

MRI Evaluation of Intracranial Cystic Lesions

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ABSTRACT

Background: Various intracranial cystic lesions were evaluated and their imaging appearances have been described, classified the cystic lesions into five groups like post infective/ inflammatory, developmental/ congenital, post traumatic/vascular, neoplastic etiology and normal variants. Histopathological examinations of lesions were studied in few operated cases.

Material and Methods: 50 patients referred to radiology department suspected to have intracranial signs and symptoms were studied for a period of two years from January 2013 to December 2014. Patient age group ranging from 5-65 years were subjected to MRI scan using standard intracranial protocols.

Results: Out of 50 cases, 24 were post infective/ inflammatory, 16 were developmental/ congenital, 4 were post traumatic/ vascular, 4 were neoplastic etiology and 2 were normal variants.

Key words: Intracranial Cystic Lesions, Magnetic Resonance Imaging (MRI), Diffusion Weighted Imaging (DWI).

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INTRODUCTION

A true cyst is an abnormal closed epithelium-lined sac in the body that contains a liquid or semisolid substance.¹ A pseudocyst is an abnormal or dilated cavity resembling a true cyst but not lined with epithelium also called as false cyst.¹

INTRACRANIAL CYSTIC LESIONS^{2,6}

1. Normal Variants

CAVUM SEPTI PELLUCIDI: The septum pellucidum consists of two thin sheets of white matter surrounded by gray matter. The persistence of this septum after birth is called cavum septi pellucidi.

CAVUM VELI INTERPOSITI: An anatomic variant that may appear as a cyst in the pineal region observed in neonatal ultrasound studies, the internal cerebral veins flows inferiorly.

CAVUM VERGAE: Consists of the permanence of the liquid cavity between the two layers of the septum pellucidum extending posteriorly between the fornices.

Enlarged tumefactive perivascular (Virchow-Robin) spaces are pial-lined, CSF-filled spaces that accompany penetrating arteries supplying the brain parenchyma. Perivascular spaces are normally less than 5 mm in diameter. Tumefactive perivascular spaces are much larger than this and have an expanding appearance. These enlarged spaces usually occur in the basal ganglia and white matter and, less commonly, in the midbrain.

2. Developmental/Congenital Lesions

CHOROID PLEXUS CYSTS: Choroid plexus cysts (CPCs) are nonneoplastic epithelial-lined cysts of the choroid plexus. They are the most common of all intracranial neuroepithelial cysts. Most are bilateral and located in the lateral ventricular atria. Most are small, measuring 2–8 mm in diameter. Cysts greater than 2 cm are rare.

Imaging: CPCs are iso- to slightly hyperattenuated on nonenhanced CT scans compared with CSF. Peripheral calcification is common. The cysts show enhancement that varies from none to striking. Signal intensity on MR images is variable. Most are iso- or hyperintense on T1-weighted MR images compared with CSF and show rim or nodular contrast enhancement. CPCs are usually hyperintense to CSF on T2-weighted images. The majority do not become completely hypointense (suppress) on fluid-attenuated inversion-recovery (FLAIR) images and remain slightly or moderately hyperintense to CSF. Two-thirds show restriction (high signal intensity) on diffusion-weighted images.⁷ Real-time prenatal ultrasonographic (US) findings demonstrate a cyst greater than 2 mm surrounded by echogenic choroid.

EPENDYMAL CYSTS: Ependymal cysts are rare, benign, ependymal-lined cysts of the lateral ventricle or juxtaventricular region of the temporoparietal region and frontal lobe. They can also occur in other sites like subarachnoid spaces, brainstem,

cerebellum spinal cord, and very rarely in the cerebellopontine angle.^{8,9} Most are incidental, but symptomatic cysts may manifest with headache, seizure, and/or obstructive hydrocephalus.¹⁰ Ependymal cysts are thought to arise from sequestration of developing neuroectoderm during embryogenesis. They are thin

walled and filled with clear serous fluid secreted from ependymal cells. Columnar cells, with or without cilia, line ependymal cysts. They have vesicular nuclei and eosinophilic cytoplasm.

Imaging: The best diagnostic clue is a nonenhancing thin-walled CSF-containing cyst of the lateral ventricle.¹⁰

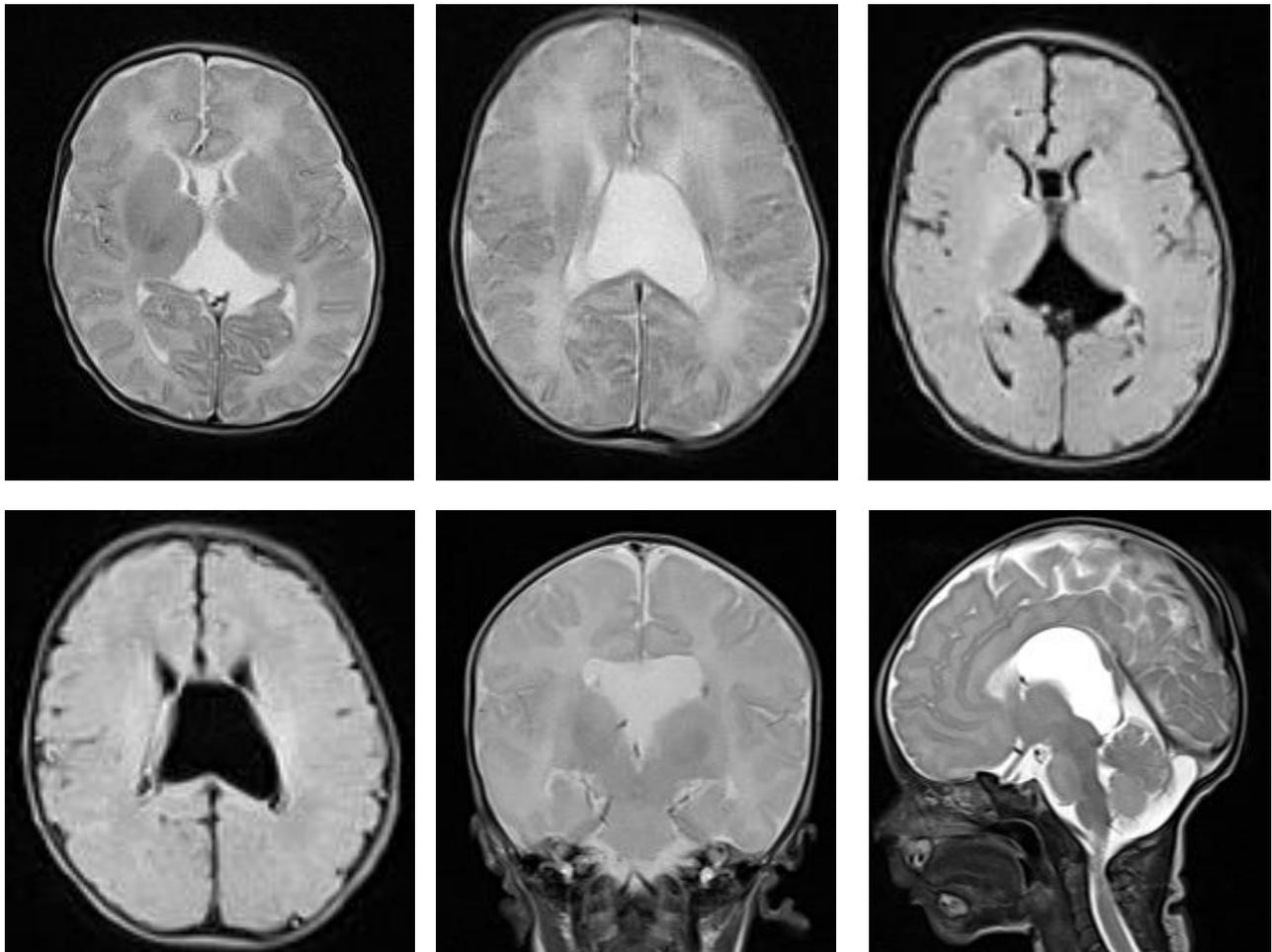


Figure 1: CAVUM VELUM INTERPOSITUM CYST - triangular CSF signal intensity lesion located behind the foramen of Monro, beneath the fornices of bilateral lateral ventricles and above the tela choroidea of III ventricle, the tip of the triangle is pointing towards foramen of Monro and base posteriorly displacing the splenium of corpus callosum. This cystic lesion is causing elevation/splaying of fornices, posterior displacement of splenium of corpus callosum and inferior displacement of internal cerebral veins.

NEUROGLIAL CYSTS: Neuroglial (also called gliependymal) cysts are benign epithelial-lined lesions that occur anywhere in the neuraxis. They are uncommon, representing fewer than 1% of intracranial cysts, the frontal lobe is the most typical location. Also, intraparenchymal neuroglial cysts are more common than extraparenchymal cysts.¹¹

Intraparenchymal neuroglial cysts are congenital lesions, arising from embryonic neural tube elements that become sequestered within the developing white matter. They are rounded, smooth, and unilocular and contain clear fluid that resembles CSF. They are lined by ependymal (columnar epithelium) or choroid plexus cells (low cuboidal epithelium).

