Section

I

Neuroradiology

Case 1

Clinical History

History of 4 episodes of GTCS with speech delay.

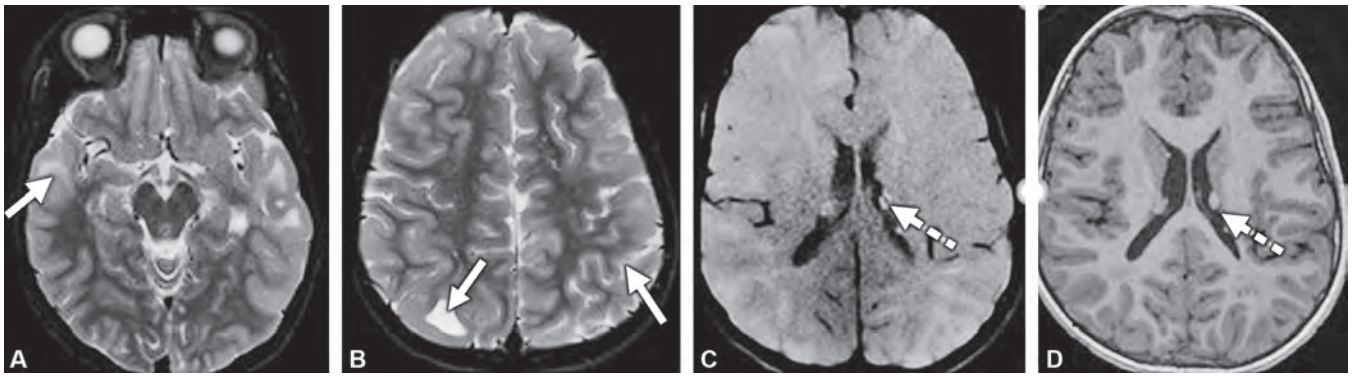

Fig. 1.1 A-D: Axial sections of T2WI (A,B) MRI brain at the level of temporal and parietal lobes show numerous scattered areas of cortical/subcortical hyperintensities in bilateral cerebral hemispheres. Axial sections of T1WI (C,D) show multiple tiny subependymal nodules (dotted arrows)

Diagnosis

Tuberous Sclerosis

Differential Diagnosis

1. Focal cortical dysplasia
2. Subependymoma
3. Medullary veins
4. Enlarged perivascular spaces

Discussion

Tuberous sclerosis is an inherited neurocutaneous disease with an autosomal dominant inheritance pattern characterized by multisystem involvement. It is also known as Bourneville disease. The typical clinical presentation is with seizures, intellectual disability, and facial angiofibroma.

Seizures are the most frequent neurologic complication, occurring in up to 90% of individuals. Additional neurodevelopmental issues include intellectual disability, autism spectrum disorders, ADHD, and sleep disturbances.

Table 1.1: Hallmark central nervous system (CNS) findings in tuberous sclerosis complex (TSC)
• Cortical and subcortical tubers
• White matter heterotopia
• Subependymal nodules
• Subependymal giant cell astrocytomas (SEGAs)

These abnormalities are common and contribute significantly to the morbidity and mortality seen in TSC, affecting over 80% of patients.

Cortical and subcortical tubers result from disrupted cortical development and are seen in approximately 90% of TSC cases. These lesions are broad and have expanded gyrus, and they present with seizures that are nonresponsive to treatment.

On CT, they are initially hypodense. Calcification increases with age. Fifty percent of patients develop more than one calcified tuber.

On MRI, tubers typically exhibit T1 hypointense and T2/FLAIR hyperintense in subcortical regions and may exhibit central cystic changes. Cortical tubers are seen in 90 percent of the individuals of TS patients.

DTI and PET help identify epileptogenic areas for surgical planning.

White matter heterotopia, also found in over 80% of patients, consists of migration abnormalities along the radial glial pathways. Like tubers, it is linked to epilepsy and cognitive deficits. It is best seen on MRI with T2/FLAIR hyperintense radial lines and wedges. CSF-like cysts are seen in deep periventricular white matter.

Subependymal nodules are present in more than 90% of TSC patients and frequently calcify with age.

Such lesions present as calcified lesions along the ventricles, which are appreciated on CT. On MRI, they show variable signal and enhancement, and may exhibit susceptibility artifacts when calcified. Calcification is rare in the first year, and it increases with age. 50% eventually calcify and do not enhance.

SEGAs are benign tumors >10 mm that typically arise from subependymal nodules and occur near the foramen of Monro, leading to obstructive hydrocephalus. These lesions occur in 10–15% of TSC patients.

On CT, SEGA shows mixed density mass at the foramen of Monro with moderate enhancement. On MR, SEGA shows a heterogeneous signal with avid enhancement. Calcifications are more common in SEGA.

Surveillance MRI is recommended every 1–3 years until age 25, with lifelong monitoring if SEGAs are present.

Surgical resection or mTOR inhibitor therapy is indicated when symptomatic or enlarging.

Patients with tuberous sclerosis often exhibit microcephaly, with significantly reduced gray and white matter volumes compared to age-matched controls.

Diffusion tensor imaging (DTI) aids in identifying white matter abnormalities in TSC. It shows high apparent diffusion coefficients in tubers and low fractional anisotropy (FA) values in adjacent white matter, indicating gliosis and hypomyelination.

PET/MRI fusion imaging allows noninvasive identification of epileptogenic tubers by detecting areas of hypometabolism disproportionate to their MRI size, improving surgical outcomes.

α-11C-methyl-l-tryptophan PET can also help localize epileptogenic foci but is not widely available.

Magnetoencephalography (MEG), combined with MRI, provides further spatial localization of epileptic activity and complements PET/MRI data for pre-surgical evaluation.

Early surgical resection of the epileptogenic tuber is recommended to prevent progression to multifocal epilepsy.

Additional Systemic Manifestations Expected

Abdomen: Angiomyolipoma, renal cysts, renal cell carcinoma, retroperitoneal lymphangioma, gastrointestinal polyps, pancreatic neuroendocrine tumors.

Thoracic: Cardiac rhabdomyoma, lymphangioleiomyomatosis, multifocal micronodular pneumocyte hyperplasia, thoracic duct and aortic and pulmonary artery aneurysm.

MSK: Sclerotic bone lesions, hyperostosis of inner table calvaria, scoliosis, bone angiofibroma, retinal angiomas.

Table 1.2: Teaching points	
Differential diagnosis	**Imaging features**
Focal cortical dysplasia	• Transmantle sign seen in FCD – T2 hyperintensity extending from deep white matter to cortical surface. • Calcifications can be seen in cortical tubers.
Subependymoma	• Well-defined non-enhancing benign nodules in periventricular regions, typically <2 cm. • It should be differentiated from SEGA, which shows intense enhancement.
Medullary veins	• White matter radial migration lines in tuberous sclerosis may resemble medullary veins.
Enlarged perivascular spaces	• Cystic cortical tubers resemble enlarged perivascular spaces. • Perivascular spaces are predominantly situated in basal ganglia.

Further Reading

1. Tuberous Sclerosis: Current Update. Mindy X. Wang, Nicole Segaran, Sanjeev Bhalla, Perry J. Pickhardt, Meghan G. Lubner, Venkata S. Katabathina, and Dhakshinamoorthy Ganeshan: RadioGraphics 2021 41:7, 1992–2010.
2. Manoukian SB, Kowal DJ. Comprehensive imaging manifestations of tuberous sclerosis. AJR Am J Roentgenol. 2015;204(5):933–943.
3. B. Kalantari, N. Salamon. Neuroimaging of tuberous sclerosis: spectrum of pathologic findings and frontiers in imaging: Am J Roentgenol, 190 (2008), pp. W304–W309.

Case 2

Clinical History

A 7-year-old girl presents with episodes of headache in the past six months and projectile vomiting for three days.

