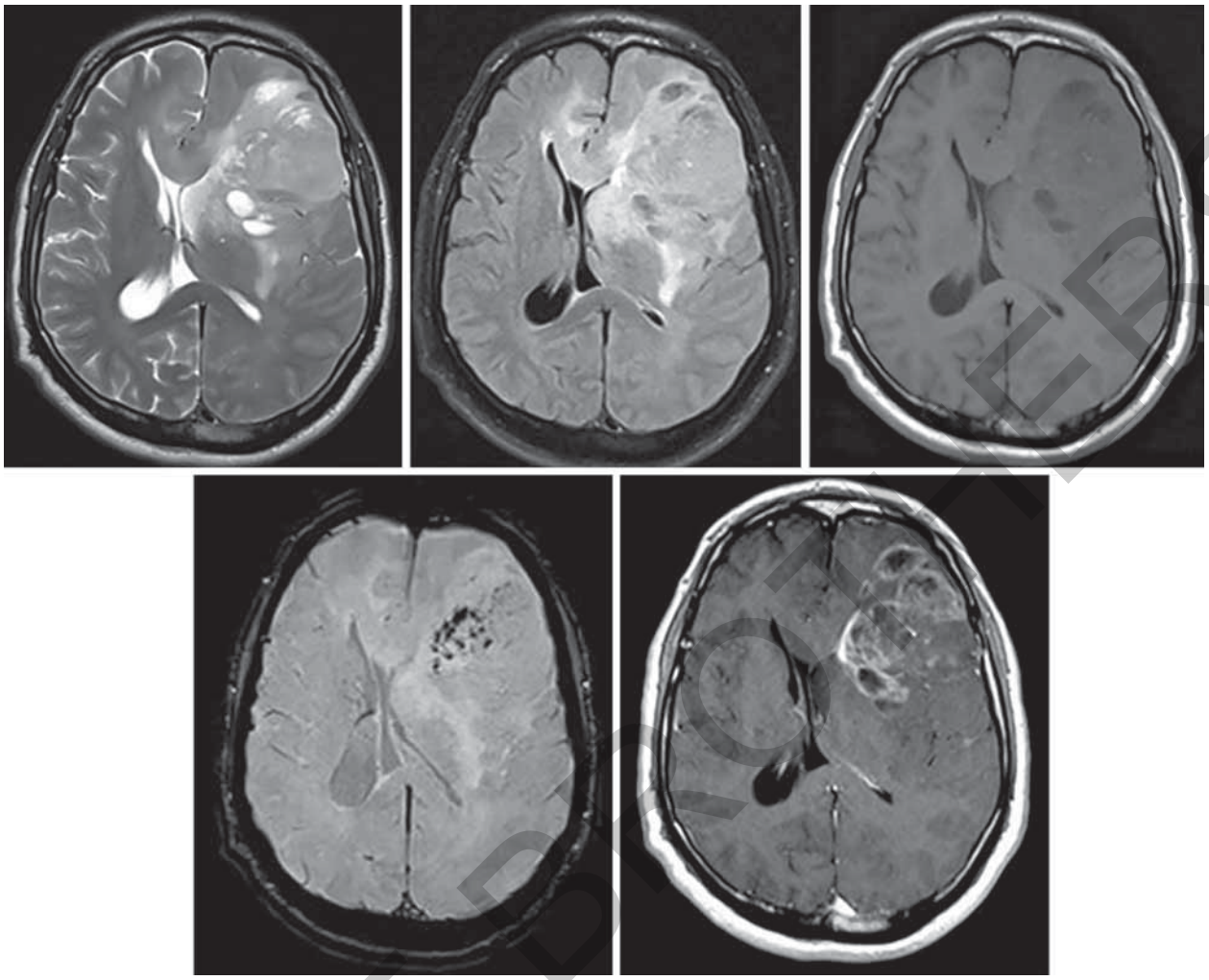


FIG. 2: Axial T2W and fluid-attenuated inversion recovery (FLAIR) sequences of brain reveal a heterogeneous intra-axial lesion involving the left frontal lobe and adjacent gangliocapsular region with extension across the genu of corpus callosum to involve the right frontal lobe. The cystic areas within the lesion show partial inversion on FLAIR sequence. Foci of blooming are noted within the lesion on susceptibility weighted imaging (SWI) sequence. Heterogeneous predominantly peripheral and patchy enhancement is noted on postcontrast T1W study.