Imaging: The best diagnostic clue to a neuroglial cyst is a nonenhancing CSF-like parenchymal cyst with minimal to no surrounding signal intensity abnormality. The cysts are benign-appearing lesions with smooth, rounded borders.¹¹

PINEAL CYSTS: Pineal cysts and cystic degeneration of the pineal gland with some residual pineal parenchyma are common;

Microscopically, benign pineal cysts exhibit three distinct layers. The outer layer consists of a delicate layer of fibrous connective tissue. The middle layer is composed of pineal parenchyma with or without calcium. The inner layer is composed of finely fibrillar glial tissue that often contains hemosiderin deposits.

They are smooth unilocular cysts with a soft tan to yellow wall. Contents vary from clear to yellow (most common) to hemorrhagic. Eighty percent are smaller than 1 cm in diameter. Cysts larger than 1.5 cm may result in hydrocephalus by causing compression of the tectum and aqueduct.¹²⁻¹⁴

Imaging: The best diagnostic clue is unilocular fluid-filled mass within the pineal gland. Attenuation or signal intensity varies with cyst content. One-fourth have rim or nodular calcium in the cyst wall on nonenhanced CT scans.

Rim or nodular enhancement is also common. On T1-weighted MR images, 55%–60% are slightly hyperintense to CSF. Most do not appear hypointense on FLAIR images, and 60% enhance with use of contrast material.^{15,16}

ARACHNOID CYSTS: Arachnoid cysts are benign, congenital, intraarachnoidal space-occupying lesions that are filled with clear CSF. They do not communicate with the ventricular system. Most arachnoid cysts are supratentorial. Fifty to 60% are found in the middle cranial fossa, anterior to the temporal lobes. Other locations include the suprasellar cistern and posterior fossa (10%), where they occur most commonly in the cerebellopontine angle cistern. Less common locations are within the interhemispheric fissure; over the cerebral convexity; or in the choroidal fissure, cisterna magna, quadrigeminal cistern, and the vermian fissures.

Imaging: NECT Usually CSF density, hyperdense, if intracyst hemorrhage present (rare), may expand, thin/ remodel bone,

doesn't enhance on CECT: CTA: Posterior displacement of MCA in MCF ACsCT: Cisternography may demonstrate communication with subarachnoid space.

MR Findings: Sharply-margined extra-axial fluid collection isointense with CSF on T1WI, Isointense with CSF on T2WI, suppresses completely on FLAIR, no blooming on T2* GRE unless hemorrhage present, no restriction on DWI and doesn't enhance on contrast. MRA: Cortical vessels displaced away from calvarium and phase-contrast cine MR - flow quantification can help distinguish arachnoid cyst from enlarged subarachnoid space.¹⁷⁻²⁰ Arachnoid cysts are generally stable over time, although cases of sudden or progressive enlargement, as well as spontaneous resolution, have been reported.^{21,22}

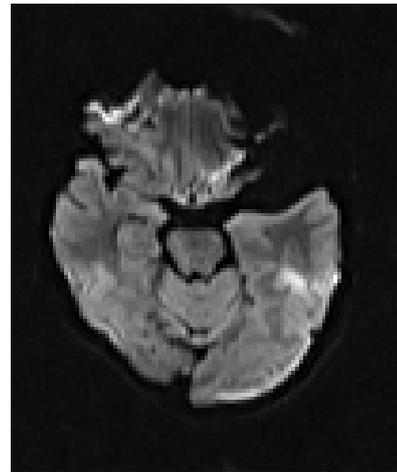


Figure 2: ARACHNOID CYST –Cystic lesion in left temporal lobe convexity following CSF signal intensity, suppressed on FLAIR with no restricted diffusion on DWI causing mild scalloping of temporal lobe

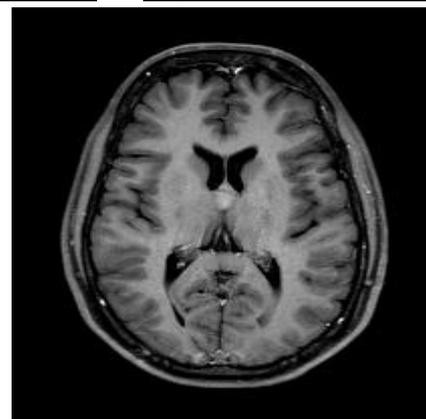
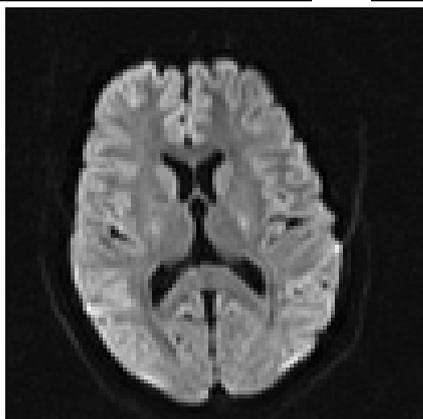
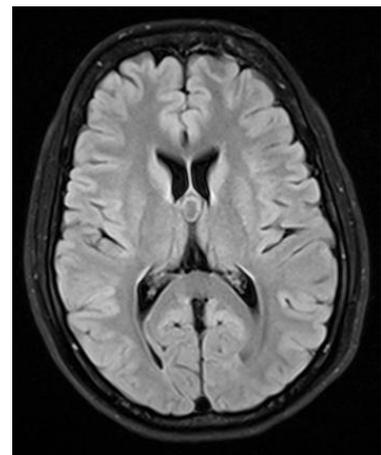
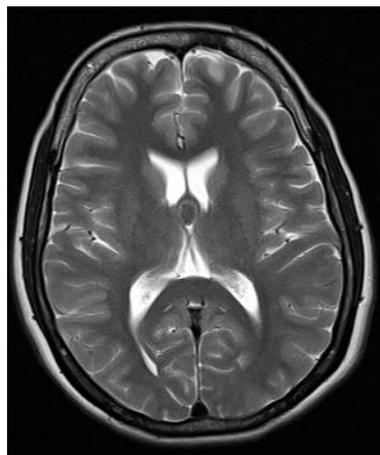
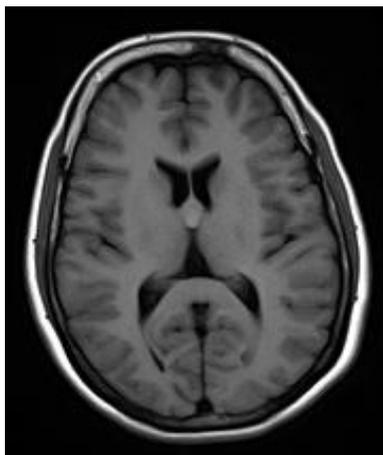


Figure 3: COLLOID CYST – Cystic lesion at foramen of monro which is hyperintense on T1W, iso to hyperintense on T2W, not suppressed on FLAIR, no restricted diffusion on DWI and no enhancement on post contrast.

COLLOID CYSTS: Approximately three people per million per year receive a diagnosis of a colloid cyst, Colloid cysts are benign mucin-containing cysts and account for 0.5%–1% of primary brain tumors and 15%–20% of intraventricular masses. More than 99% are found wedged in the foramen of Monro. Even relatively small colloid cysts may produce sudden acute hydrocephalus. Occasionally brain herniation with rapid clinical deterioration and even death ensue.^{23,24}

The cysts are smooth and spherical, varying in size from 0.3 cm to more than 4 cm in diameter. The mean size is 1.5 cm. The cysts are filled with viscous gelatinous material that consists of mucin, blood degradation products, foamy cells, and cholesterol crystals.^{23,24}

The cysts are typically attached to the anterosuperior portion of the third ventricular roof.

Imaging: The best diagnostic clue to a colloid cyst is its location at the foramen of Monro. The classic colloid cyst appears as a well-delineated hyperattenuated mass on nonenhanced CT scans. Attenuation correlates inversely with hydration state. On T1-weighted MR images, two-thirds of colloid cysts are hyperintense. The majority are isointense to brain on T2-weighted images. Some demonstrate peripheral rim enhancement.^{23,24}

Rarely, cysts are found at other sites, including the lateral ventricles, cerebellar parenchyma, pituitary and various extra axial locations.^{25,26}

EPIDERMOID CYSTS: Congenital inclusion cysts that comprise 0.2%–1.8% of primary intracranial tumors and are four to nine times as common as dermoid cysts.^{27,28} The most common

location for epidermoid cysts is the cerebellopontine angle cistern (40%–50%), where they are the third most common overall cerebellopontine angle cistern–internal auditory canal mass (after acoustic schwannoma and meningioma). Epidermoid cysts also occur in the fourth ventricle (17%) and the sellar and/or parasellar regions (10%–15%). Less common locations include the cerebral hemispheres or brainstem.^{29,30} Ten percent of epidermoid cysts are extradural, located in the skull or spine. All are located off the midline, most are asymptomatic but may occasionally result in mass effect, cranial neuropathy, or seizure.^{29,30} Occasionally, epidermoid cysts rupture and may excite a granulomatous meningitis

Imaging: The best diagnostic clue is a CSF-like mass that insinuates within cisterns, encasing adjacent nerves and vessels. On CT scans, most epidermoid cysts are well-defined hypoattenuated masses that resemble CSF and do not enhance. Calcification is present in 10%–25% of cases. Most epidermoid cysts are isointense or slightly hyperintense to CSF on both T1- and T2-weighted MR images. They do not suppress completely on FLAIR images and restrict (show high signal intensity) on diffusion-weighted images. Most epidermoid cysts do not enhance, although some minimal rim enhancement occurs in approximately 25% of cases.

