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Hydatid Disease of the Brain: Diagnostic and Therapeutic Nuances in Four Atypical Cases – Case Report

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Case Report

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Abstract

Introduction: Primary intracranial hydatid lesions are uncommon. We report four cases of cerebral hydatidosis, each with unique clinical presentations and management strategies, emphasizing the importance of tailored treatment.

Case details:

Case 1: A 19-year-old female presented with seizures and a left parietal lobe cyst. Managed conservatively with albendazole, she demonstrated significant radiological improvement and remained seizure-free at 9-month follow-up.

Case 2: A 39-year-old male exhibited right-sided weakness due to a left thalamic cyst. Surgical excision followed by albendazole therapy led to symptom resolution without recurrence at 12 months.

Case 3: A 19-year-old male developed quadriparesis from cerebellar and cervical spinal cord involvement. Surgical intervention resulted in substantial neurological recovery.

Case 4: A 64-year-old male with multiple left temporal lobe cysts and seizures was treated medically with albendazole, achieving partial cyst reduction and seizure control over 6 months.

Conclusion: Intracraniall hydatidosis, although rare, should be considered in patients from endemic regions with brain lesions. Atypical presentations demand high clinical suspicion. While surgery is primary, medical therapy is crucial for inoperable or multiple cysts. These cases highlight the importance of individualized, multidisciplinary management for achieving optimal outcomes.

Introduction

Hydatid disease is a zoonotic disease caused by helminth *Echinococcus granulosus*, which forms larval cysts in human body tissue as accidental intermediate hosts. The most common location of these cysts are liver. CNS involvement is infrequent, and hydatid cyst in the brain is seen in only 1–2% [1]. Hydatid disease as such is seen as an endemic disease in Turkey, Libya, Tunisia, Algeria, Iraq, Iran, Morocco, Egypt, and also in India [2]. Surgery is the primary management of cerebral hydatid cysts, with medical management playing a supportive role. In this article, we present four different presentations of primary cranial hydatidosis, along with their respective management strategies. All the patients underwent X-ray chest, ultrasound abdomen to rule out extracranial hydatid cysts.

Case Details

Case 1

A 19-year-old female presented to the emergency department with complaints of the sudden onset of abnormal body movements for 8 months, involving the face and right half of the body, with uprolling of

eyeballs. The patient was receiving treatment from a local doctor but had two similar episodes, following which the patient came to our hospital. On examination, the patient had mild right-sided weakness of grade 4/5 in the right upper and lower limbs. The MRI scan of the patient's brain was suggestive of a thin-walled, well-defined intraaxial lesion in the left parietal lobe, measuring 4.5 x 4.3 x 4.2 cm, with surrounding vasogenic edema causing a mass effect. Mild wall enhancement was observed on the post-contrast study, and a possible diagnosis of a Hydatid cyst of the brain was made. On lab investigations, absolute eosinophil count was 1330 cells/cm³ (normal 20–500), serum total IgE was 2410 IU/ml (normal 0-378), and serum IgGEchinococcal antigen was 1.97 (normal < 0.9). The patient was receiving anthelminthics drugs until then. A detailed discussion was undertaken with the parents about risks associated with the disease, and they were advised to undergo surgery as the sole modality of treatment.

The patient did not return for follow-up until 8 months later. On interrogation, parents revealed the patient was taking the advised anthelminthics medication (Tab Albendazole 200 mg BD) apart from the antiepileptic drug. The NCCT scan was completed, showing that the lesion had reduced to an ill-defined parietal hypodensity in the region adjacent to the left lateral ventricle's occipital horn. Subsequent MRI of the brain was T1 isointense, T2/FLAIR hyperintense bilobed cystic lesion 1.8x1.6x1.7 cm in size in the peri trigonal white matter of the left parietal lobe [Figure 1]. The patient was discharged on anthelminthics drugs and antiepileptic drugs. At the 9-month follow-up, the patient is seizure-free and reports no additional complaints.

Case 2

A 39-year-old previously healthy male presented with a two-month history of persistent headaches, vomiting, and a gradual worsening of weakness in his right upper and lower limbs. Neurological examination revealed reduced muscle strength (MRC grade 4-/5) in the right upper and lower limbs, increased tone, and exaggerated reflexes in the same limbs. The NCCT head and MRI brain revealed a distinct cystic lesion with a hypodense rim, conspicuously located within the left thalamus. Moreover, the scans revealed surrounding edema and a noticeable midline shift, accentuating the critical nature of the condition [Figure 2].