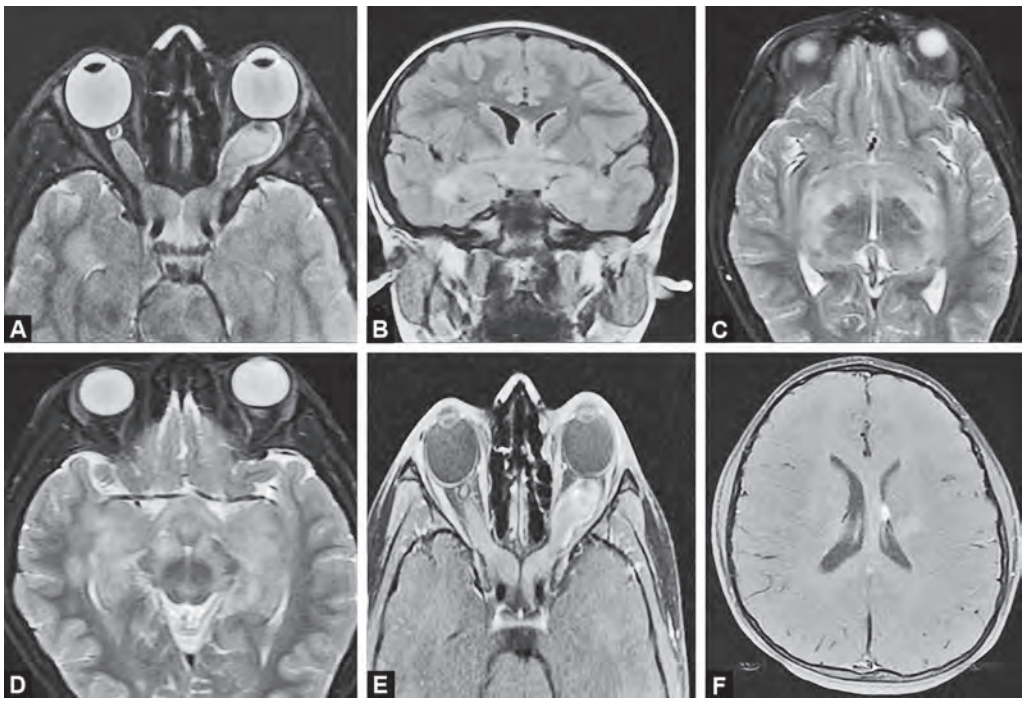

Fig. 1.2: (A) Axial T2 image shows both optic nerves demonstrating fusiform thickening and tortuosity, patchy areas of T2 hyperintensities (left>right), and widening of the optic canals. (B) Coronal FLAIR and (C,D) axial T2 images show confluent hyperintensities involving the optic tracts, anterior commissure, both fornices, inferior aspects of both lentiform nuclei, thalami, hypothalamus, and both medial temporal lobes. (E,F) Axial post contrast fat-suppressed T1 images show relatively homogeneous enhancement of the intraorbital segment of the left optic nerve. A nodular focus of enhancement is seen in the intraventricular aspect of the body of the left lateral ventricle

Diagnosis

Gliomas affect both optic pathways, extending intracranially and involving the hypothalamus. Focal (FASI) and diffuse areas of abnormal signal intensities are also seen. Small enhancing intraventricular lesion within the body of the left lateral ventricle–indicative of neurofibromatosis type I.

Differential Diagnosis

1. Meningioma
2. Optic neuritis
3. Orbital pseudotumor
4. Dural ectasia of optic nerve sheath: The optic nerve is normal.

Discussion

Optic pathway gliomas (OPGs) are the most frequently encountered tumors affecting the optic pathway. These are typically low-grade glial neoplasms originating in the optic system's pre-cortical segments. OPGs can develop in different regions, such as the optic nerves, optic chiasm, optic tracts, radiations, and the hypothalamus. They are most commonly identified in children, particularly within the first decade of life.

OPG is associated with neurofibromatosis type 1 in around 10% to 70% of cases. In adults, these tend to be aggressive. In children, these tend to be benign astrocytomas. Clinical features include vision loss, proptosis, headache, and nystagmus.

OPGs are usually WHO-grade I pilocytic astrocytomas. Tumor cells have spindle-shaped nuclei and appear hair-like. Intracytoplasmic Rosenthal fibers and eosinophilic granular bodies can be seen. Focal calcifications and microcystic areas are also observed within these lesions.

CT Findings

- The optic nerves demonstrate a tubular or spindle-like expansion and kinking.
- Enlargement of optic canal.
- Usually, there are no calcifications.

MRI Findings

- Enlarged optic nerves with altered signals are seen best on T2/FLAIR.
- Optic sheath enlargement.
- It may extend into the optic chiasm, radiations, and hypothalamus.
- Mild-moderate inhomogeneous post-contrast enhancement.
- Often associated with focal areas of abnormal signal intensity (FASIs).

Neurofibromatosis-1 (NF-1)

Two or more of the following criteria:

- Six or more café-au-lait macules.
- The presence of one plexiform neurofibroma or two or more neurofibromas.
- Axillary or inguinal region freckles.
- Optic nerve glioma.
- The presence of at least two iris hamartomas (Lisch nodules).
- A notable bone lesion is evident, which could be sphenoid dysplasia or cortical thinning of long bones and may or may not include pseudarthrosis.
- A first-degree relative (parent, sibling, or offspring) who meets the diagnostic criteria for NF1.

CNS manifestations of NF-1:

- Optic nerve glioma (bilateral ONGs are virtually pathognomonic of NF-1)
- FASI
- Brainstem glioma
- Dural ectasia
- Moyamoya pattern
- Aneurysms
- Buphthalmos
- Cord astrocytoma
- Lateral thoracic meningocele

Finding gliomas affecting both optic nerves strongly suggests a diagnosis of NF-1. Furthermore, understanding that focal areas of abnormal signal intensity (FASIs) on MRI in conjunction with optic pathway involvement should raise suspicion for underlying NF-1.

These lesions usually lead to fusiform expansion of the optic nerve. On MRI, they typically appear iso- to hypointense on T1-weighted images and hyperintense on T2-weighted sequences, with variable contrast enhancement. A thin hypointense rim on T2-weighted images, corresponding to the dura, may also be visible.

NF-1-related optic nerve gliomas are usually quiescent. However, if visual disturbance or an increase in size is documented, treatment needs to be administered.

Treatment includes surgery, chemotherapy (vincristine, carboplatin), and radiotherapy.

Table 1.3: Teaching points

Differential diagnosis	Imaging features
Meningioma	Usually unilateral, the tumor surrounds the nerve and shows a tram-track appearance with calcifications.
Optic neuritis	Shows enhancement of the optic nerve without apparent mass
Orbital pseudotumor	Perineural enhancement and extra ocular muscle involvement
Dural ectasia of optic nerve sheath	Optic nerve is normal

Further Reading

1. Huang M, Patel J, Patel BC. Optic Nerve Glioma [Internet]. PubMed. Treasure Island (FL): StatPearls Publishing; 2020.
2. Fried I, Tabori U, Tihan T, Reginald A, Bouffet E. Optic pathway gliomas: a review. CNS Oncology [Internet]. 2013 Mar 1;2(2):143–59.
3. Optic Pathway Glioma Radiology [Internet]. Pedsoncologyeducation.com. 2025 [cited 2025 Jun 2].

Case 3

Clinical History

A 13-year-old boy with seizure disorder.

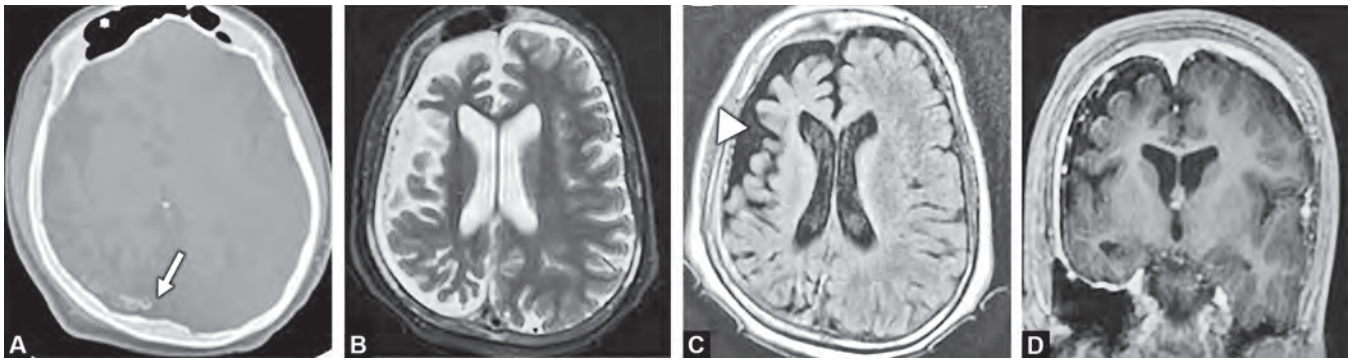

Fig. 1.3: (A) Axial non-contrast CT brain in bone window depicting serpentine calcifications along the gyri (arrow) involving the right cerebral hemisphere, with adjacent calvarial thickening and ipsilateral dilated frontal sinus. (B) Axial T2WI demonstrates right cerebral atrophy. (C) Axial FLAIR shows dirty CSF (arrowhead) in the subarachnoid space with serpentine hyperintensities- ''ivy sign''. (D) Post-contrast axial T1 images show pachymeningeal enhancement

Diagnosis

Sturge–Weber Syndrome

Differential Diagnosis

1. Meningioangiomatosis
2. Dyke Davidoff syndrome
3. Rasmussens encephalitis
4. Hemimegalencephaly

Discussion

Sturge–Weber syndrome, also known as encephalo trigeminal angiomatosis, is a sporadically occurring, non-inherited, non-familial neurocutaneous syndrome. It is commonly located in the parieto-occipital regions and is unilateral in 80% of cases.

The clinical features include unilateral port wine stain (nevus flammeus) along V1, V2 > V3 segments of the trigeminal nerve, seizures developing in the first year of life, migraine-like headaches, hemiparesis, and congenital glaucoma.

Leptomeningeal angiomatosis causes enlarged capillaries and venous channels. Dystrophic laminar cortical calcifications are typical, and cortical venous ischemia and atrophy are also seen.

In plain radiographs, gyriform subcortical calcifications representing the 'tram track sign' may be seen.

On computed tomography, cortical calcifications increase with age, atrophy of the involved cortex, thickening of ipsilateral calvaria, enlargement of ipsilateral sinuses, and choroid plexus.

T1 and T2W MRI shows volume loss with prominent adjacent subarachnoid space. Enlarged veins in subarachnoid space appear as serpentine hyperintensities on FLAIR sequences suggestive of the "Ivy sign".