Rare “white epidermoids” have high protein content and may appear hyperattenuated on CT scans. Compared with the classic epidermoid cyst, these “dense” or white epidermoids show reversed signal intensity on MR images, with high signal intensity on T1- and low signal intensity on T2-weighted images.^{27,28}

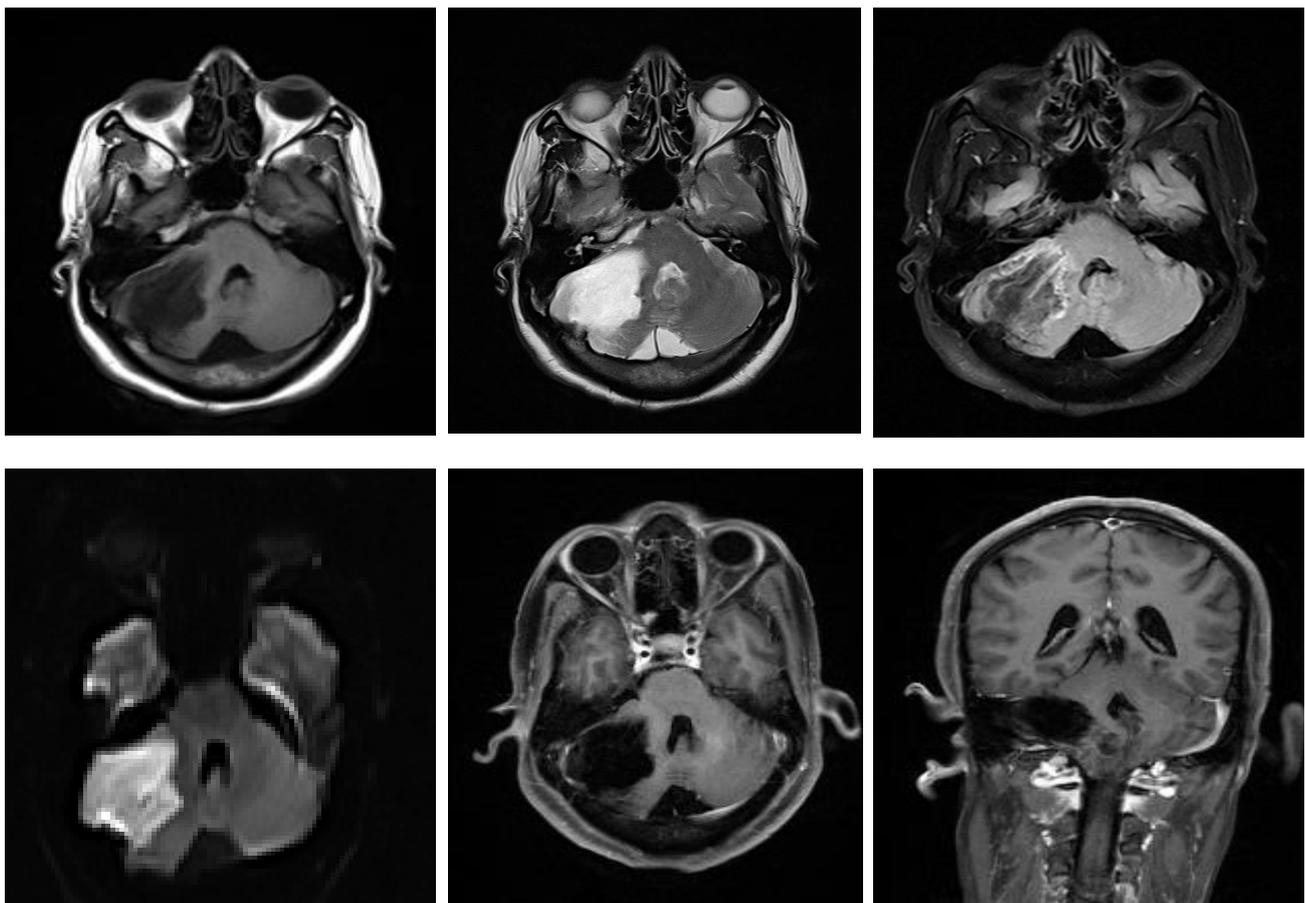


Figure 4: CP ANGLE EPIDERMOID CYST - Well defined irregular lobulated contoured T1 iso to hypointense/ T2 hyperintense/ FLAIR mixed intensity lesion with restricted diffusion on DWI seen in right CP angle extending anteroinferiorly to right paramedullary space and showing no significant enhancement on post contrast

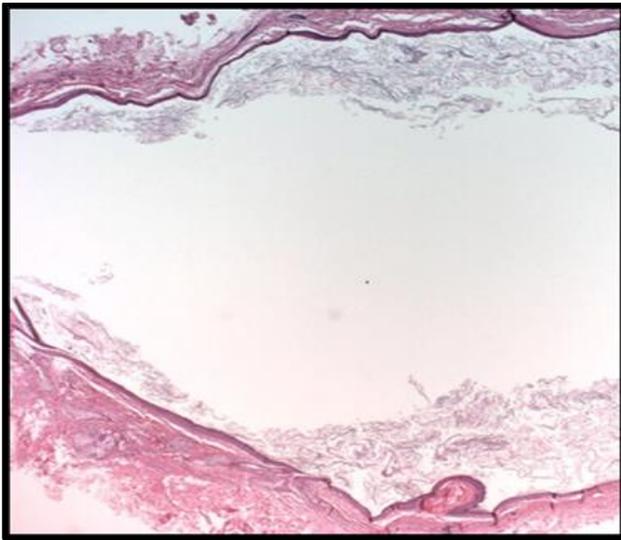


Figure 5: Section show features of epidermoid cyst (H&E 10X)

DERMOID CYSTS: Dermoid cysts, like epidermoid cysts are lined by stratified squamous epithelium. Unlike epidermoid cysts; they contain epidermal appendages as well as hair follicles, sweat glands and sebaceous glands. The latter secrete the sebum that gives the characteristic appearance of these lesions on CT and MRI. Like epidermoid cysts, dermoid cysts are congenital ectodermal inclusion cysts, they are extremely rare, constituting fewer than 0.5% of primary intracranial tumors and are four to nine times less common than epidermoid cysts,³¹ they tend to occur in the midline sellar, parasellar, or frontonasal regions, other dermoid cysts are midline in the posterior fossa, where they occur either as vermian lesions or within the fourth ventricle,³¹ these cysts increase in size by means of glandular secretion and epithelial desquamation. Growth can lead to rupture of the cyst contents, causing a chemical meningitis that may lead to vasospasm, infarction, and even death, malignant transformation into squamous cell carcinoma has also been described.^{31,32}

Imaging: Imaging findings vary, depending on whether the cyst has ruptured. Unruptured cysts have the same imaging characteristics as fat because they contain liquid cholesterol, all are hyperintense on T1-weighted images and do not enhance. The masses have heterogeneous signal intensity on T2-weighted MR images and vary from hypo- to hyperintense, the best diagnostic clue of a ruptured dermoid cyst is fatlike droplets in the subarachnoid cisterns, sulci, and ventricles, extensive pial enhancement can be seen from chemical meningitis caused by ruptured cysts.^{31,32}

NEURENTERIC CYSTS: Neurenteric cysts are congenital, benign, malformative endodermal lesions in the central nervous system. They are approximately three times as common in the spine, compared with the brain, most intracranial neurenteric cysts are found in the posterior fossa. They are typically in the midline, anterior to the brainstem. They can also be found in the cerebellopontine angle or clivus. Supratentorial cysts have rarely been reported. The size of the cysts is variable, usually measuring less than 2 cm.³³⁻³⁷

Imaging: The best diagnostic clue for a neurenteric cyst is a round and/or lobulated, nonenhancing, slightly hyperintense mass in front of the medulla. The signal intensity characteristics vary depending on the protein content of the cysts. Most are proteinaceous with a T1-weighted imaging appearance that is iso-

to slightly hyperintense compared with the CSF and a T2-weighted imaging appearance that is very hyperintense, neurenteric cysts are hyperintense on FLAIR images and may show mild restriction on diffusion-weighted images. They very rarely show rim enhancement.³³⁻³⁵

RATHKE CLEFT CYSTS: Rathke cleft cysts probably arise from the failure of obliteration of the rathke pouch, which develops as a rostral outpouching of the primitive oral cavity during the 3rd or 4th week of embryogenesis.^{15,18} Rathke cleft cysts are congenital nonneoplastic cysts arising from remnants of the embryonic rathke cleft. Forty percent are completely intrasellar, while 60% have some suprasellar extension. Completely suprasellar cysts are rare, symptoms occur from compression of the optic chiasm, hypothalamus, or pituitary gland.^{15,18}

Rathke cleft cysts are smoothly margined cysts that vary in size from a few millimeters to 1–2 cm.^{38,39}

Imaging: The best imaging clue is a nonenhancing noncalcified intra- and/or suprasellar cyst with an intracystic nodule, while this is the typical picture, the imaging characteristics vary widely. Approximately half are hyperintense on T1-weighted images, while half are hypointense. On T2-weighted images, 70% are hyperintense and 30% are iso- or hypointense. A small nonenhancing intracystic nodule is considered a virtually pathognomonic sign of a rathke cleft cyst. These nodules show high signal intensity on T1-weighted images and low signal intensity on T2-weighted images, and they do not enhance.