Other epileptogenic cortically based tumors include DNET and GG. GGs have "cyst + nodule" appearance and commonly involve the temporal lobe. DNETs have a "bubbly" appearance.

Central neurocytomas are usually intraventricular, whereas OGs are cortically based lobar masses.

Extraventricular neurocytoma may be completely indistinguishable from OG on imaging.

Ependymoma

Differential diagnosis of ependymoma is location dependent.[8] Medulloblastoma is the main differential of infratentorial ependymoma. Ependymomas arise from the floor of the fourth ventricle whereas medulloblastomas arise from the roof. Medulloblastomas show diffusion restriction, are hyperdense on CT, and usually show CSF dissemination at the time of initial diagnosis. Hemorrhage, cysts, and calcification are more common in ependymoma as compared with medulloblastoma.

Pilocytic astrocytoma usually involves the cerebellar hemispheres and is common in children and young adults.

Glioblastoma or anaplastic astrocytoma is the main differential diagnosis of supratentorial ependymoma.

Atypical teratoid/rhabdoid tumor (AT/RT) and primitive neuroectodermal tumor may mimic parenchymal ependymoma in young children.

Subependymoma

In elderly patients, the main differential is intraventricular metastasis. Intraventricular metastases usually involve the choroid plexus. In young adults, central neurocytoma is a differential. A typical location of central neurocytoma is the body of the lateral ventricle and it has a "bubbly" appearance.

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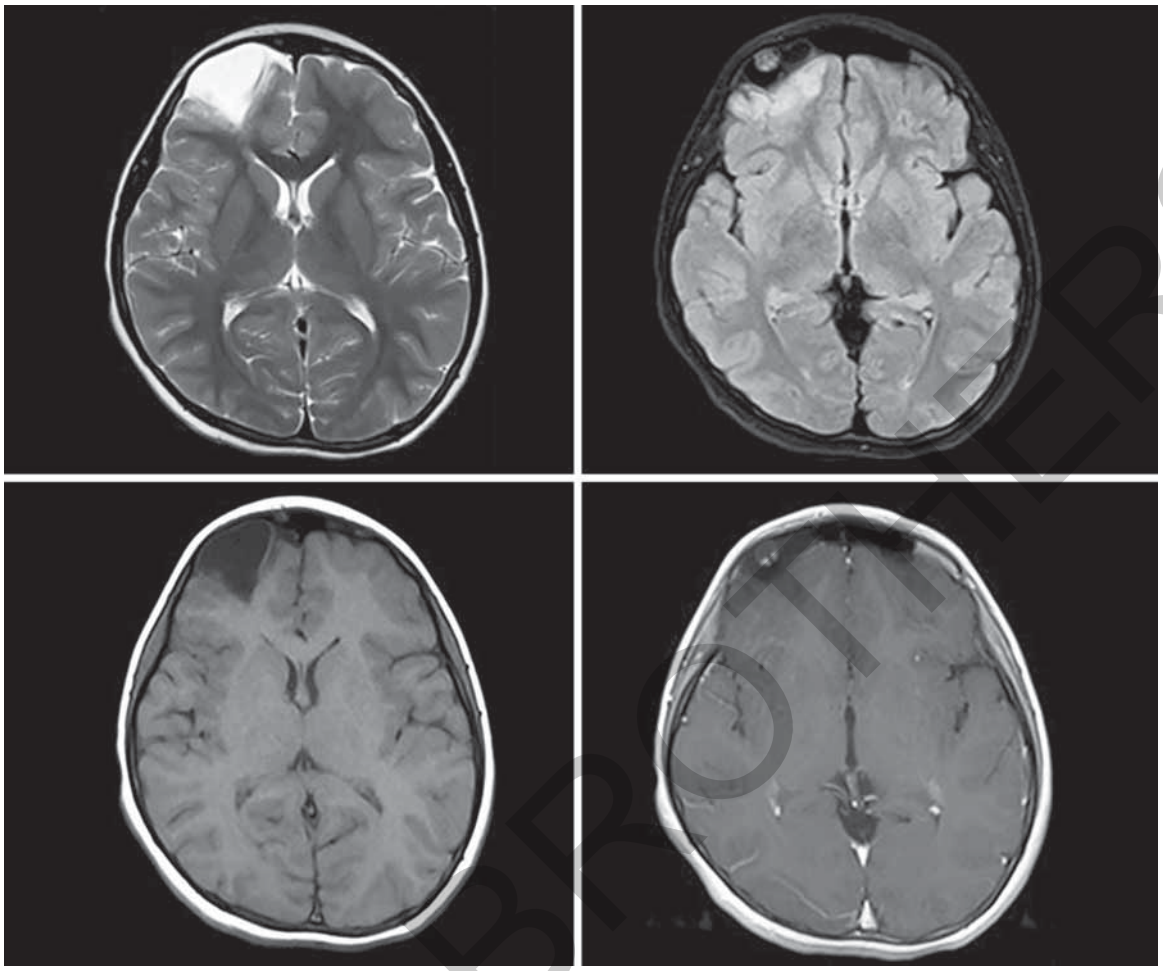