Calcification 'blooms' on gradient images. The angiomas enhance in the initial phase; as the disease progresses, they burn out and show a lack of enhancement.

As the disease evolves, it results in atrophy and gliosis of the involved segments of the brain due to chronic venous ischemia.

Angiographic imaging demonstrates the dilation of trans-parenchymal veins that link the superficial and deep venous systems.

The commonly associated complications are seizures which are often refractory and worsen with time, focal neurological deficits, and mental retardation are also seen in untreatable cases.

Anti-epileptic drugs commonly treat it, and control is achieved in less than half of all cases. Early lobectomy is done in infants with drug-resistant seizures.

Table 1.4: Teaching points

Differential diagnosis	Imaging features
Meningioangiomatosis	• There is an absence of port-wine stain, and the meningeal angiomas extend into the neighboring brain tissue via the perivascular spaces.
Dyke Davidoff syndrome	• Unilateral cerebral atrophy • Compensatory thickening of the ipsilateral skull vault thickening. • Pneumosinus dilatans ipsilaterally • Ipsilateral shift of falx • Absent port-wine nevus
Rasmussen encephalitis	• Chronic inflammatory unilateral cerebral atrophy • Absent compensatory skull vault thickening and pneumosinus dilatans.
Hemimegalencephaly	• Congenital cortical malformation due to hamartomatous over growth. • Enlarged ipsilateral cerebral hemisphere • Ipsilateral calvarial thickening • Contralateral shift of posterior falx • Associated cortical malformations like pachygyria, polymicrogyria, lissencephaly, heterotopia may be present

Further Reading

1. Comi AM. Update on Sturge-Weber syndrome: diagnosis, treatment, quantitative measures, and controversies. Lymphat Res Biol. 2007;5 (4): 257–64. doi:10.1089/lrb.2007.101.
2. Adams ME, et al . A spectrum of unusual neuroimaging findings in patients with suspected Sturge-Weber syndrome . Am J Neuroradiol. 2009;30:276–281.

Case 4

Clinical History

A 64-year-old male has been experiencing headaches for the past year.

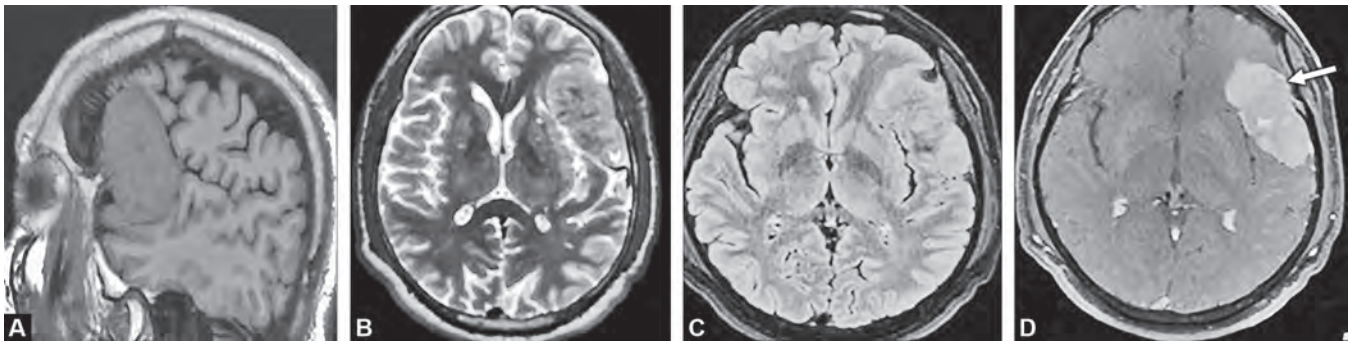

Fig. 1.4: (A) Sagittal T1 W image shows a well-defined lobulated, extra-axial, dural-based lesion predominantly isointense to gray matter seen in the left frontotemporal region with effacement of the adjacent sulci and gyri. (B) On T2 WI and (C) FLAIR, the lesion is isointense to gray matter with CSF cleft sign and displacement of the subarachnoid vessels. The lesion in the (D) post-contrast T1 FS image shows a heterogeneous moderate to intense enhancement

Diagnosis

Left Frontotemporal Meningioma

Differential Diagnosis

1. Dural metastases (e.g. breast and lung cancer).
2. Solitary fibrous tumors of the dura.
3. Dural hemangioma.
4. Extramedullary hematopoiesis.
5. Tb/sarcoid granuloma/Rosai–Dorfman syndrome.
6. IgG4 related disease.
7. Gliosarcoma.

Discussion

Meningiomas are WHO-grade I tumors. These are the most prevalent primary intracranial neoplasm, accounting for about 36%, of which more than 90% occur in the supratentorial region and are usually single lesions detected incidentally on imaging.

Multiple meningiomas are seen in NF-2 and meningiomatosis.

Intracranially, 25% of the tumors are seen in the parasagittal aspect, along the convexity in 20%, followed by the sphenoid wing, olfactory groove, posterior fossa, and the parasellar region. They can also be seen within the orbit.

It is mostly asymptomatic; only 10% are symptomatically present with seizures or headaches, and these are related to tumor site and size. Grossly, two configurations of meningioma are seen, which include the round/globose type of meningioma or the flat sheet-like lesion representing en plaque meningioma.

On plain radiographs, widened grooves of the meningeal arteries may be visible along with hyperostosis of the nearby bone. In some cases, larger lesions can lead to bone erosion, and in rare instances, the mass effect from the expanding tumor may displace calcified structures such as the choroid plexus or pineal gland.

On computed tomography, the lesions are hyperdense compared to normal brain tissue. The tumors may show areas of calcifications within. On administration of contrast, the lesion shows bright, homogeneous enhancement, and in malignant/cystic variants, more heterogeneity and less intense enhancement are seen.

Hyperostosis, occurring in approximately 5% of cases, is more frequently observed in tumors located at the skull base and the anterior cranial fossa. Lytic/destructive regions are noted in higher-grade tumors but should prompt consideration of alternative diagnoses like hemangiopericytoma or metastasis.

On MRI, the lesion is isointense with gray matter, with a positive CSF vascular cleft and enhancing dura representing the dural tail sign. In contrast, administration meningioma shows intense homogeneous enhancement. Except for large and aggressive tumors, there may be heterogeneity due to necrosis or cystic foci with or without vascular flow voids.

MR spectroscopy shows alanine (1.4 ppm) and glutamate–glutamic acid peaks (2.1–2.6 ppm).

Conventional angiography notes sunburst vascularity, dual arterial supply (pial and dural arteries), and prolonged and dense vascular blush.

The observed complications include mass effects on underlying brain parenchyma and malignant degeneration to atypical or anaplastic variants.

Meningiomas are commonly treated by surgical excision, and in case of incomplete resection (base of skull), then external-beam radiation therapy can be used.

Table 1.5: Teaching points

Differential diagnosis	Imaging features
Dural metastases (e.g. breast and lung cancer)	Indistinguishable from meningioma commonly
Solitary fibrous tumors of the dura	More aggressive, often destroying bone with extensive peripheral vascularity. MR spectroscopy–myoinositol peak
Dural hemangioma	Very bright on T2WI. However, most meningiomas are isointense-mildly hyperintense. On dynamic CEMRI shows delayed centripetal filling-in
Extramedullary hematopoiesis	Homogeneously enhancing dural based mass, smooth appearance
Tb/sarcoid granuloma/Rosai–Dorfman syndrome	Meningeal/dural based masses or thickening, substantial enhancement
IgG4 related disease	Dural thickening showing homogeneous contrast enhancement
Gliosarcoma	Heterogeneously enhancing intraparenchymal mass with dural involvement, and is very rare

Further Reading

1. Näslund O, Strand PS, Solheim O, Al Masri M, Rapi O, Thurin E, Jakola AS. Incidence, management, and outcome of incidental meningioma: what has happened in 10 years? J Neurooncol. 2023 Nov;165(2):291-299. doi: 10.1007/s11060-023-04482-5. Epub 2023 Nov 8. PMID: 37938444; PMCID: PMC10689551.
2. Tamrazi B, Shiroishi M, Liu C. Advanced Imaging of Intracranial Meningiomas. Neurosurg Clin N Am. 2016;27(2):137-43. doi:10.1016/j.nec.2015.11.004.

Case 5

Clinical History

A 25-year-old male presenting with on and off headaches for 6 months.