Rathke cleft cysts do not enhance after contrast material administration, although an enhancing rim of displaced compressed pituitary gland is present in approximately half of the cases.³⁸⁻⁴¹

SCHIZENCEPHALY: Characterized by the presence of slots extending through the whole cerebral hemisphere, from the ependymal surface to the cortex. These grooves are lined by dysplastic gray matter. They can be open or closed. If they are open, cerebrospinal fluid (CSF) fills the path between the ventricle and the subarachnoid space of the convexity. If it is closed, an indentation is seen in the ventricular wall.⁴²

VENTRICULOMEGALY: Consists in dilation of the ventricles due to hydrocephalus, it may

mimic cystic structures. Linear ultrasound imaging transducer may be useful in identifying the thinned cortical hydrocephalus present and absent in the hydrancephaly.

MEGA CISTERNA MAGNA: It has an incidence of 1%. It is considered megacisterna magna over 10mm when measured in the sagittal plane without involvement of the cerebellar hemispheres.

DANDY-WALKER MALFORMATION: This is a congenital anomaly characterized by the classic triad of hydrocephalus, absence or agenesis of the cerebellar vermis and posterior fossa cyst communicating with the fourth ventricle. Secondarily can cause hydrocephalus and intracranial hypertension.⁴³

POST-TRAUMATIC/VASCULAR

PERIVENTRICULAR LEUKOMALACIA: Periventricular leukomalacia is necrosis of white matter with a characteristic distribution external to the lateral ventricles involving the centrum semiovale that occurs in preterm infants less than 32 weeks. In ultrasound hyperechoic areas are displayed first periventricular this being more sensitive to ischemia region.⁴⁴

MULTICYSTIC ENCEPHALOMALACIA: It is due to a diffuse insult, caused by ischemia, infection, or developmental insults to the brain late in gestation, during birth, or after birth. Multiple cystic cavities of variable size separated by glial septations form in the necrotic area. These are encephalomalacic cysts.⁴⁴

PORENCEPHALIC CYSTS: These are CSF-filled intra-parenchymal lesions. These lesions, which may be congenital or acquired following an infarct, may communicate with the ventricular system. Imaging reveals a cystic intra-parenchymal cavity corresponding to specific arterial territory, with enlargement of the adjacent ventricle. Porencephalic cysts are isodense to CSF on CT and isointense to CSF on all MRI sequences.⁴⁵

LEPTOMENINGEAL CYSTS: They can form when there is a loculation of CSF onto or through a skull defect, typically when a linear fracture in the frontal or parietal bone (>4 mm wide) is accompanied by a dural tear. Arachnoid tissue herniates into the dural defect, and CSF pulsations produce fracture diastasis. This may consequently also be referred to as the "growing" fracture. Leptomeningeal cysts are frequently accompanied by an adjacent area of encephalomalacia. Imaging should show a cyst that has the density of CSF. It will be isointense to CSF on all MR sequences and will communicate with the subarachnoid space. This lesion should also be observed adjacent to a skull defect and have indistinct scalloped margins.⁴⁶

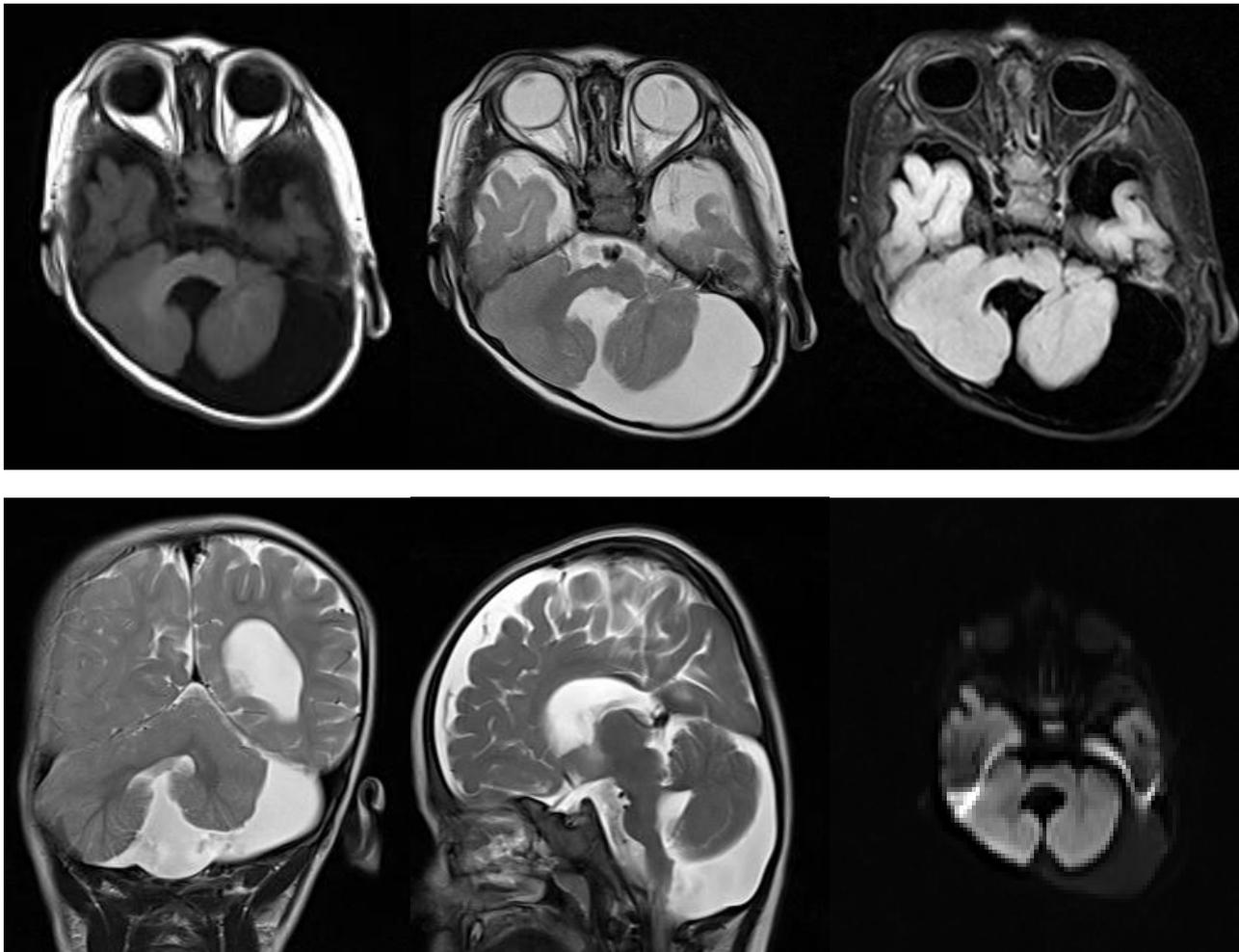


Figure 6: DANDY WALKER MALFORMATION – well defined CSF signal intensity cystic lesion showing no restricted diffusion on DWI noted in retrocerebellar location more on left side communicating with fourth ventricle associated with vermian hypoplasia, displacing the cerebellum and torcula heterophili superiorly.

POSTINFECTION/INFLAMMATION

ABSCCESS: A focal pyogenic infection of the brain parenchyma that has a thin uniform wall, homogeneous center, and surrounding florid edema is an abscess. Most abscesses occur in the supratentorial region in the frontal or parietal lobes at the gray-white interface. Up to 14% of these lesions occur infratentorially.^{47,48}

Imaging findings on CT vary according to the stage of the abscess. An ill-defined hypodense subcortical lesion with mass effect is seen in the early cerebritis stage. Imaging of the late cerebritis stage shows a central hypodense lesion with peripheral

edema and an increased mass effect. In the early capsule stage, there will be a hypodense mass and moderate vasogenic edema with mass effect. Edema and the mass effect decrease in the late capsule stage.^{47,48}

Abscesses appear with a hypointense rim on T2-weighted MR sequences, hyperintense on diffusion-weighted MR sequences, and hypointense on apparent diffusion coefficient mapping. MR spectroscopy shows an increase in levels of acetate, lactate, pyruvate, alanine, succinate, and amino acids. This finding permits the differentiation of an abscess from necrotic or cystic tumors.⁴⁷⁻⁴⁹