To address this complex situation, the patient underwent a microsurgical procedure guided by neuronavigation, involving a left frontotemporal craniotomy. During the maneuvering of the cyst, an unintended breach occurred in its wall, resulting in the aspiration of its clear, watery contents. Interestingly, the visual characteristics of the cyst wall closely resembled those typically associated with a hydatid cyst. This prompted a swift decision to rinse the cystic cavity using a solution of 3% saline. Subsequently, the membranous lining of the cyst was easily excised using forceps [Figure 3].

The histopathological analysis of the cyst wall confirmed the diagnosis of a hydatid cyst. Following the surgical intervention, the patient’s treatment regimen included the administration of albendazole at a dose of 400mg twice daily. The postoperative period was uneventful, and the patient was discharged on

postoperative day 4. The patient has not reported any recurrence of symptoms, and follow-up NCCT 12 months after surgery was unremarkable.

Case 3

19 year old male child presented with complaints of headache since 6 months with imbalance while walking, parasthesias in bilateral upper and lower limbs and progressive spastic quadriparesis since 4 months and acute retention of urine 2 months back following which suprapubic cystostomy was done. Upon examination, the patient exhibited impaired vibration sense, proprioception, and 2-point discrimination below the level of the clavicle, whereas all other sensations were intact. On motor examination, bilateral foot drop was noted; tone in bilateral upper limbs was MAS 0, whereas bilateral lower limbs were MAS 0; power was MRC 1/5 at bilateral shoulders and 0/5 in all remaining muscle groups. On reflex examination, both superficial and deep reflexes were absent at all joints.

The NCCT head with MRI brain and cervical revealed well-defined cystic retrocerebellar and upper cervical spine multiple and multiloculated lesions measuring 5.1 x 5 cm, which were T1 hypo and T2 hyperintense, causing cerebellar and brainstem compression. Post-contrast subtle peripheral enhancement was observed [Figure 4]. The findings mentioned above pointed towards a possible diagnosis of disseminated cerebellar hydatid cyst in the cervical spinal cord. Laboratory investigations revealed an absolute eosinophil count of 3000 cells/cm^3 and a serum IgG Echinococcal antigen level of 2.09 (normal < 0.9). CT urography and IVP were suggestive of a dilated, large, round, smooth bladder.

The patient was planned for surgery with midline suboccipital craniotomy with C1 to C4 laminectomy and C2 to C4 lateral mass fixation. Multiple pearly white cystic lesions were present, with the largest thick-walled cyst in the retrocerebellar region and small multiple cysts in the cervical spinal cord up to the upper border of C5 lamina. The area was draped with 3% hypertonic saline soaked cottonoids, and cysts were removed one by one, carefully using forceps and gentle manipulation. The cervical spinal cord was found to be severely compressed anteriorly. Cyst wall in the retrocerebellar region was very densely attached to surrounding cerebellar parenchyma and vessels, dural venous sinuses, and tentorium superiorly, and hence left in situ after lavage of the cavity with 3% hypertonic saline [Figure 5].

Following surgical management, the patient underwent intensive physiotherapy and was discharged from the hospital after an uneventful hospital stay. At the 6-month follow-up, the patient had improved, with bilateral upper limb MRC grade of 4/5 and lower limb MRC grade of 3/5. Repeat radiological investigations did not suggest any recurrence.

Case 4

A 64-year-old male patient presented with complaints of headache for 3 months and two episodes of generalized tonic-clonic seizures (GTCS). Upon examination, the patient's GCS was 15/15, and no sensory or motor deficits were noted. The MRI brain scan revealed multiple well-defined, T1-hypointense and T2-hyperintense cystic lesions in the left temporal lobe, without perilesional enhancement. The

largest lesion measured 3.5 x 1.5 cm, accompanied by perilesional edema. Laboratory investigations revealed an absolute eosinophil count of 3,500 cells/cm³, and a serum IgG Echinococcal antigen level of 2.19. The patient was advised to undergo surgical excision of presumptive hydatid cysts. Still, the patient did not consent to this, so a trial of watchful medical management with Tab Albendazole 400 mg BD for 42 days was planned. [Figure 6]

On follow-up after 6 weeks, repeat imaging was suggestive of a partial reduction in the size of the cysts and perilesional edema. The patient was advised to complete a three-month treatment. On a 6-month follow-up, the patient is seizure-free and without any deficit.