FIG. 3: Axial T2W image reveals a cortical-based cystic lesion with involvement of the adjacent white matter in the right frontal lobe. Scalloping of the adjacent calvarium is noted. Inversion of the cystic component of the lesion is noted on fluid-attenuated inversion recovery (FLAIR) study. The nodular component of the lesion is FLAIR hyperintense with subtle foci of enhancement on postcontrast T1W study.

Choroid Plexus Papilloma

The main differential diagnoses of choroid plexus papilloma (CPP) are atypical CPP and choroid plexus carcinoma (CPCa).[9] Brain parenchymal invasion is more common with CPCa than CPP.

Villous hypertrophy of the choroid is usually bilateral and diffusely involves the entire choroid plexus.

Choroid plexus xanthogranulomas appear as bilateral multiloculated cysts within the choroid plexus glomus.

NEURONAL AND GLIONEURONAL TUMORS

Ganglioglioma

The main differential diagnosis of GG is diffuse astrocytoma. However, diffuse infiltrating astrocytomas are typically nonenhancing.

Pleomorphic xanthoastrocytoma usually has "cyst + mural nodule" appearance with a dural "tail," thereby distinguishing it from other epileptogenic cortical neoplasms.

Dysembryoplastic neuroepithelial tumors typically have multicystic "bubbly" appearance with a fluid-attenuated inversion recovery (FLAIR) hyperintense rim. In contrast to GG (**Fig. 3**), enhancement is rare.

Oligodendroglioma is typically more diffuse and less well delineated than GG.[10]

Desmoplastic Infantile Astrocytoma/ Ganglioglioma

The major differential diagnosis of desmoplastic infantile astrocytoma (DIA)/desmoplastic infantile ganglioglioma (DIG) is teratoma. Teratoma is much more heterogeneous appearing than DIA/DIG and often extends extracranially.[11,12]

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Dysembryoplastic Neuroepithelial Tumor

The main imaging differential diagnoses are focal cortical dysplasia, GG, and multinodular and vacuolating neuronal tumor (MVNT) of the cerebrum.[13-15] The "bubbly" appearance of DNET and hyperintense rims demonstrable in FLAIR sequences are helpful distinguishing features. MVNTs are typically multifocal and occur in the deep layers of the cortex and adjacent white matter.