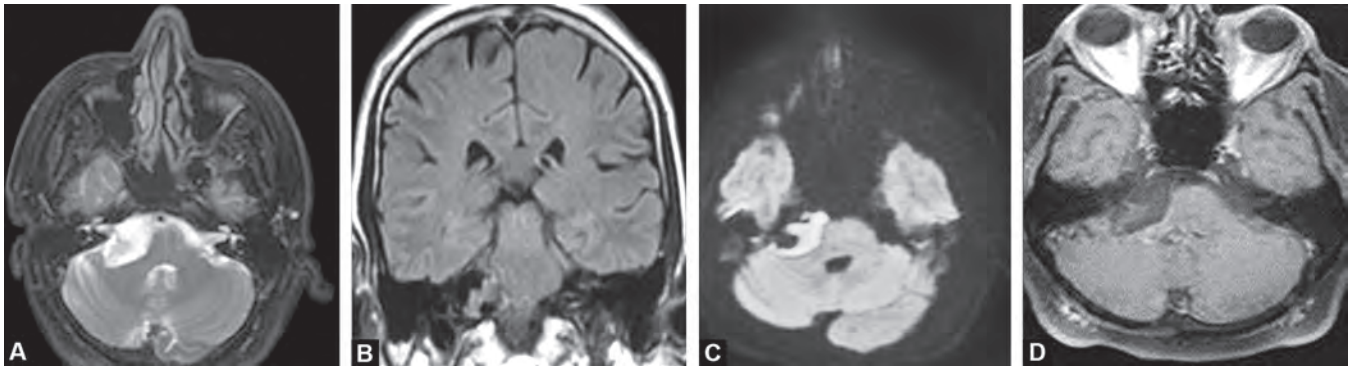

Fig. 1.5: (A) Axial T2WI image shows a lobulated hyperintense extra-axial lesion in the right cerebellopontine angle with incomplete suppression on FLAIR (B), with restricted diffusion on DWI (C). The lesion demonstrates no enhancement on post-contrast T1-weighted imaging (D)

Diagnosis

Epidermoid Cyst

Differential Diagnosis

1. Arachnoid cyst.
2. Parasitic cysts like NCC.
3. Cystic neoplasms like craniopharyngioma.
4. Dermoid cysts.
5. Neuroenteric cyst.

Discussion

Epidermoid cysts can be congenital or acquired. The intracranial lesions are congenital benign ectodermal inclusion cysts due to defects in the neural tube, while the spinal epidermoid cysts are often acquired.

The peak age of incidence is 20–60 years. The lesions are usually slow-growing and are rarely symptomatic.

Epidermoid cysts most commonly occur in the cerebellopontine angle cistern, followed by the fourth ventricle and basal cisterns, which tend to encase surrounding cranial nerves and blood vessels. In rare cases, intradiploic epidermoids can be found within the skull bones. Clinically, they present with headaches, and features of cranial neuropathy (V, VII, and VIII) can occur.

The outer surface of the lesion is shiny, with multiple cauliflower-like excrescences, and filled with waxy/creamy material. The cyst is enclosed by a capsule lined with stratified squamous epithelium and contains keratinous material along with crystalline cholesterol.

The epidermoid on CT is usually hypodense, consistent with CSF, and calcification is seen in around 10–25% of the lesions. No enhancement is seen on contrast administration. White epidermoid cysts can be hyperdense due to high protein content/saponification.

On magnetic resonance imaging, the lesion is isointense to CSF on T1W and T2W sequences. The key imaging features are incomplete suppression on FLAIR images and diffusion restriction on DWI.

Complications include rupture of the cyst and malignant transformation, which is rare.

Commonly treated by resection. However, the insinuating characteristics make them difficult to resect.

Table 1.6: Teaching points

Differential diagnosis	Imaging features
Arachnoid cyst	Complete suppression on FLAIR with no diffusion restriction
Parasitic cysts like NCC	Usually, it is multiple, small, and often contains scolex, with no restriction on DWI
Cystic neoplasms like craniopharyngioma	Cyst wall and nodule enhance
Dermoid cysts	Consists of dermal appendages and fat
Neuroenteric cyst	Do not behave exactly like CSF

Further Reading

1. Chen CY, et al. Intracranial epidermoid cyst with hemorrhage: MR imaging findings. Am J Neuroradiol. 2006;27:427–429. Osborn AG, Preece MT. Intracranial cysts: radiologic–pathologic correlation and imaging approach. Radiology. 2006;239: 650–664.

Case 6

Clinical History

An 8-year-old girl with a history of seizures in the past 1 year.

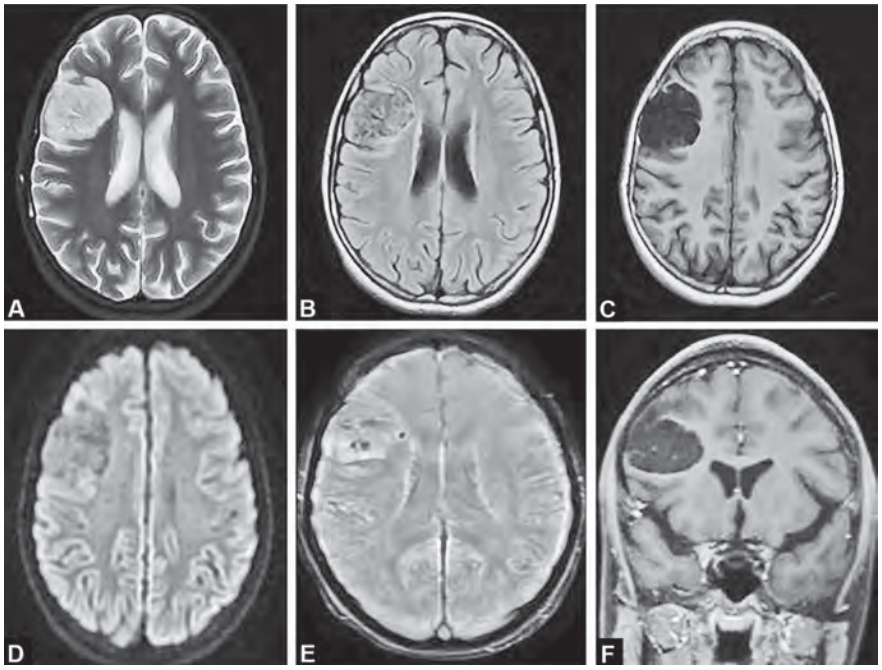

Fig. 1.6 : (A) Axial T2W, (B) T2-FLAIR, (C) T1W, (D) DWI, (E) SWI, and (F) coronal post-contrast T1W images show an intra-axial well-circumscribed T2 hyperintense/T1 hypointense lobulated cortical–subcortical lesion is noted in the right frontal lobe, demonstrating partial signal suppression on FLAIR imaging. Susceptibility-weighted imaging (SWI) reveals a few small hypointense foci within the lesion, possibly representing calcifications. There is no evidence of diffusion restriction or post-contrast enhancement. Additionally, no surrounding edema or significant mass effect was observed

Diagnosis

Dysembryoplastic Neuroepithelial Tumor (DNET)

Differential Diagnosis

1. Ganglioglioma
2. Cystic glioma
3. Focal cortical dysplasia
4. Multinodular vacuolating tumor

Discussion

Dysembryoplastic neuroepithelial tumors (DNETs) are benign, intra-axial lesions typically located in the supratentorial region, primarily involving the cortex or deep gray matter. They exhibit a multinodular growth pattern and most often present with drug-resistant seizures in children and young adults. The temporal lobe is the most frequently affected site, followed by the frontal lobe.

Patients usually present with partial seizures with or without generalization beginning before 20 years of age.

This tumor is a mixed glial-neuronal neoplasm exhibiting a multinodular architecture. It is defined by the presence of 'specific glioneuronal elements' (SGNEs), which are composed of axonal bundles encircled by oligodendrocyte-like cells aligned perpendicular to the cortical surface. Interspersed among these SGNEs are stellate astrocytes and floating neurons, often accompanied by areas of focal cortical dysplasia.

CT can show a superficially located low-density mass in the cortex with or without calvarial scalloping. Around one-third of the lesions show calcifications within.

MRI shows a cystic-appearing lesion with a multiloculated 'bubbly' appearance. T2-FLAIR imaging reveals a peripheral hyperintense rim with central signal suppression. There is no associated mass effect or perilesional edema.

Histologically, the FLAIR hyperintense ring corresponds to loosely arranged glioneuronal elements at the lesion's margin. These lesions typically lack diffusion restriction, contrast enhancement, and surrounding edema.

Differentiating gangliogliomas from DNET, gangliogliomas have a cyst with a strongly enhancing mural nodule, and they frequently demonstrate calcification within the lesion. On PET, they show hypermetabolism, whereas DENT exhibits variably low metabolism.

Antiepileptic medications have been used to treat intractable seizures. Surgical excision is a definitive treatment modality with very low recurrence rates.

Table 1.7: Teaching points

Differential diagnosis	Imaging features
Gangliogliomas	Cyst with a strongly enhancing mural nodule and it frequently demonstrate calcification within the lesion
Cystic glioma	Cyst with enhancement and diffusion restriction.
Multinodular vacuolating tumor	Bubbly lesion in juxta cortical white matter

Further Reading

1. Luzzi S, Elia A, Del Maestro M, Elbabaa SK, Carnevale S, Guerrini F, et al. Dysembryoplastic Neuroepithelial Tumors: What You Need to Know. World Neurosurg. 2019 Jul;127:255–65.
2. Raz E, Kapilamoorthy TR, Gupta AK, Fiorelli M. Case 186: Dysembyoplastic Neuroepithelial Tumor. Radiology. 2012 Oct;265(1):317–20.
3. Phi JH, Kim SH. Dysembryoplastic Neuroepithelial Tumor: A Benign but Complex Tumor of the Cerebral Cortex. Brain Tumor Res Treat. 2022 Jul;10(3):144–150. doi: 10.14791/btrt.2022.0015. PMID: 35929111; PMCID: PMC9353162.

Case 7

Clinical History

Fever, altered sensorium × 1 day.