Discussion

We present these cases as atypical due to their locations and presentations. The cases were managed on an individual basis. Hydatid cyst is a zoonotic disease caused by the ingestion of the larval stage of Echinococcus granulosus and Echinococcus multilocularis, also commonly known as the dog tapeworm, in humans. It is accidentally introduced through exposure to an infected dog, which serves as a definitive host, or by ingestion of contaminated water or vegetables [1]. Primary cysts are usually single in number and fertile. Secondary cysts are generally multiple in number and sterile in terms of causing recurrence [3, 4]. The primary hydatid cyst in the brain can be attributed to immune malfunction or cardiac abnormalities, such as patent ductus arteriosus and patent foramen ovale.

Among the various identified genotypes of *Echinococcus granulosus*, certain forms of the organism have a greater affinity for brain tissue, and MHC class II variations also lead to increased and variable susceptibility to the organism [4, 5]. 75% of hydatid cysts occur in the pediatric population, and males have a higher predilection than females [5].In the present series, all patients were adults, comprising three males and one female. Most reported cases had solitary occurrences, with multiple cysts being a rare occurrence. Intraparenchymal cysts in the supratentorial region are common and predominantly located in the territory of the middle cerebral artery, due to the disease's embolic spread [6–8]. The calvarium, cavernous sinus, extradural region, aqueduct, ventricles, pons, and cerebellum are other less commonly involved areas of the brain [9]. The locations of the hydatid cysts were in the watershed region of the MCA-PCA territory in the first, second, and fourth cases, and at the craniovertebral junction in the third patient, who is an uncommonly reported case.

Hydatid cysts are relatively rare in the brain, occurring in only 1–2% of total cases. Brain hydatid cysts, as previously discussed, can be either primary or secondary. Primary cysts have scolices and brood capsules. Therefore, they result in recurrence when they rupture. Secondary multiple cysts usually occur due to spontaneous or traumatic, or iatrogenic rupture of the primary cysts. Secondary cysts do not have a brood capsule or scolices. Hence, they are sterile, and their rupture does not result in recurrence. Multiple primary cysts are rare, and only isolated cases have been reported thus far in the current literature [6, 10]. In our series, the 3rd and 4th patients had multiple cysts. Their radiological

characteristics were suggestive of secondary hydatid cyst, but the exact etiology could not be established in these patients.

The cyst wall consists of an inner germinal layer, called the endocyst, and an outer laminar layer, called the ectocyst. Due to the host reaction to the cyst wall, a fibrous capsule forms around the cyst, providing nutrition to the cyst, known as the pericyst. In the brain, the pericyst is very thin due to little tissue reaction [11].

Patients are usually asymptomatic or present with nonspecific symptoms, as cyst growth is slow; therefore, they typically present with compressive symptoms in the areas involved due to the increase. Common signs and symptoms include headache, vomiting, and papilledema. However, any symptom resulting from raised intracranial pressure can be observed. Focal symptoms, such as limb weakness, hemiparesis, epileptiform disorder, gait disturbances, or visual problems, may be present depending on the region or size of the cyst involved. The diagnosis mainly depends on clinical suspicion, particularly in individuals with a history of living in or visiting a recent endemic area. [12, 13]

In cases of cerebral hydatid disease, serological tests often yield false-negative results, making imaging evaluations, such as CT and MRI scans, more reliable. These scans usually show a well-defined spherical cystic lesion, smooth, thin-walled, with or without septation or calcification. The cyst wall is isodense or hyperdense on NCCT, showing a rim of low intensity on T1- and T2-weighted images. Daughter cysts, if and when present, are considered pathognomonic of hydatid disease, but they are very rarely seen. Wall calcification is reported to be seen in less than 1% of cerebral hydatid cyst cases. Mass effect can be seen as compression of midline structures and the ventricles, and it is a common finding, but peripheral edema and cyst rim enhancement are usually absent in uncomplicated cases, and it was observed in two of our patients (3rd and 4th patients). [2, 14].