The rare angiocentric glioma closely resembles DNET on imaging, but a hyperintense rim is seen on T1WI, not FLAIR.

Rosette-forming Glioneuronal Tumor

Metastasis may be a rare differential of Rosette-forming glioneuronal tumor (RGNT). Ependymoma with hemorrhage and cysts may mimic RGNT. Pilocytic astrocytoma rarely shows hemorrhage. CPP shows frond-like papillary appearance with intense contrast enhancement.

Diffuse Leptomeningeal Glioneuronal Tumor

The differential diagnosis of diffuse leptomeningeal glioneuronal tumor (DLGNT) is limited.[16] Severe tubercular or pyogenic meningitis may be associated with leptomeningeal enhancement but lacks multifocal leptomeningeal T2/FLAIR hyperintense cysts.

Gangliocytoma

The main differential diagnosis of gangliocytoma is GG. Cortical dysplasia is another cause of epilepsy; however, it is nonenhancing.

Dysplastic Cerebellar Gangliocytoma

Dysplastic cerebellar gangliocytoma is also known as Lhermitte–Duclos disease (LDD).[17] Imaging findings of LDD are classical. Differential diagnoses include subacute cerebellar infarction and medulloblastoma.

GGs typically show enhancement but do not show "tiger stripes."

Rare cases of cerebellar cortical dysplasias and posterior fossa tuberous sclerosis complex (TSC) may mimic LDD.

Central Neurocytoma

The main differential diagnosis of central neurocytoma (CNC) is subependymoma.

Subependymal giant cell astrocytoma occurs close to the foramen of Monro. Other imaging features of tuberous sclerosis such as cortical tubers and subependymal nodules are usually associated.

An intraventricular metastasis may be considered as a differential in older patients.

True intraventricular OG is rare.[18,19] Differentiation from CNC is on the basis of IHC. OGs are synaptophysin-negative and show mutations of *OLIG2* and 1p,19q.

PINEAL AND GERM CELL TUMORS

Pineal Region Tumors

Pineocytoma

The main differential diagnosis of pineocytoma is a pineal cyst. Germinoma characteristically "engulfs" the pineal calcifications and shows diffusion restriction on DWI and avid contrast enhancement. Pineal parenchymal tumor of intermediate differentiation (PPTID) shows a more "aggressive" imaging appearance than a pineocytoma.

Pineoblastoma

The main differential diagnosis of pineoblastoma (PB) is PPTID.[20] PBs usually affect children. Germinomas more commonly affect the adolescent age group.

Germ Cell Tumors

Germinoma

The main differential diagnoses of germinoma are mixed germ cell tumor and nongerminomatous germ cell tumors. Bifocal lesions are almost always germinomas.[21,22]

PBs may mimic germinoma but "explode" rather than "engulf" pineal calcifications. Pineal parenchymal tumor of intermediate differentiation generally affects middle-aged and older adults.

The most important differential diagnosis of germinoma involving the suprasellar region is Langerhans cell histiocytosis (LCH). LCH does not produce oncoproteins.

Neurosarcoidosis in an adult may give rise to a suprasellar mass resembling germinoma.

EMBRYONAL NEOPLASMS

Medulloblastoma

Histologically defined medulloblastomas:[23,24]

- Classic medulloblastoma
- Medulloblastoma variants:
  - Desmoplastic/nodular medulloblastoma
  - Medulloblastoma with extensive nodularity
  - Large cell/anaplastic medulloblastoma

The main differential diagnosis of classic medulloblastoma is a medulloblastoma variant. A posterior fossa AT/RT can also mimic classic medulloblastoma.

Lhermitte–Duclos disease can mimic desmoplastic MB, but the striated pattern is quite characteristic of LDD.