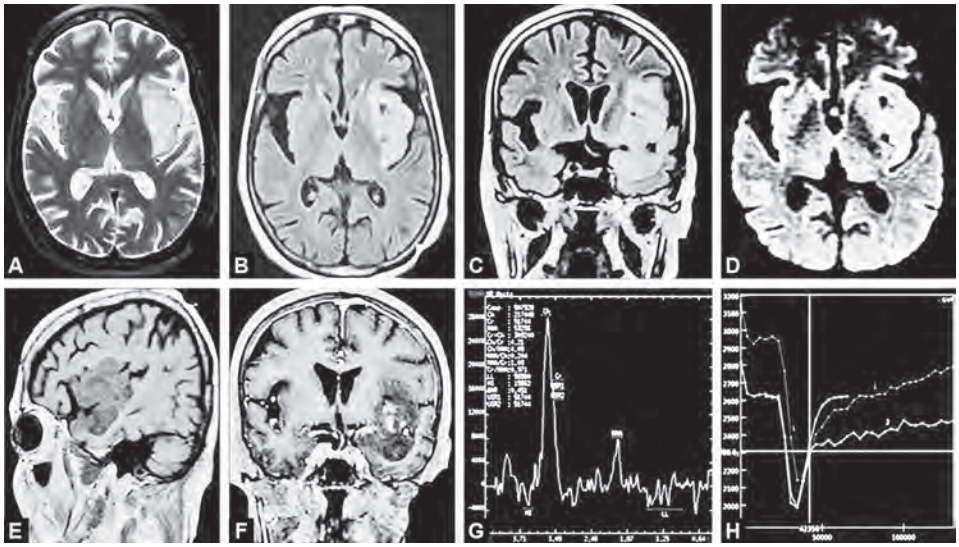

Fig. 1.7: (A) Axial T2WI, (B,C) Axial and coronal T2/FLAIR, (D) Axial diffusion, (E) Sagittal T1 and (F) Coronal post-contrast T1WI of the brain shows a well-defined heterogeneous predominantly T2/FLAIR hyperintense, T1 hypointense solid lesion with few internal cystic spaces is noted involving left external capsule, insular cortex, anterior temporal lobe, and posterior frontal lobe with mild perilesional edema. Patchy areas of diffusion restriction are pointed out within the lesion. Gyral expansion is observed, resulting in a mass effect with mild effacement of the adjacent sulci and the ipsilateral Sylvian fissure. There is no evidence of any areas of calcification. No evidence of T2/FLAIR mismatch. On contrast administration, heterogeneous patchy contrast enhancement was seen. (G) MR spectroscopy shows increased choline levels, decreased NAA levels, and elevated choline creatinine ratio of 4.2. (H) MR perfusion shows increased cerebral blood flow at the lesion

Diagnosis

Adult-type Diffuse Glioma—Glioblastoma IDH Wild-type

Differential Diagnosis

1. Oligodendroglioma
2. Anaplastic astrocytoma
3. Gliosarcoma
4. Tumefactive demyelination

Discussion

Tumors are clinically subdivided into primary and secondary glioblastoma based on whether they are thought to have arisen *de novo* as glioblastoma or progressed from lower-grade glial tumors. Glioblastoma (GBM), classified as a type of adult diffuse glioma, is a primary central nervous system tumor believed to originate from neuroglial stem cells or their progenitors located in the subventricular zone.

According to the fifth edition of the WHO classification of CNS tumors, glioblastoma includes IDH wild-type tumors, and those diagnosed as glioblastoma, IDH-mutant, under the 2016 criteria are now reclassified as astrocytoma, IDH-mutant, CNS WHO grade 4.

Furthermore, the 2021 guidelines specify that the designation of glioblastoma, IDH-wildtype, also applies to IDH-wildtype astrocytic tumors exhibiting specific genetic alterations, namely TERT promoter mutation, EGFR amplification, or +7/ 10 chromosomal copy number changes.

Clinically, the patient can present with seizures, focal neurological deficits, and headaches from elevated intracranial pressure.

Pathological necrosis and microvascular proliferation are the histologic hallmarks of GBM, which distinguishes it from anaplastic astrocytomas. These are WHO-grade IV neoplasms with a high proliferative index.

CT shows a hypodense central mass surrounded by iso- moderately hyperdense rim on NECT. Hemorrhage is common, but calcification is rare. Marked peritumoral edema and mass effect are seen.

MRI reveals an infiltrative mass with ill-defined borders, displaying mixed signal intensity on T1-weighted images. T2/FLAIR sequences show heterogeneous hyperintensities with poorly defined tumor margins and widespread vasogenic edema. Common features include cystic components, hemorrhages at different stages, fluid-debris levels, and flow voids indicating prominent vascular structures.

Diffusion-weighted imaging shows no restricted diffusion. Following contrast administration, an irregular ring of enhancement is seen around a central necrotic core. Nodular, punctate, or patchy foci of enhancement outside the main mass represent macroscopic tumor extension into adjacent structures.

Perfusion images show increased rCBV in the tumor "rind" and increased vascular permeability. Spectroscopic analysis indicates higher choline, lower NAA and mI, and a lipid/lactate signal at 1.33 ppm.

The pattern of spread of glioblastoma is through white matter spread, which includes dissemination along the corpus callosum, fornices, anterior commissure, and corticospinal tract. This can result in brain-to-brain metastasis to the pons, cerebellum, medulla, and spinal cord.

CSF dissemination fills the sulci and cisterns. A frequent pattern of tumor progression involves the widespread encasement of the cranial nerves and the pial surface of the brain. Furthermore, tumor dissemination can extend downwards into the spinal canal, consistent with drop metastases.

Ependymal and subependymal spread can occur to the interior of lateral ventricles. Direct invasion of the tumors can occur through the pia into the dura and arachnoid.

Tumors rarely can invade calvaria, extending into subgaleal soft tissues. Metastasis outside the central nervous system may involve the bone marrow, liver, lungs, and lymph nodes.

Surgical resection can be done for debulking the tumor and reducing raised intracranial pressure.

Based on the HPE analysis, postoperative adjuvant radiotherapy and chemotherapy with temozolomide can be given. The STUPP protocol is the most common treatment. Bevacizumab can be used for immunotherapy.

Table 1.8: Teaching points

Feature	Anaplastic astrocytoma (WHO Grade III)	Gliosarcoma	Tumefactive demyelination
Age group	30–50 years	>50 years	20–40 years
Enhancement	Usually minimal or no enhancement	Peripheral rim enhancement, sometimes heterogeneous	Incomplete ring enhancement (open ring sign)
Margins	Ill-defined but less necrotic	Well-circumscribed or lobulated	Relatively well-defined
Necrosis	Rare or mild	Prominent (sarcomatous areas)	Usually absent or mild
Edema	Moderate	Significant	Disproportionate edema (can be large)
Mass effect	Variable	Prominent	Often mild relative to lesion size
Location	Supratentorial	Similar to GBM, often temporal or frontal	Periventricular/subcortical white matter
Diffusion (DWI/ADC)	Variable	Restricted in solid portions	Mild restriction or none
Spectroscopy	High choline, low NAA	Similar to GBM	Elevated choline, decreased NAA, but often lacks lipid–lactate
Perfusion (rCBV)	Moderately elevated	High rCBV	Mild to moderate increase (lower than GBM)
T2 signal	Hyperintense	Mixed signal, necrosis/ sarcomatous areas variable	Hyperintense with incomplete ring on T2 FLAIR
MR spectroscopy clue	No lipid-lactate unless necrosis	Similar to GBM + fibrous component	Absence of lipid–lactate; increased glutamate/glutamine sometimes
Enhancement pattern clue	Minimal or patchy	Well-defined peripheral ring	Open ring enhancement (toward cortex)
MRS Cho:NAA ratio	2–3	Similar to GBM	Variable, often <2
Clinical clue	Slower course than GBM	GBM variant, may show biphasic histology	Often recent-onset neurologic symptoms post infection/vaccine

Further Reading

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3. Melhem JM, Detsky J, Lim-Fat MJ, Perry JR. Updates in IDH-Wildtype Glioblastoma. Neurotherapeutics. 2022 Oct;19(6): 1705–1723. doi: 10.1007/s13311-022-01251-6. Epub 2022 May 31. PMID: 35641844; PMCID: PMC9154038.

Case 8

Clinical History

A 7-year-old boy with headache since 1 year, vomiting for 10 days.