Differential diagnosis of CCE cysts with other space-occupying lesions includes cysts-like masses of other parasitic origin, with different indices of suspicion depending on the epidemiological context and history (Tania solium causing neurocysticercosis and Echinococcus multilocularis causing alveolar echinococcosis), arachnoid cysts, porencephaly, cystic astrocytoma and other cystic tumors, and brain abscess [15, 16]. As we delved into the available literature, it became evident that intracranial hydatid cysts are primarily encountered in other locations, such as the cerebral hemispheres and supratentorial spaces. Their occurrence within the thalamus, a region deep within the brain, is genuinely extraordinary and often results in non-specific clinical symptoms due to its concealed location.

Surgery is the mainstay treatment of cerebral hydatid cysts. The primary goal of the surgery is to completely remove the cyst without rupturing it or its contents. The Dowling technique, which Arana-Iniguez and San Julian modified, is widely used in the surgical treatment of hydatid cysts of the central nervous system [17]. In both of our patients who underwent surgery, the Dowling technique was employed for flushing the cysts from their tissue bed and out of the brain. Unfortunately, the patient with a thalamic cyst had an accidental rupture. In contrast, the cyst was extirpated; however, in the case of the patient with a retrocerebellar cyst extending into the cervical spinal cord, the cysts were removed

successfully. Recent literature with large cases also advocates similar management of surgical removal [18].

Surgery should be followed by albendazole as a part of chemotherapy in the medical part of the treatment to avoid recurrence. Isolated case reports have shown that near complete disappearance in cases with multiple intracranial hydatid cysts is possible with the use of albendazole therapy, given with a daily dose of 10 mg/kg, taken three times a day for 4 months. In cases 1 and 4 of this article, medical therapy alone resulted in almost 90% reduction of the cyst size. Preoperative and postoperative albendazole treatment may be considered to sterilize the cyst, decrease the risk of anaphylaxis, reduce tension in the cyst wall (thus minimizing spillage during surgery), and lower the recurrence rate [19].

In our case, the postoperative administration of albendazole in both Case 2 and Case 3 patients aligns with established therapeutic protocols aimed at preventing recurrence or dissemination of the parasitic infection. The successful outcome of the postoperative course underscores the significance of meticulous surgical technique and appropriate postoperative care in achieving a favourable prognosis.

A recently published systematic review by Jaradat et al [20] on cerebellar hydatid cysts also shows no documented report of any retro cerebellar midline hydatid cyst with dissemination into cervical spinal cord and presenting with quadriparesis and this is the only documented report of the pathology. All the cases had atypical presentation in one or the other way and this makes this small constellation of hydatidosis one of its kind. All the cases were managed with good outcomes and no recurrence was reported on follow-up in any of the four cases. It also highlights the role of medical management with regular interval observation in resolving the disease despite major emphasis on surgical management.

Conclusion

Intracranial hydatidosis is a rare but important differential diagnosis in patients from endemic areas presenting with space-occupying brain lesions. The disease may present with atypical clinical and radiological features, necessitating a high index of suspicion. Surgical excision remains the mainstay of treatment, but medical therapy has a significant role, particularly in inoperable or multiple cysts. The cases presented herein exemplify the diverse manifestations of cerebral hydatidosis and highlight the importance of individualized, multidisciplinary management to achieve favorable outcomes.

Declarations

Ethics approval and consent to participate – not required due to restrospective nature and as patients were not treated as part of any trials.

Consent for publication - participants/patients gave written informed consent for their personal or clinical details along with de-identified images to be published in this study

Availability of data and materials - yes

Competing interests – Rahul Jain is an editorial board member of BMC Neurology editorial board.

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Authors' contributions – All authors contributed equally to the drafting and preparation of the manuscript

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Author Contribution

RJ, VCJ, VS, AR- design of the work;NK, RJ, AA, VS, GV -the acquisition, interpretation of data; RJ, AA, AR, VCJ, VS have drafted the work and substantively revised itall authors have approved the submitted version (and any substantially modified version that involves the author's contribution to the study)