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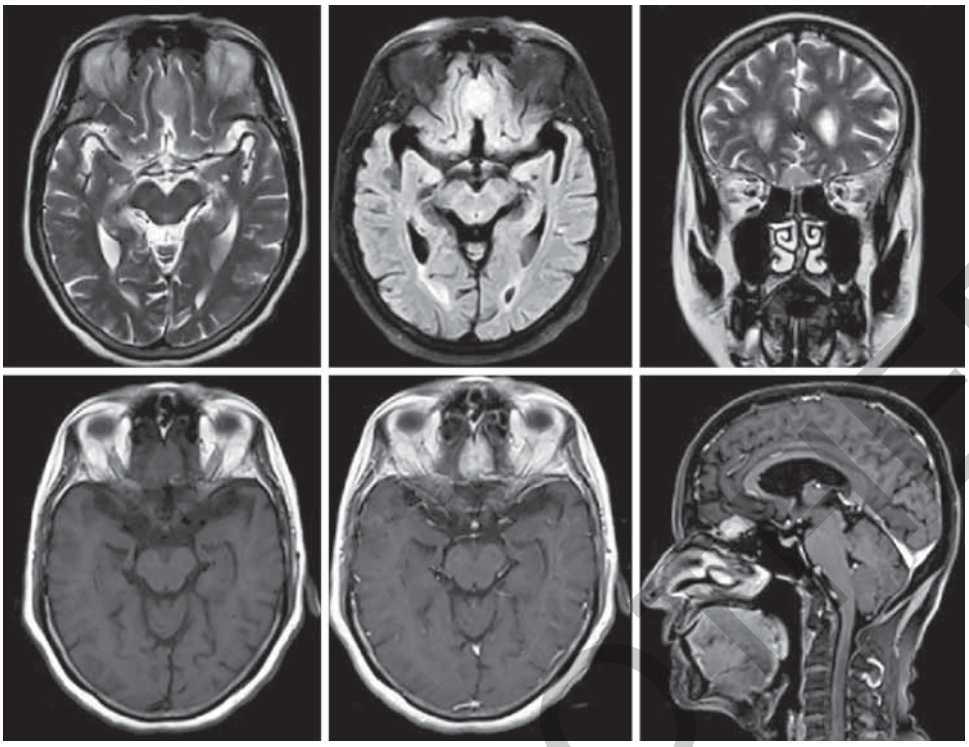


FIG. 4: Axial and coronal T2W images of brain reveal a dural-based lesion in the anterior cranial fossa which is slightly T2 hyperintense. The olfactory groove lesion is fluid-attenuated inversion recovery (FLAIR) hyperintense. The lesion is T1 hypointense with homogeneous intense enhancement on postcontrast T1W study.

Metastasis is the most common cause of posterior fossa mass in adults.

Esthesioneuroblastoma

The main differential diagnoses of esthesioneuroblastoma (ENB) are sinonasal neuroendocrine carcinoma (SNEC) and sinonasal squamous cell carcinoma (SCCa). Sinonasal SCCa commonly involves the maxillary antrum and is relatively less enhancing compared to ENB.

Sinonasal undifferentiated carcinoma (SNUC) is another differential diagnosis. Sinonasal non-Hodgkin lymphoma is hyperdense on CT and shows diffusion restriction on DWI.

Anterior skull base meningioma causes hyperostosis and shows a "sunburst" pattern of vascularity.

TUMORS OF THE MENINGES

Meningomas

The main differential diagnoses of typical meningioma (TM) are atypical or malignant meningioma. Although there are no pathognomonic imaging features that reliably distinguish TM (**Fig. 4**) from these more aggressive variants, TMs are statistically far more common. Malignant meningiomas characteristically show brain parenchymal invasion.

Other differentials include granuloma (TB, sarcoid) and focal idiopathic hypertrophic pachymeningitis.[25,26] Idiopathic hypertrophic pachymeningitis may show bony invasion mimicking an "en plaque" meningioma.

Solitary fibrous tumor/hemangiopericytoma and hemangioma are rare considerations. Hemangiomas are very hyperintense on T2WI, whereas most meningiomas are iso- to mildly hyperintense. Hemangiomas show delayed slow centripetal "filling in" of the mass on dynamic contrast-enhanced MR.