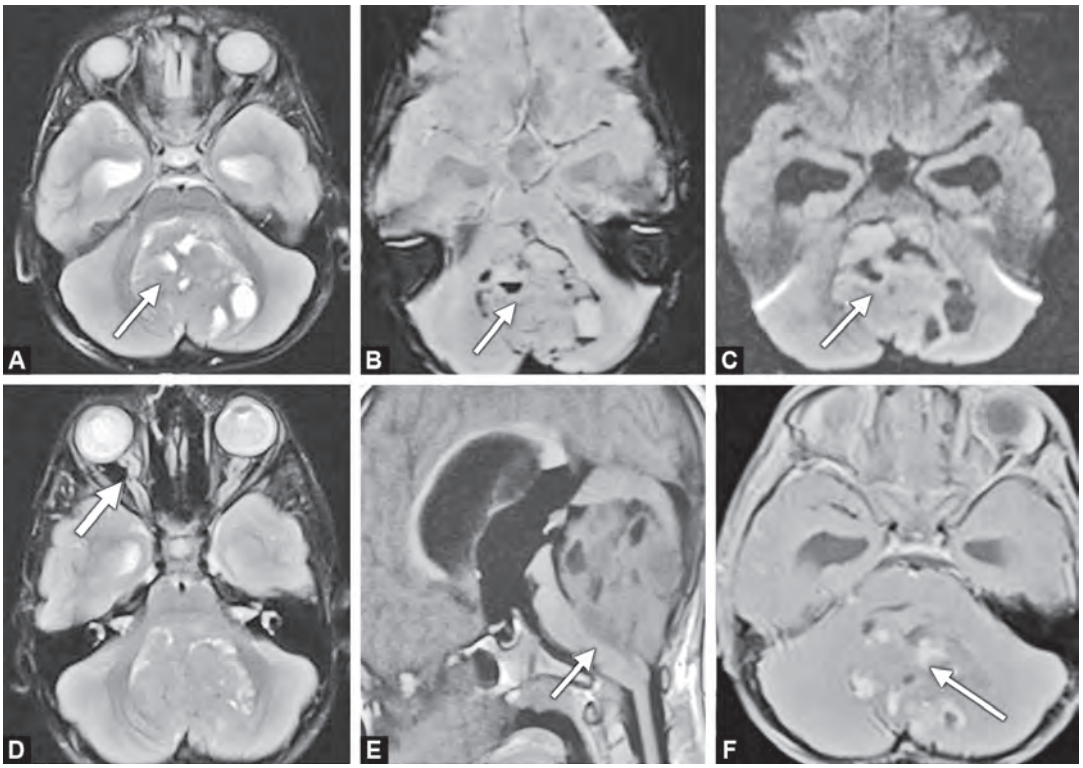

Fig. 1.8: (A) Axial T2W image shows a solid-cystic mass centered within the fourth ventricle, causing a mass effect on the brainstem and cerebellum (arrow) with associated hydrocephalus in the visualized temporal horns. (B) Axial SWI image shows the cystic areas with multifocal blooming and blood levels (arrow). (C) Axial DWI shows restricted diffusion in the solid components of the mass (arrow). (D) In addition, axial T2W shows kinking and tortuousity of both optic nerves (arrow). (E) The sagittal T1W image shows intraventricular predominantly isointense mass causing compression and displacement of the brainstem (arrow) and marked supratentorial hydrocephalus. (F) Axial T1 post-contrast imaging reveals patchy enhancement within the mass (arrow)

Diagnosis

Medulloblastoma

Differential Diagnosis

1. Ependymoma
2. Pilocytic astrocytoma
3. Choroid plexus papilloma
4. ATRT (atypical teratoid rhabdoid tumor)
5. Hemangioblastoma

Discussion

Medulloblastomas are aggressive embryonal tumors arising from primitive multipotent cells of the cerebellum. Most tumors originate in the cerebellum, particularly the vermis, and extend into the fourth ventricle from its roof. In adults, medulloblastomas are typically located laterally in the cerebellar hemispheres. Advanced imaging techniques have also revealed rare extra-axial variants, though their origins are still uncertain. Some speculate they may be associated with migration pathways or the neuroepithelial roof of the fourth ventricle.

It is the second most common malignant tumor in children, accounting for 30–40% of pediatric posterior fossa tumors, with most cases occurring in age <20 yrs with a peak during 3–7 yrs of age. It is more prevalent in males (2:1).

Known syndromic associations are with Cowden syndrome, Gardner syndrome, Gorlin syndrome, and Turcot syndrome.

The 2021 WHO classification recognizes three molecular subtypes:

- WNT-activated
- SHH-activated

- Non-WNT/Non-SHH groups
  - Group 3, Group 4
  - Subgroups 1–8

Clinically, medulloblastomas present with nonspecific symptoms like headache, vomiting, and cerebellar dysfunction, with signs of increased intracranial pressure emerging as the disease advances.

On CT, they appear as hyperdense, well-enhancing masses on plain CT due to high cellularity. Heterogeneous areas can be caused by cyst formation. Calcifications are uncommon.

MRI findings: Heterogeneous areas (cystic areas and necrosis) show restricted diffusion due to high cellularity.

Diffusion-weighted imaging and apparent diffusion coefficient (ADC) analysis assist in tumor grading and preoperative planning. An ADC ratio below 1 is highly sensitive and specific for medulloblastoma.

MRS shows elevated choline and reduced NAA.

Involvement/extension of tumor into CSF spaces can help differentiate from ependymoma.

Perfusion MRI: High relative tumor blood flow; helps differentiate from pilocytic astrocytomas.

Radionuclide imaging is currently limited by low specificity and resolution. However, the development of tumor-specific radiotracers such as 3-deoxy-3-[18F] fluorothymidine (FLT) offers potential, especially in detecting high-grade tumors due to blood-brain barrier disruption.

PET/CT or PET/MRI fusion enhances diagnostic accuracy, particularly in differentiating tumor recurrence from post-therapy changes.

Dynamic susceptibility contrast imaging reveals increased permeability in medulloblastomas, especially desmoplastic types, and helps distinguish them from other posterior fossa tumors.

Medulloblastomas also show the highest relative tumor blood flow among pediatric brain tumors, aiding differentiation from pilocytic astrocytomas and ependymomas.

Table 1.9: The prognosis depends on the location of the tumor

Location	Prognosis
Cerebellar peduncle/Foramen of Luschka	WNT type–thus, best prognosis
Cerebellar hemispheres	SHH type–thus, intermediate prognosis.
Midline cerebellum	Groups 3 and 4 (group 3 – worst prognosis). Groups 3 and 4 show Taurine peak and high Creatinine levels
	Other poor prognostic factors: Age <3 years at diagnosis. Residual tumor >1.5 cm^3 post-surgery. Presence of CSF metastases at diagnosis.

Management is centered on surgical excision, radiotherapy and chemotherapy. However, prognosis is strongly influenced by molecular subtype.

Table 1.10: Teaching points

Feature	Ependymoma	Pilocytic astrocytoma	Choroid plexus papilloma	ATRT	Hemangioblastoma
Typical age group	Children (posterior fossa), adults (spinal)	Children and young adults	Infants and young children	<3 years	Adults (30–60 yrs), VHL in younger
Location	4th ventricle, spinal canal	Cerebellum, optic pathway, brainstem	Lateral ventricles (children), 4th ventricle (adults)	Posterior fossa, supratentorial, spinal	Cerebellum (most common), spinal cord
T1 signal	Iso- to hypo–intense	Hypointense	Isointense	Iso- to hypo–intense	Iso- to hypo–intense
T2 signal	Hyperintense, heterogeneous	Hyperintense with cyst	Hyperintense	Heterogeneous	Hyperintense (solid & cystic)

(Contd...)

Table 1.10: Teaching points *(Contd...)*

Feature	Ependymoma	Pilocytic astrocytoma	Choroid plexus papilloma	ATRT	Hemangioblastoma
Enhancement	Heterogeneous, moderate to intense	Intense mural nodule, cyst wall non-enhancing	Intense, homogeneous	Heterogeneous, variable	Intense enhancement of mural nodule
Calcification	Common	Rare	Rare	May be present	Rare
Cystic component	Possible	Common (cyst + mural nodule)	May be present	Often present	Common (cyst + mural nodule)
Diffusion restriction	Mild to moderate	No significant restriction	No restriction	Marked restriction	None
Flow voids/ vascularity	Minimal	Not prominent	Highly vascular	May be present	Prominent flow voids
Hemorrhage	Occasional	Rare	Rare	Common	Rare
Hydrocephalus	Common	Less common	Very common	Frequent	Rare
Spectroscopy	↑ Choline, ↓ NAA	↑ Choline, ↓ NAA	↑ Choline, ± Lactate	↑ Choline, lipid–lactate	High lipids, vascularity
Associated syndrome	NF2 (spinal)	NF1 (optic)	None	INI1 (SMARCB1 loss)	VHL syndrome
Radiological clue	Extends through foramina, plastic	Cyst + enhancing mural nodule	Cauliflower-like fronds	Restricted diffusion + hemorrhage	Enhancing nodule + flow voids

Further Reading

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2. Yeom KW, Mobley BC, Lober RM et al. Distinctive MRI features of pediatric medulloblastoma subtypes: AJR Am J Roentgenol 200:895–903.

Case 9

Clinical History

An 11-year-old male with focal seizures with decreased visual acuity.