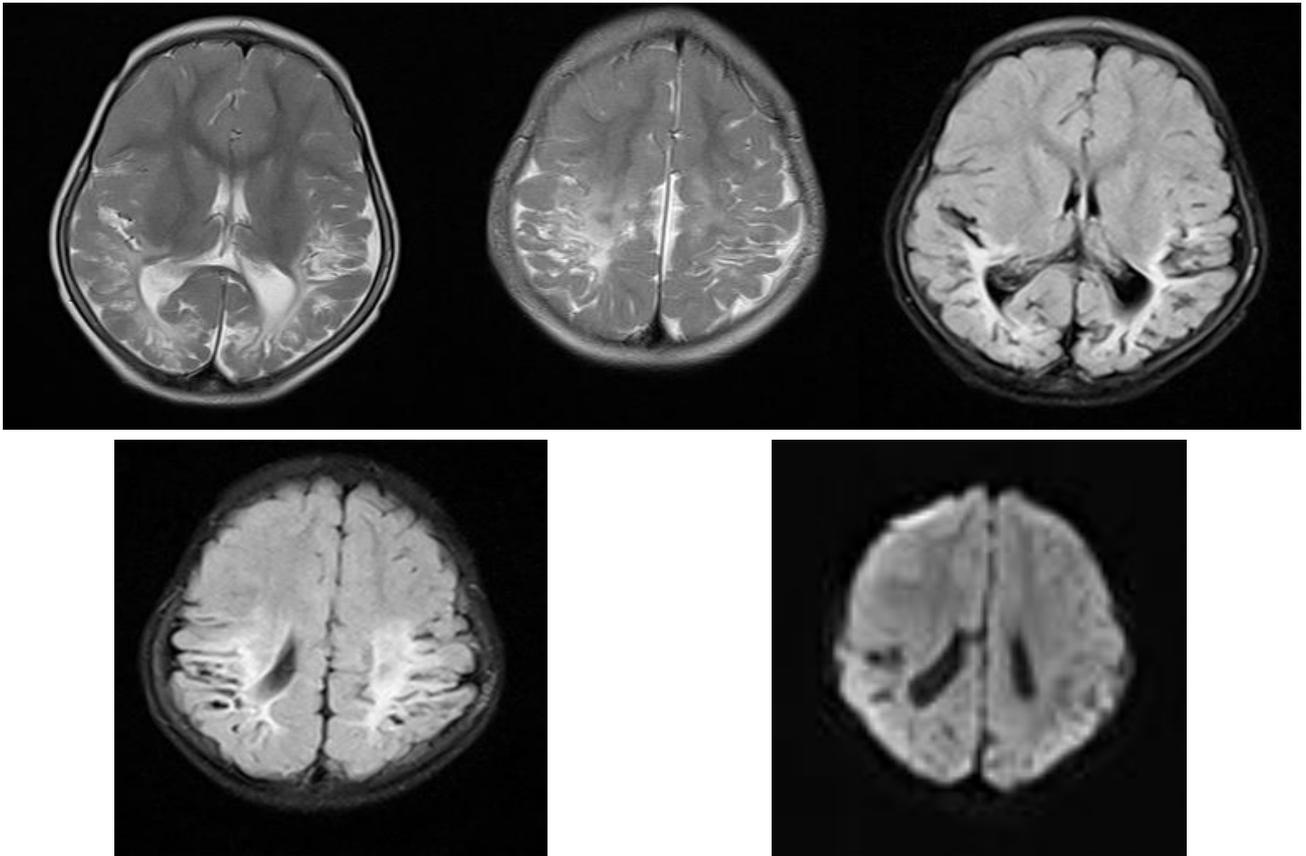


Figure 7: CYSTIC PERIVENTRICULAR LEUKOMALACIA– Symmetrical T2W/FLAIR hyper intensities with no restriction on DWI ,interspersed with cystic spaces& gliosis noted in periventricular deep white matter, sub cortical white matter and grey white matter junction of bilateral parieto-occipital lobes associated with asymmetrical dilatation of atrium and occipital horns of bilateral lateral ventricles

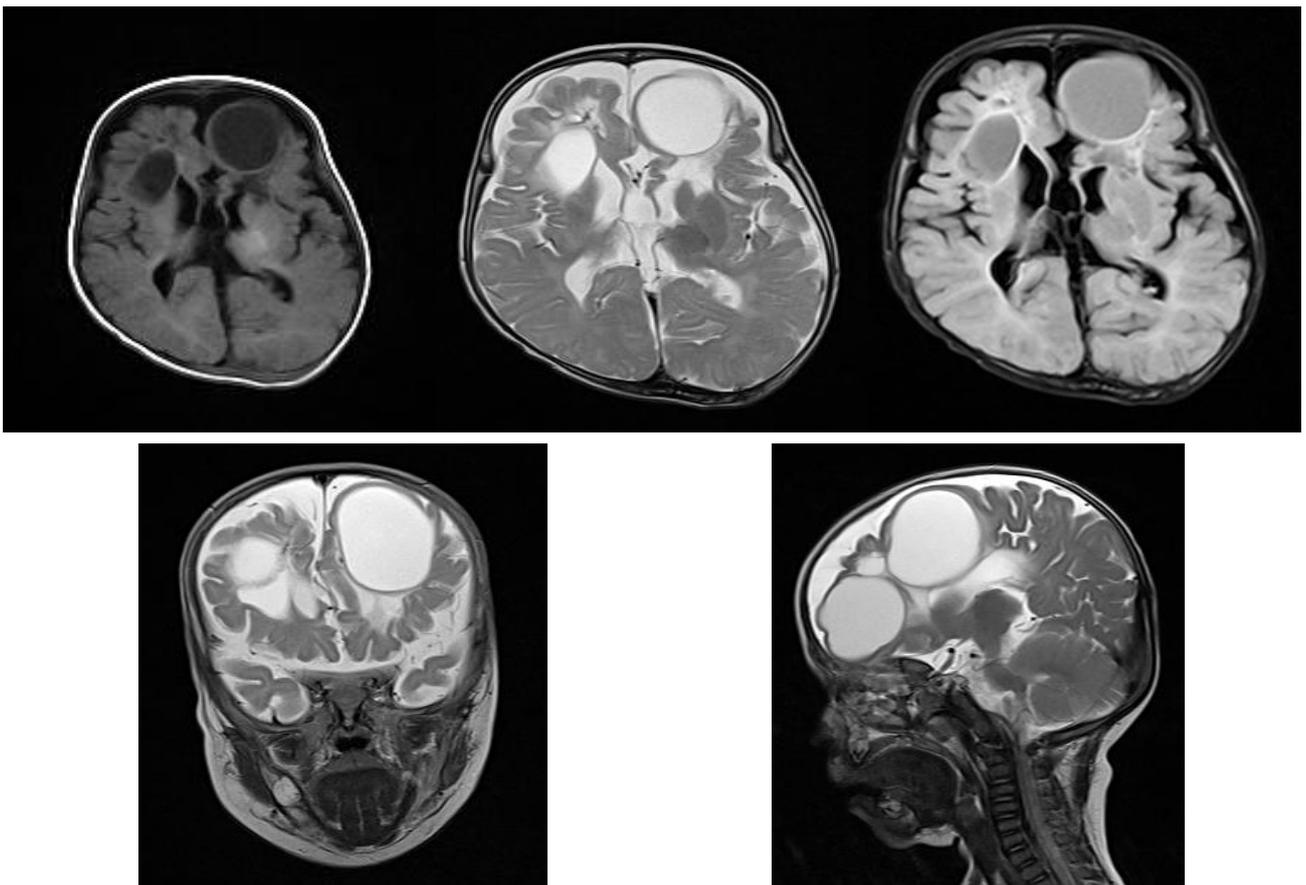


Figure 8: MULTI CYSTIC ENCEPHALOMALACIA - Well defined T1 hypointense/ T2 hyperintense/FLAIR hyperintense cysts seen in both frontal lobes associated with adjacent gliosis, paucity of white matter and corpus callosal agenesis