Extramedullary hematopoiesis can mimic "en plaque" solitary or multiple meningiomatosis.

NERVE SHEATH TUMORS

Vestibular Schwannoma

The main differential diagnosis of vestibular schwannoma (VS) is cerebellopontine angle meningioma. Most meningiomas "cap" the internal auditory canal and do not extend deep to the porus. Vestibular schwannomas typically extend into the internal auditory canal and widen the canal.

Facial nerve schwannomas usually have a labyrinthine segment "tail."

Metastases can coat (ensheath) the facial and vestibulocochlear nerves within the internal auditory canal. They are usually bilateral with other lesions present.

Other cerebellopontine angle masses include arachnoid cysts, aneurysms, and epidermoid cysts (ECs).

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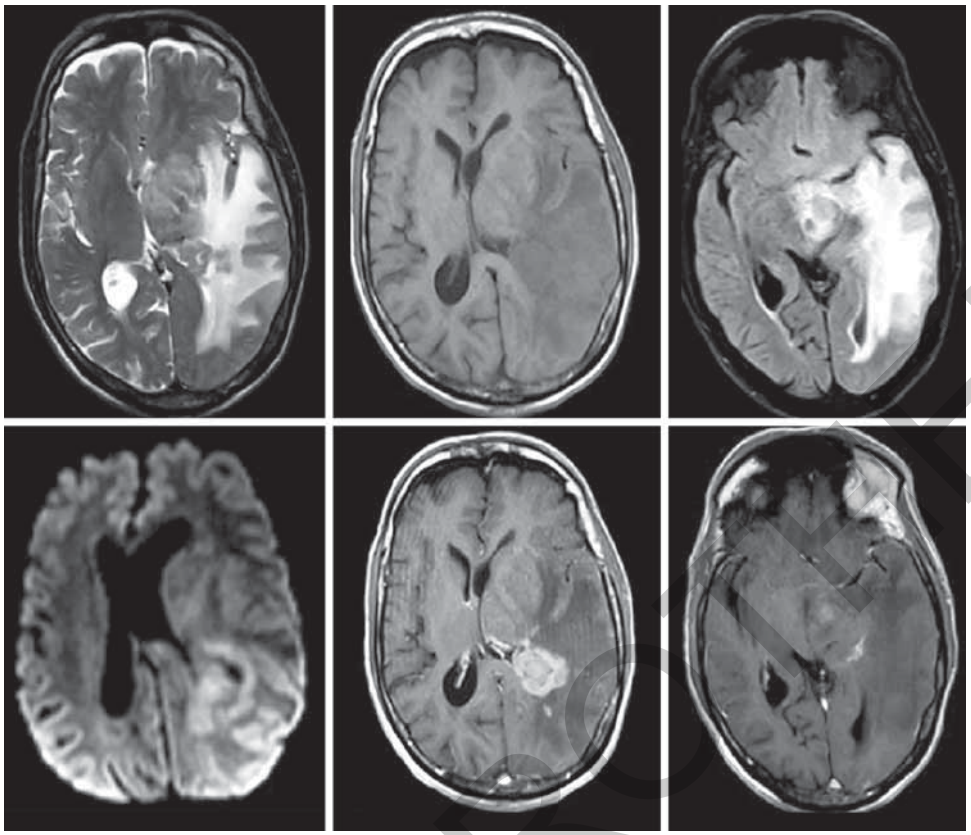


FIG. 5: Axial T2W and fluid-attenuated inversion recovery (FLAIR) sequences of brain reveal lesion involving the left periatrial region, adjacent left basal ganglia, and left half of midbrain with adjacent parenchymal T2/FLAIR hyperintensity. The left periatrial lesion appears hyperintense on diffusion-weighted imaging (DWI). The lesion is T1 hypointense with patchy and nodular enhancement on postcontrast T1W study.