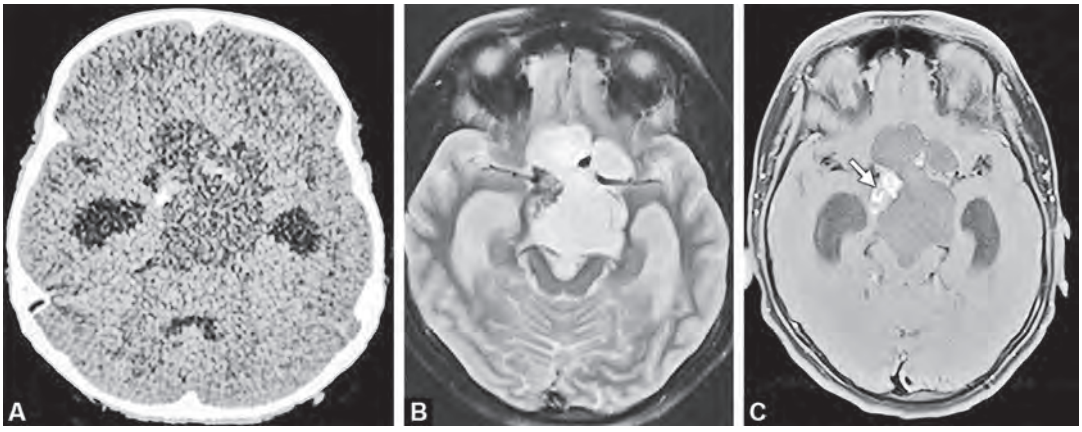

Fig. 1.9: (A) Non-contrast axial CT image demonstrates a well-defined predominantly cystic suprasellar lesion with few calcific foci and associated hydrocephalus. (B) Axial T2W image demonstrates a well-defined multilobulated predominantly cystic lesion with peripheral hypointense solid component in the suprasellar region. (C) Post-contrast T1 FS image demonstrates enhancing solid component with subtle enhancement of the cyst wall along the left anterior aspect of the cyst (arrow)

Diagnosis

Craniopharyngioma

Differential Diagnosis

1. Rathke cleft cyst.
2. Pituitary macroadenoma (with cystic degeneration or necrosis)
3. Pituitary apoplexy.
4. Intracranial teratoma.
5. Epidermoid cyst.

Discussion

Craniopharyngiomas are rare tumors, with an incidence of about 0.13 per 100,000 person-years. It originates from rathke's pouch remnants. It is bimodal in distribution, primarily between ages 5 to 15 years and > 50 years.

It is usually suprasellar with presence or absence of a sellar component. It has two types namely adamantinomatous and papillary type.

Adamantinomatous type (ACP) are composed of multiple cysts, a squamous epithelium with wet keratin and cholesterol-rich 'machinery' oil fluid. It is characterized by the rule of 90: 90% cystic, 90% calcify, 90% enhance.

Papillary type (PCP) are predominantly characterized by solid component however cystic component can occur in adults.

First described by Jakob Erdheim as "hypophyseal duct tumors," craniopharyngiomas are believed to develop via two main theories:

1. **Embryogenetic theory:** Suggests ACPs originate from remnants of Rathke's pouch, an embryonic structure derived from the primitive mouth (stomodeum), explaining their odontogenic histologic features and potential to arise along the entire craniopharyngeal tract.
2. **Metaplastic theory:** Proposes that PCPs arise from squamous metaplasia of oral ectodermal tissue in the suprasellar region.

Children often present with symptoms like headache, visual disturbance, vomiting, lethargy, polyuria, or endocrine abnormalities. Behavioral and cognitive changes may also occur. Adults show similar signs but more commonly present with cognitive deficits.

Table 1.11: Imaging features

Feature	Adamantinomatous type	Papillary type
Age	Mostly children (5–15 yrs)	Mostly adults (>50 yrs)
CT	Cystic dominant, calcification common (90%), enhances on contrast administration	Solid dominant, calcification rare, enhances on contrast administration
MRI T1	Variable (high if proteinaceous cyst)	Iso- to slightly hyperintense solid parts
MRI T2/FLAIR	Cystic parts hyperintense	Solid parts hyperintense
Post-contrast MRI	Peripheral/nodular enhancement	Homogeneous or mild enhancement
Spectroscopy	Lipid–lactate peak (from cystic debris)	Less commonly notable lipid–lactate peak
Cyst content	"Machine oil" (protein-rich, cholesterol-rich fluid)	Less thick cysts (if present)
Calcification	Very common (chunky)	Rare

Complications include malignant transformation to squamous cell carcinoma, which is rare. Total resection is curative in the majority of the cases. The rate of recurrence is ~20–30% at 10 years.

Table 1.12: Teaching points

Differential diagnosis	Imaging features
Rathke cleft cyst	Mostly intrasellar lesion with less heterogeneous appearance than craniopharyngioma. No calcification or nodular enhancement
Pituitary macroadenoma (with cystic degeneration/necrosis)	Primarily intrasellar epicentre but secondary suprasellar extension may occur. It is characterized by the enlargement of pituitary fossa. Calcification is usually absent
Pituitary apoplexy	Presents with acute symptoms such as sudden headache, vision loss. Hemorrhage or infarction can occur within a pituitary adenoma
Intracranial teratoma	Rare in the sellar region but highly important to recognize. Characterized by the presence of fat. Calcification and cystic parts may be present
Epidermoid cyst	Usually in the off-midline location with diffusion restriction on MRI and mimic arachnoid cysts but distinguished by DWI

Further Reading

1. Shih RY, Schroeder JW, Koeller KK. Primary Tumors of the Pituitary Gland: Radiologic-Pathologic Correlation. RadioGraphics. 2021 Nov;41(7):2029–46.
2. Lee IH, Zan E, Bell WR, Burger PC, Sung H, Yousem DM. Craniopharyngiomas : Radiological Differentiation of Two Types. Journal of Korean Neurosurgical Society [Internet]. 2016 Sep 1 [cited 2021 Aug 13];59(5):466–70.

Case 10

Clinical History

A 35-year-old female with headache and giddiness for 2 years.

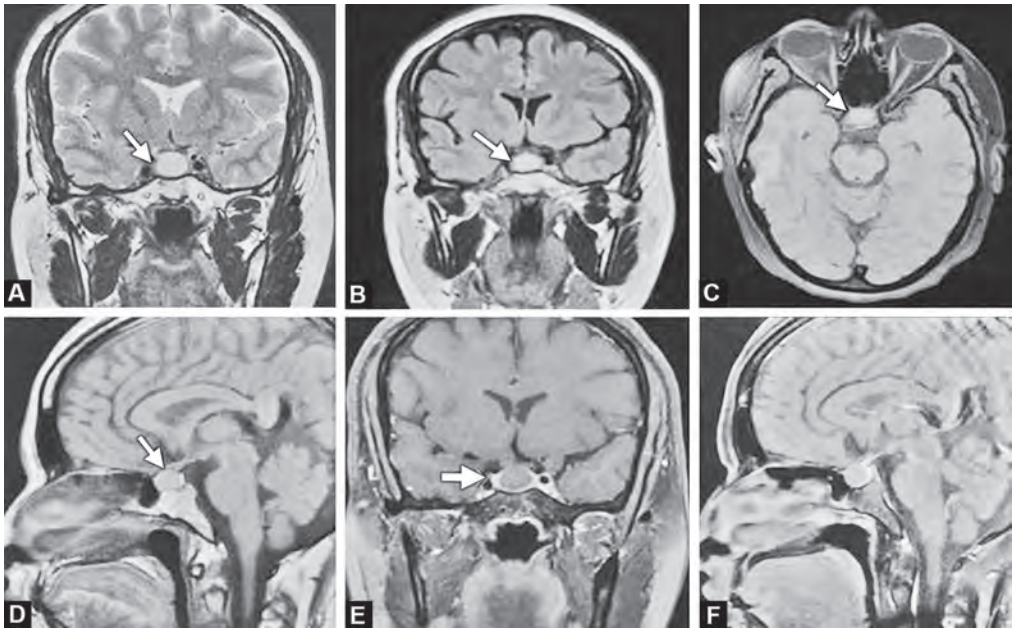

Fig. 1.10: (A) Coronal T2WI, (B) Coronal T2 FLAIR, (C) Axial FLAIR, (D,E,F) Sagittal T1, coronal and Sagittal post-contrast T1WI of brain shows a well-defined T2/FLAIR hyperintense, T1 isointense lesion expanding the sella (thin arrow). The lesion shows no post-contrast enhancement. The normal pituitary gland is noted to be compressed and displaced peripherally by this lesion (thick white arrow in Fig. E)

Diagnosis

Rathke Cleft Cyst

Differential Diagnosis

1. Cystic pituitary adenoma
2. Craniopharyngioma

Discussion

Rathke's cleft cyst supposedly arises from the failure of obliteration of the Rathke's cleft, which is part of Rathke's pouch, a focal outpouching from the superior aspect of the primitive oral cavity in the first month of gestation.

These cysts are mostly asymptomatic. When present, symptoms include headache, hypopituitarism, and visual disturbance. Rarely causes galactorrhea, visual disturbances, from enlargement and compression of the pituitary, pituitary stalk, optic chiasm, and hypothalamus.

Rathke's cyst is usually lined by a layer of columnar epithelium containing ciliated and goblet cells, resembling the embryonic Rathke's cleft. Squamous metaplasia may occasionally be observed within the cyst.

On imaging, the detection of an intracystic nodule is crucial. It usually appears hyperintense on T1 and hypointense on T2, with contrasting cyst fluid and no post-contrast enhancement, which helps in the preoperative diagnosis of Rathke's cleft cyst.

The differentiation of this entity from cystic pituitary adenoma and craniopharyngioma is essential as it impacts management.

Rathke's cleft cyst typically lacks any enhancing component. It may be encased by normal pituitary tissue, which demonstrates normal early enhancement on dynamic contrast imaging. In contrast, cystic pituitary adenomas exhibit an enhancing wall, while craniopharyngiomas often present with an enhancing mural nodule, with or without calcification.