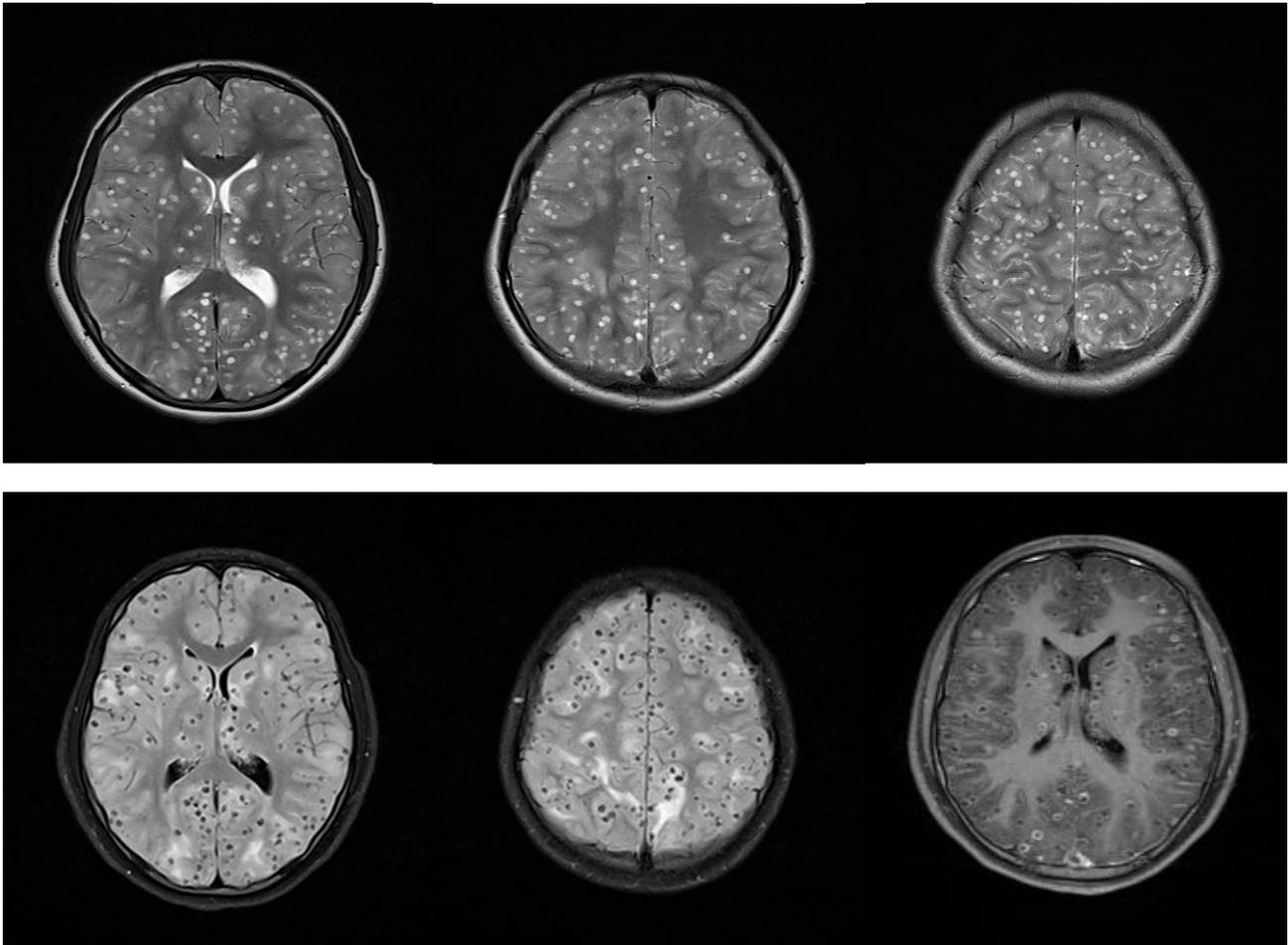


Figure 9: NEUROCYSTICERCOSIS- multiple ring enhancing lesions of varying stages with few showing perilesional edema noted scattered in cerebral parenchyma- (Starry sky appearance)

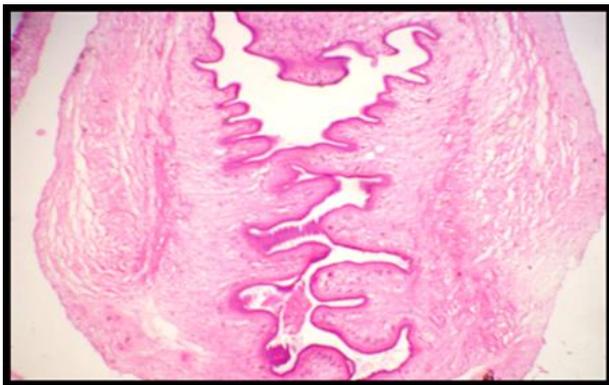


Figure 10: Section show cyst wall and bladder of cysticercus cellulosae (H&E 10X)

NEUROCYSTICERCOSIS: Neurocysticercosis, which is caused by the larva of taenia solium, is the most common parasitic infection of the central nervous system. The lesion can be located anywhere within the CNS, though the parenchymal cortico-medullary junction is the most common site in the brain. CT reveals the four distinct stages of neurocysticercosis. Imaging-based identification of the lesion's different stages is pathognomonic.^{50,51}

In the vesicular stage, when the larva is viable, CT shows a cystic lesion with a central hyperdense nodule, representing the scolex of the Taenia. No edema or enhancement is observed. The degenerating larva induces edema in the colloidal vesicular stage.

The lesion then appears as a focal hypodense area with ring enhancement. As the lesion heals during the granular nodular stage, it becomes isodense or mildly hyperdense with a calcified scolex. Lesion enhancement and edema are both characteristic. The final stage is reached when the lesion has healed. CT reveals a completely calcified nodule, without edema or enhancement.^{50,51}

MR Findings^{51,52}

TIWI -Vesicular stage: Cystic lesion isointense to CSF, may see discrete, eccentric scolex (hyperintense);

Colloidal vesicular stage: Cyst is mildly hyperintense to CSF;

Granular nodular stage: Thickened, retracted cyst wall;

Nodular calcified stage: Shrunken, Calcified lesion.

T2WI-Vesicular stage: Cystic lesion isointense to CSF, may see discrete, eccentric scolex No surrounding edema;

Colloidal vesicular stage: Cyst is hyperintense to CSF, surrounding edema may be mild to marked;

Granular nodular stage: Thickened, retracted cyst wall; edema decreases;

Nodular calcified stage: Shrunken, Calcified lesion.

FLAIR- Vesicular stage: Cystic lesion isointense to CSF, may see discrete, eccentric scolex (hyperintense to CSF) with no edema.

Colloidal vesicular stage: Cyst is hyperintense to CSF with surrounding edema, mild to marked, useful to detect intraventricular cysts (hyperintense).

T2* GRE: Useful to demonstrate calcified scolex.

DWI: Cystic lesion typically isointense to CSF, ADC values usually higher than tuberculomas.

TI C+ -Vesicular stage: No enhancement typical, may see discrete, eccentric scolex enhancement;

Colloidal vesicular stage: Thick cyst wall enhances with enhancing marginal nodule (scolex);

Granular nodular stage: Thickened, retracted cyst wall; may have nodular or ring-enhancement;

Nodular calcified stage: rare minimal enhancement; In children, may see "encephalitic cysticercosis" with multiple small enhancing lesions and diffuse edema. Intraventricular cysts may cause ventriculitis and/or hydrocephalus.

Cisternal NCC may appear racemose (multilobulated, grape-like), typically lacks scolex). Complications: Meningitis, hydrocephalus, vasculitis.

CEREBRAL HYDATID DISEASE: Cerebral hydatid disease, which is caused by *Echinococcus granulosus* (the hydatid worm), is extremely rare. Hydatid cysts may be single or multiple, uni- or multiloculated, and thin- or thick-walled. More specific signs include visualization of a calcified wall, presence of daughter cysts, and membrane detachment. Hydatid cysts are typically supratentorial. They can occur anywhere within the brain but are more often located in the middle cerebral artery territory. The parietal lobe is the most frequently involved region. A mass effect is observed when imaging a hydatid cyst, but contrast enhancement is absent.⁵³⁻⁵⁵

CT Findings⁵³⁻⁵⁵

NECT: Unilocular or multilocular cyst, isodense to CSF with no edema. CECT: No enhancement.

MR Findings⁵³⁻⁵⁵

TIWI: Cyst isointense to CSF, T2WI-Cyst isointense to CSF with hypointense rim with no perilesional edema and TI C+ - No enhancement and may see fine peripheral enhancement.

CRYPTOCOCCOSIS: Cryptococcosis, the most common mycotic infection of the central nervous system, primarily manifests as meningitis. Several reports have described extension of the meningeal infection along perivascular spaces giving rise to small cysts, termed gelatinous pseudocysts, in the PVSs and adjacent basal ganglia. Punctate hyperintensities, representing dilated PVSs or cryptococcomas, are frequently seen in the basal ganglia, thalami, and midbrain on T2WI. Larger gelatinous pseudocysts tend to give a —soap bubble appearance, with hypointensity on T1WI and FLAIR and hyperintensity on T2WI.⁵⁶

TUMEFACTIVE MULTIPLE SCLEROSIS: Tumefactive multiple sclerosis is cystic in appearance and may mimic the appearance of neoplasms. Lesions show semilunar (horseshoe-shaped) enhancement on MRI following intravenous injection of gadolinium contrast.^{57,58} MRS reveals low levels of N-acetylaspartate (NAA) and a low NAA/creatine ratio. Choline and lactate levels are both elevated.⁵⁹

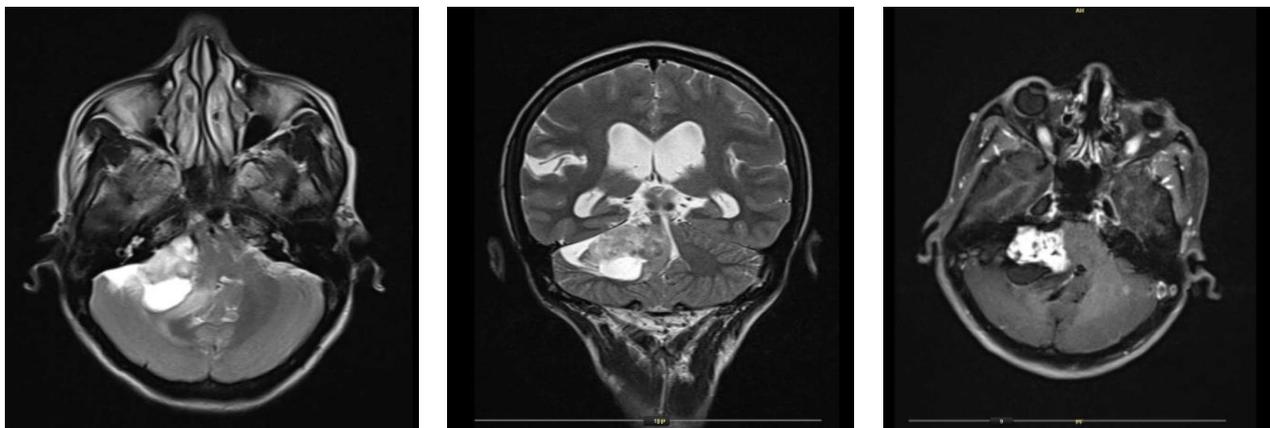


Figure 11: CYSTIC SCHWANOMMA—Solid cystic lesion noted in right CP angle showing intense heterogenous enhancement in solid component with no enhancement in cystic component.