Trigeminal Schwannoma

The appearance of a bi- or a tricompartmental trigeminal schwannoma is distinctive. The main differential diagnoses of a Meckel cave schwannoma include metastasis and meningioma.

LYMPHOMAS AND HISTIOCYTIC TUMORS

Primary CNS Lymphoma

The main differential diagnosis of primary CNS lymphoma is GBM. Although both tumors often cross the corpus callosum, hemorrhage and necrosis are rare in PCNSL.[27,28] Enhancement in immunocompetent patients with PCNSL (**Fig. 5**) is strong and relatively homogeneous.

Metastasis is the second most common differential diagnosis.

Dura-based PCNSLs may mimic meningioma.

In the setting of hematopoietic stem cell or solid organ transplants, post-transplant lymphoproliferative disorder (PTLD) and lymphomatoid granulomatosis may mimic PCNSL.

Langerhans Cell Histiocytosis

Lytic calvarial lesions that may mimic Langerhans cell histiocytosis (LCH) include burr holes and other surgical defects, dermoid and epidermoids, leptomeningeal cysts, and infection.[29] Neuroblastoma can cause osseous metastases in children.

Differentials to be considered in a thickened infundibular stalk are germinoma, neurosarcoid, astrocytoma, and hypophysitis.

SELLAR NEOPLASMS

Pituitary Adenoma

The differential diagnosis of pituitary adenoma varies with size and patient demographics.

Pituitary macroadenoma: The main differential diagnosis of pituitary macroadenoma is pituitary hyperplasia.[30]

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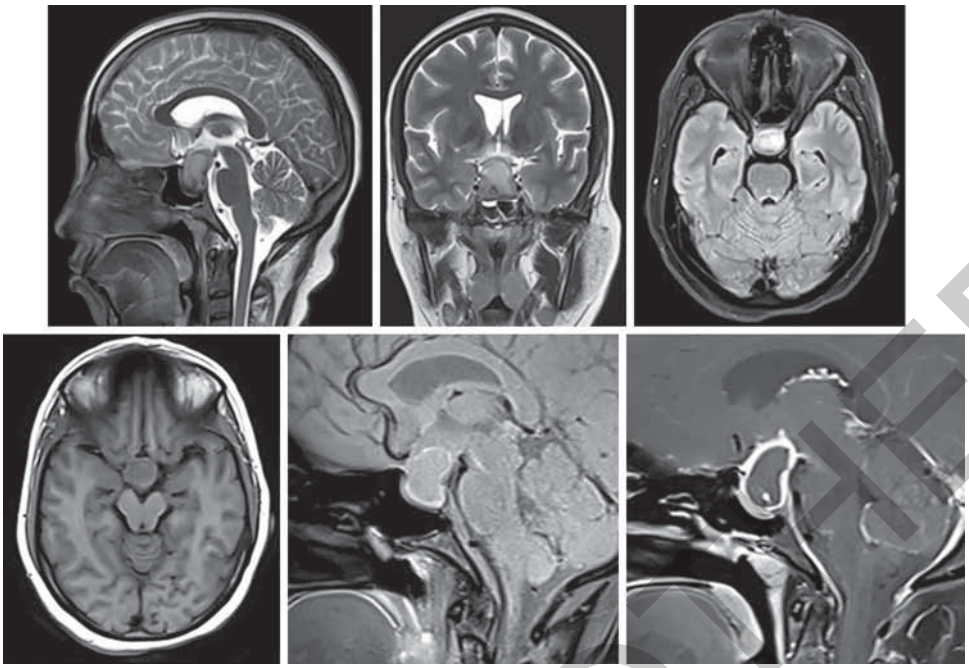


FIG. 6: Axial and coronal T2W images of brain reveal enlarged sella. A well-defined sellar-suprasellar mass lesion is noted. Pituitary gland is not separately identified. Peripheral enhancement is noted on postcontrast T1W study.

Tumors that can resemble pituitary adenoma (**Fig. 6**) include meningioma, metastasis, and craniopharyngioma.