Complications are rare. However, larger lesions may cause compression of adjacent structures like the pituitary, pituitary stalk, optic chiasm, and hypothalamus.

Small asymptomatic cysts are usually kept on follow-up. Cyst aspiration and partial removal of the cyst suffice for small symptomatic cysts. Post-procedure recurrences are attributed to squamous metaplasia.

Table 1.13: Teaching points

Rathke cleft cyst	Cystic pituitary adenoma	Craniopharyngioma
• Centered in sella	• Centered in sella with suprasellar extension	• Centered in suprasellar region
• Displaces pituitary	• Enhancing wall/solid component	• Enhancing solid component
• Absent post-contrast enhancement	• Can show fluid-fluid levels or hemorrhagic debris	• Tend to calcify
• Hemorrhage/calcification is rare	• Absence of intracystic nodule	
• Presence of non-enhancing intracystic nodule		

Further Reading

1. Byun WM, Kim OL, Kim D sug. MR Imaging Findings of Rathke's Cleft Cysts: Significance of Intracystic Nodules. American Journal of Neuroradiology [Internet]. 2000 Mar [cited 2025 May 8];21(3):485–8.
2. Brou C, Idil Gunes Tatar. Different Faces of Rathke's Cleft Cyst. Journal of the Belgian Society of Radiology. 2023 Jan 1;107(1).
3. Okamoto S, Handa H, Yamashita J etal. Computed tomography in intra- and suprasellar epithelial cysts (symptomatic Rathke cleft cysts). AJNR Am J Neuroradiol. 6 (4): 515-9.

Case 11

Clinical History

A 28-year-old female with c/o paresthesia of bilateral limbs.

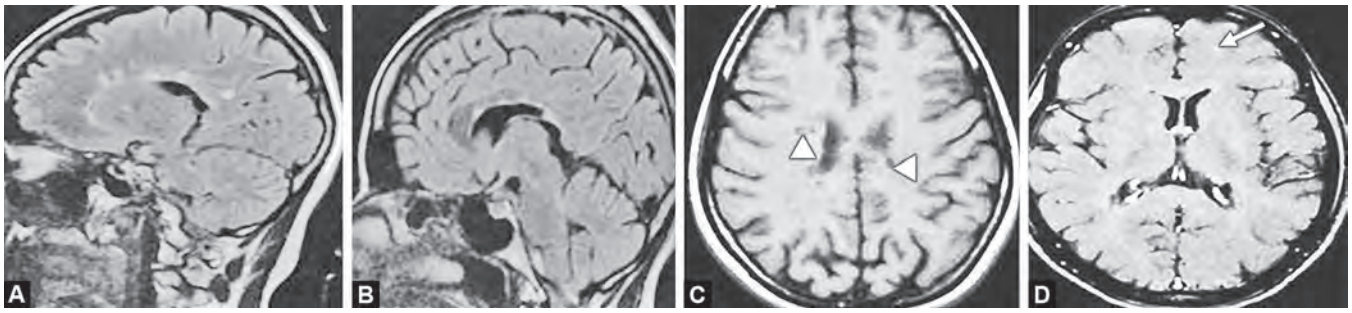

Fig. 1.11: (A, B) Sagittal FLAIR image shows multiple hyperintense round/ovoid lesions that radiate from the lateral ventricles known as "Dawson's fingers" and a triangular shaped plaque with alternating linear hyperintensity in the ependyma known as "Ependymal dot dash sign. (C) T1WI showing black holes (arrowheads). (D) The T1 post-contrast image shows a subtle enhancing lesion (arrow) in the left frontal lobe, suggesting an active demyelinating plaque

Diagnosis

Multiple Sclerosis

Differential Diagnosis

Multifocal T2/FLAIR hyperintensities:

1. Acute disseminated encephalomyelitis
2. Lymes disease.
3. Vasculitis.
4. Susac syndrome.

Mass-like (tumefactive) lesions:

1. Neoplasm—Glioblastoma multiforme, metastasis
2. Progressive multifocal leukoencephalopathy
3. Medication related—Enbrel

Discussion

Multiple sclerosis is an autoimmune inflammatory condition marked by demyelination and axonal damage. Multiple central nervous system lesions occur at different times and locations, and no single diagnostic test can conclusively confirm the disease.

It is common in young and middle-aged adults with a female preponderance.

Clinically presents with weakness, paresthesia, vertigo, visual and urinary disturbances.

Unpredictable relapsing and remitting symptoms can present with optic neuritis (50% eventually develop MS).

Table 1.14: The clinical phenotypes of multiple sclerosis	
Relapsing–remitting	Periods of new and worsening neurological symptoms with periods of remission.
Secondary progressive	The relapsing–remitting type becomes secondary progressive as the intervening remission period vanishes.
Primary progressive	Steady decline in neurological symptoms from the onset.
Atypical presentations	Tumefactive, Marburg type (fulminant course), Schilder type (diffuse cerebral sclerosis), Balo concentric sclerosis

Grossly, the plaques have relatively sharp borders, with perivascular chronic inflammation and reactive astrocytes. Long-standing plaques are characterized by glial scarring and no active inflammation.

On imaging the typical lesions of multiple sclerosis are located in the juxtacortical and cortical region of brain, brainstem, corpus callosum and spinal cord. Temporal lobe is most commonly involved.

On computed tomography, patchy or confluent hypodensities with mild patchy/ring enhancement can be noted.

On magnetic resonance imaging, the following features are observed:

Table 1.15: MRI imaging features of multiple sclerosis	
T1	• **T1 black holes:** Hypointense plaques • **Venus necklace:** Multiple hypointense lesions in the calloso-septal interface. • Hyperintense rim surrounding the black holes—"beveled/lesion within lesion appearance". • Mild cerebral and corpus callosal atrophy.
T2	• Hyperintense lesions with surrounding edema in active state.
FLAIR	• **Dawson fingers:** Multiple ovoid or round hyperintense lesions radiating from the lateral ventricles, encircling the medullary veins. • **Ependymal dot-dash sign:** Triangular-shaped plaques alternating with linear hyperintensity along the ependyma.
SWI	• **Central vein sign:** A hypointense focus located at the center of a surrounding hyperintense lesion.
DWI/ADC	• Restricted diffusion in active lesions.
On contrast administration	• Active lesions enhance with "Horseshoe" pattern of enhancement: open ring enhancement with non-enhancing segment facing cortex.

Spinal cord involvement is seen more than 80% of the cases of multiple sclerosis. The lesions are typically peripheral, multifocal, involves a short segment (less than two vertebral segments in length) and enhancement is not common.

2017 McDonald Criteria for Dissemination in Space and Time

Dissemination in space (DIS): More than or equal to one T2 lesion in at least two out of four areas of the CNS: Juxtacortical/ intracortical, periventricular, infratentorial, spinal cord.

Dissemination in time (DIT): A new T2 and or gadolinium-enhancing lesion on follow up MRI, with reference to a baseline scan, irrespective of the timing of the baseline MRI OR

Simultaneous presence of asymptomatic gadolinium-enhancing and non-enhancing lesions at any time.

**In elderly patients and patients with cardiovascular risks factors, it is better to look for atleast three periventricular lesions.

Associated complications are:

- Progressive neurological impairment,
- Progressive multifocal leukoencephalopathy in patients treated with natalizumab,
- Primary CNS lymphoma appears to arise from previously identified demyelinating lesions,
- >2-times increased risk of stroke, possibly related to chronic inflammatory vasculopathy.

Management includes disease-modifying therapies. Early diagnosis helps in early treatment and improves the overall prognosis.

Table 1.16: Teaching points

Differential diagnosis	Imaging features
Multifocal T2/FLAIR hyperintensities	
ADEM	Usually history of viral prodrome/recent vaccination
Lymes disease	Cranial nerves are more commonly involved
Vasculitis	Preferentially basal ganglia involved with sparing of callososeptal interface
Susac syndrome	Involves the middle of corpus callosum
Mass like (tumefactive) lesions	
Neoplasm—Glioblastoma multiforme, metastasis	GBM—large irregular lesions with heterogeneous contrast enhancement, central necrosis and vasogenic edema. Metastasis—Highly variable appearance, uniform/ punctate/ ring enhancing pattern. May or may not be associated with hemorrhagic component.
PML	Bilateral, asymmetric, periventricular and sub cortical involvement. More common in the occipital and parietal lobes.
Medication related—Enbrel	History of medication intake, new onset or exacerbation of demyelinating diseases (multiple sclerosis, optic neuritis, transverse myelitis).

Further Reading

1. Thompson AJ, Banwell BL, Barkhof F, Carroll WM, Coetzee T, Comi G, Correale J, Fazekas F, Filippi M, Freedman MS, Fujihara K, Galetta SL, Hartung HP, Kappos L, Lublin FD, Marrie RA, Miller AE, Miller DH, Montalban X, Mowry EM, Sorensen PS, Tintoré M, Traboulsee AL, Trojano M, Uitdehaag BMJ, Vukusic S, Waubant E, Weinshenker BG, Reingold SC, Cohen JA. Diagnosis of multiple sclerosis: 2017 revisions of the McDonald criteria. Lancet Neurol. 2018 Feb;17(2):162–173. doi: 10.1016/S1474-4422(17)30470-2. Epub 2017 Dec 21. PMID: 29275977.
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