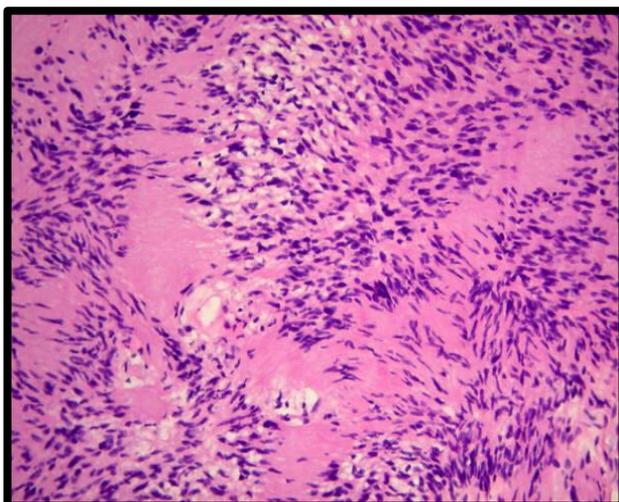


Figure 12: Section show verrucay bodies of cystic schwannomma (H&E 10X)

NEOPLASMS WITH CYSTIC COMPONENT^{3,60,61}

CYSTIC SCHWANNOMA: Cystic schwannoma is a benign well-delineated round or lobulated encapsulated tumor that arises eccentrically from schwann nerve cells. Acoustic schwannoma is the most commonly seen cerebellopontine angle tumor. Multiple schwannomas are characteristic of type 2 neurofibromatosis. Schwannomas commonly show cystic or fatty degeneration and hemorrhagic necrosis. Between 15% and 20% of schwannomas show an intralesional or peritumoral cyst.

Imaging findings: Extra-axial mass with enlarged ipsilateral cerebellopontine angle cistern, a CSF vascular cleft, displaced gray-white matter interface, brain stem rotation, and compression of the fourth ventricle. Schwannomas are iso- to hypodense on CT and hyperintense on T2WI /FLAIR, show patchy restricted diffusion on DWI, blooming on T2 GRE likely due to hemorrhage /calcifications and solid components enhance intensely after contrast administration.

MENINGIOMA: Meningioma is the most common nonglial primary brain tumor to arise from specialized meningotheial cells. Most meningiomas are extra-axial, dural-based lesions with a dural tail. Around 90% occur in the supratentorium. Typical meningiomas are hyperdense on unenhanced CT and isointense to gray matter on all MRI sequences. Enhancement after contrast administration is intense/homogenous with few shows enhancing dural tail sign. True cystic meningiomas with large intratumoral cysts are uncommon, though 10% to 15% of meningiomas show cystic areas of necrosis. An alanine peak is seen on MRS.

CRANIOPHARYNGIOMA: Craniopharyngioma is a benign slow-growing tumor that arises from the vestigial craniopharyngeal duct or Rathke cleft. These lesions are typically lobulated, well-delineated cystic masses that generally occur in the sellar and suprasellar region.

CT reveals a cystic lobulated suprasellar mass with a solid mural nodule showing nodular or rim calcification. Craniopharyngiomas have variable signal intensity on MRI, though they are most often hypointense on T1-weighted and hyperintense on T2-weighted MR sequences.

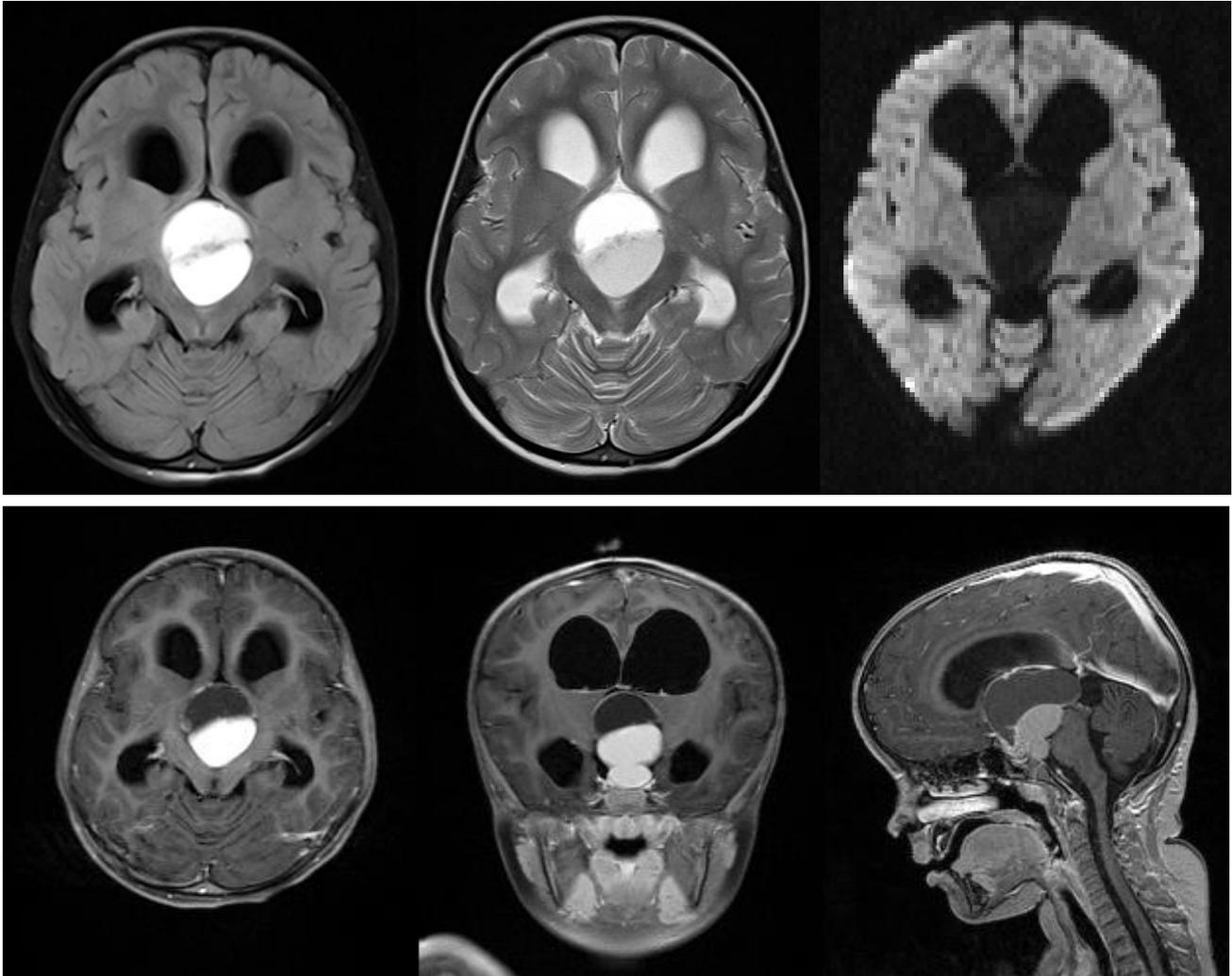


Figure 13: CYSTIC CRANIOPHARYNGIOMA—Solid cystic lesion in sellar region with suprasellar extension showing peripheral rim enhancement in cystic component on IV contrast.

PITUITARY MACROADENOMAS: Make up 70% to 80% of pituitary adenomas. Macroadenomas are greater than 10 mm across and are usually endocrinologically inactive. They have a figure of eight appearance with a suprasellar component. The lesions are usually isointense to cortical gray matter and enhance intensely on MRI after administration of gadolinium contrast. They generally contain necrotic, cystic, or hemorrhagic components that show variable signal intensity.

HEMANGIOBLASTOMA: Is an uncommon vascular neoplasm of unknown origin. The most common location is the cerebellum, with 10% to 20% of hemangioblastomas occurring as part of von Hippel-Lindau syndrome. Lesions are typically subpial well-circumscribed tumors, 60% of which will be cystic with a mural nodule. CT depicts hemangioblastoma as a low-density cyst with

a strongly enhancing mural nodule abutting a pial surface. The lesion's cystic component is hyperintense to CSF on all MRI sequences. The mural nodule has variable signal intensity characteristics and strongly enhances on administration of gadolinium contrast.