Diaphragm sellae meningioma can be identified separately from the pituitary gland. It shows a dural tail and diffuse intense enhancement.

Metastasis to the pituitary gland and/or stalk usually occurs from lung and breast.

Craniopharyngioma is the most common suprasellar tumor of childhood, and the pituitary gland can usually be identified separate from the mass.

Hypophysitis and aneurysm are non-neoplastic differentials of macroadenoma.

Pituitary microadenoma: Differentials of pituitary microadenoma include incidental non-neoplastic cysts such as pars intermedia cyst or Rathke cleft cyst. Microadenomas enhance whereas cysts do not.

Craniopharyngioma

The main differential diagnosis of craniopharyngioma (CP) is Rathke cleft cyst (RCC). RCCs do not show calcification, appear much less heterogeneous, and do not show nodular enhancement.

Hypothalamic/chiasmatic astrocytoma is generally a solid parenchymal suprasellar T2 hyperintense mass with variable enhancement.

Suprasellar dermoid is another important differential to be considered. A dermoid cyst may show calcification and may be hyperintense on T1WI. An EC is usually off-midline with DWI restriction (**Fig. 7**). Neither of the epidermoid or dermoid cysts enhance.

CONCLUDING REMARKS

Technological advances in neuroimaging and histologic analysis strategies have improved recognition of non-neoplastic diseases presenting as brain tumors. However, no isolated technique achieves 100% specificity and sensitivity, and communication among neuroradiologists, clinicians, neurologists, neurosurgeons, pathologists, and nuclear-medicine specialists is essential to avoid the potential diagnostic pitfalls.

LEARNING POINTS

1. Brain tumors encompass a wide variety of benign as well as malignant pathologies which may be mimicked by some other neoplastic or non-neoplastic pathologies on MRI studies.
2. It is important to know the differential diagnoses of brain tumors and their characteristic imaging features so as to ensure a correct radiological diagnosis.
3. Infections, granulomatous disease, demyelinating disorders, and vascular diseases may sometimes mimic CNS neoplasms.

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Pitfalls in The Diagnosis of Neurological Disorders

Salient Features

- 'To err is human' and doctors, including neurologists, are not immune from making mistakes—mistakes in making a diagnosis or missing the diagnosis and also in providing appropriate therapeutic care to his/her patients, which in most circumstances stem from an inaccurate diagnosis.
- Advances in medical technology have not totally solved the problem of diagnostic errors in medicine. The majority of diagnostic errors are due to cognitive errors by physicians, particularly in synthesizing the available information to identify the correct diagnosis. This is particularly evident in neurological practice even in the current days of sophisticated technological supports which are often not appropriately used to solve a clinical problem or the results provided are misinterpreted. It is, thus, crucial to identify the causes of diagnostic errors and define strategies to improve diagnostic accuracy to achieve a better clinical outcome.
- This is exactly what had been attempted in the present volume. An all-round attempt had been made to discuss the diagnostic strategies to a host of common and uncommon problems encountered in neurological practice, both in a clinic setting and in the setting of an intensive care unit. Such diagnostic processes would be critically evaluated to find lacunae or areas where an average clinician would be apt to make a mistake, and means to avoid making such mistakes would be highlighted.
- A host of such clinical problems have been identified which would be addressed in the light of the issues raised above, by renowned clinicians both from India and abroad. In the process, several clinical cases have been discussed highlighting issues pertaining to arriving at the correct diagnosis and avoiding pitfalls.
- A separate section had been devoted to discuss diagnostic challenges faced by neurologists in critical care units with the help of 15 authentic cases by critical care neurologists from the University of Texas Southwestern Medical Center, USA.
- Many chapters had been followed by Editorial notes and Appendices on some key issues relating to diagnostic pathways, again at times with the help of illustrative case studies.
- The present volume, most certainly, is much different from the standard neurological differential diagnosis texts available in the market and perhaps is the first one of its kind to be published from India.

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