PILOCYTIC ASTROCYTOMAS: Constitute almost one-third of pediatric gliomas. These lesions are predominantly seen in children and young adults, and they are characteristically located around the third and fourth ventricles. Almost 50% of tumors are found in the optic chiasm and hypothalamus, and 35% in the cerebellum. Less common locations include the cerebral hemispheres and ventricles.

CT reveals a uni- or multilocular cyst, which is slightly hyperdense to CSF and contains a mural nodule with inhomogeneous contrast

enhancement. Calcifications are seen in 10% to 20% of cases. The cyst wall is slightly hyperdense and does not enhance. Pilocytic astrocytomas are hypointense on T1-weighted MR sequences and hyperintense on T2-weighted sequences. Enhancement of the solid mural nodule varies

GLIOBLASTOMA MULTIFORME: Is a common primary brain tumor that typically occurs in the cerebral hemispheric white matter. Lesions are poorly delineated diffusely infiltrating partially necrotic masses. A thick irregular rind of tissue is seen around a necrotic core on imaging. Signal intensity is variable on MRI and depends on the solid and cystic components. Glioblastoma multiforme tumors enhance heterogeneously following injection of gadolinium contrast.

CYSTIC OLIGODENDROGLIOMA: Is a slow-growing neoplasm that arises from oligodendrocytes. This lesion usually occurs in adults and is most commonly located in the frontal lobe. Cystic oligodendrogliomas generally start in the hemispheric white matter and grow toward the cortex. Foci of cystic degeneration and calcification are common. Imaging depicts this neoplasm as a heterogeneous hemispheric mass that often involves the cortex. Brain stem glioma is a nonenhancing focal glioma involving the

midbrain, pons, or medulla. The brain stem is usually expansile.

PLEOMORPHIC XANTHOASTROCYTOMA: Is a rare partially cystic tumor with a discrete mural nodule that occurs in children and young adults. This lesion is typically located in the superficial temporal lobe. Imaging generally shows a superficial partially cystic hemispheric mass with an enhancing mural nodule.

GANGLIOGLIOMAS: Most commonly occur in the temporal or frontal lobes, appearing as well-delineated cysts with a partially calcified mural nodule. These lesions generally are seen in children and young adults, and they can cause chronic temporal lobe epilepsy. They may also be associated with cortical dysplasia.

METASTASES: Account for 25% to 33% of brain tumors. Although they can occur anywhere, brain metastases are most often found at the junction between gray and white matter. Between 1% and 6.6% of metastases will be cystic, and the majority will have originated from a primary lung or breast tumor.^{1,2} Cystic metastases have striking surrounding edema and ringlike contrast enhancement on imaging. They are hypointense on diffusion-weighted sequences and hyperintense on apparent diffusion coefficient mapping.

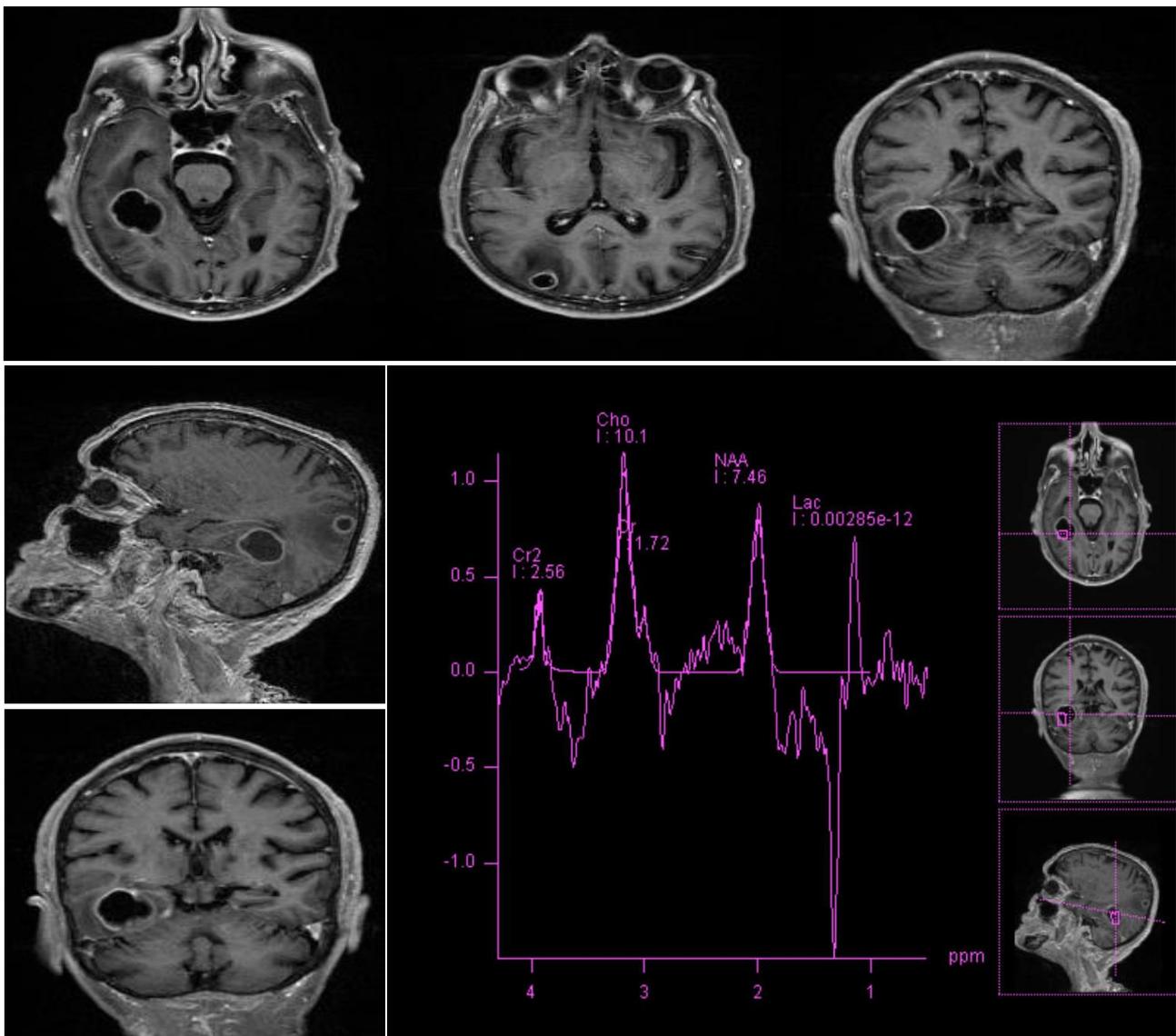


Figure 14: CYSTIC METASTASIS—Known case of lung carcinoma with multiple peripherally enhancing cystic lesions with central necrosis in right temporo parietal lobes, spectroscopy revealed raised Choline/creatinine peaks with reduced NAA within the wall and lactate peaks in necrotic areas.

AIMS AND OBJECTIVES

To enumerate and describe the various intracranial cystic lesions in literature and to diagnose cysts by MRI.

MATERIALS AND METHODS

50 patients referred to radiology department suspected to have intracranial sign and symptoms were studied for a period of two years from January 2013 to December 2014 .Patient age group was ranging from 5-65 years and they were subjected to MRI scan using standard intracranial protocols. Few cases were operated and sent for histopathological examination.

RESULTS

Out of 50 cases, 24 were post infective/inflammatory, 16 were developmental/ congenital, 4 were post traumatic/vascular, 4 were neoplastic lesions with cystic component and 2 were normal variants.(Table- I)

DISCUSSION

In the present study, most common cystic lesions of infective etiology were neurocysticercosis. Out of 16 cases of neurocysticercosis (66.67 %); 4 cases showed diffuse cerebral involvement with cysts in varying stages (starry sky appearance). Remaining cases were abscess- 6 (25 %), cerebral hydatid disease -1 (4.17 %) and tumefactive multiple sclerosis-1 (4.17%). Most common cystic lesion in developmental/congenital group was arachnoid cyst- 9 cases (56.25 %), remaining were epidermoid cysts- 3 (18.75 %), colloid cyst -1 (6.25 %), neuroglial cyst -1 (6.25 %), ependymal cyst-1 (6.25 %) and dandy walker malformation -1 (6.25 %).

Most common cystic lesion in post-traumatic/vascular group was cystic periventricular leukomalacia -2 cases (50 %), remaining were porencephalic cyst-1 (25 %) and multi cystic encephalomalacia- 1 (25 %).

Most common cystic lesion in neoplastic lesions with cystic component were cystic schwannoma -1(25 %,) craniopharyngioma- 1 (25 %), pituitary macro adenoma-1 (25 %) and cystic metastasis -1 (25 %) Among the normal variants were cavum septum pellucidum and cavum velum interpositum cyst.

CONCLUSION

A broad spectrum of diseases can cause intracranial cysts.MR signal intensity at different sequences, presence or absence of restriction on diffusion-weighted images, location of lesions, enhancement pattern and spectroscopy can narrow down the differential diagnosis when an intracranial cyst is identified on imaging studies.

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