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Figures

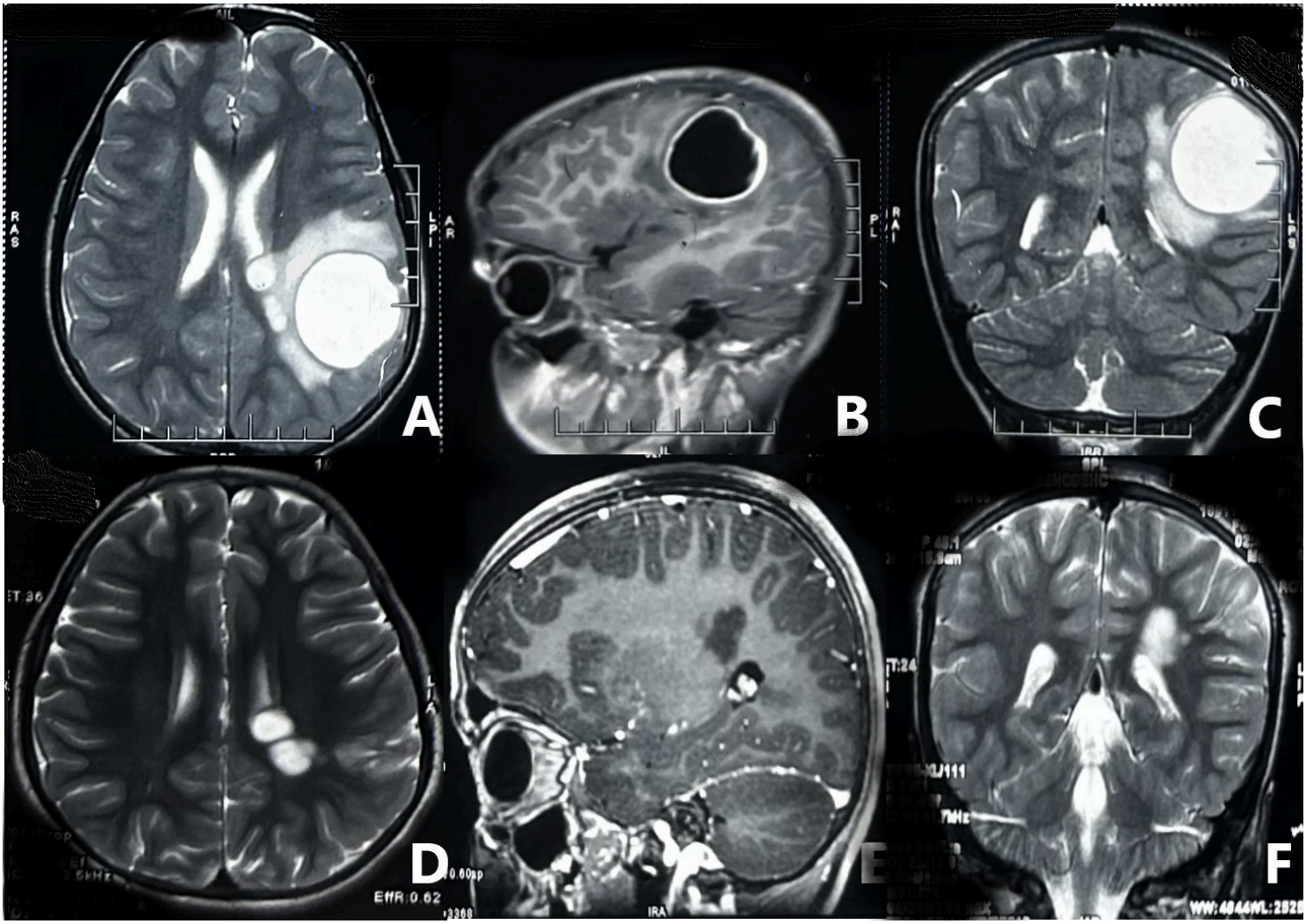


Figure 1

A – axial T2W image showing well-defined interaxial hyperintense lesion in left parietal lobe measuring 4.5x4.3x4.2 cm with surrounding vasogenic edema

Figure 1B – sagittal T1W gadolinium-enhanced image showing well-defined intraaxial lesion in left parietal lobe with peripheral rim enhancement of the lesion

Figure 1C – coronal T2W image showing well-defined interaxial hyperintense lesion in left parietal lobe measuring 4.5x4.3x4.2 cm with peripheral edema causing effacement of the occipital horn of the left lateral ventricle

Figure 1D- axial T2W image showing hyperintensity changes in peritrigonal white matter of left parietal lobe adjacent to occipital horn of left lateral ventricle suggesting residual edema.

Figure 1E – sagittal T1W gadolinium-enhanced sagittal MRI brain image image showing that the lesion has reduced in size and appears hypointense without any contrast enhancement

Figure 1F –T2 coronal MRI brain image showing a residual small hyperintense lesion in the left parietal lobe with mild surrounding vasogenic edema abutting the wall of the occipital horn of the left lateral ventricle.

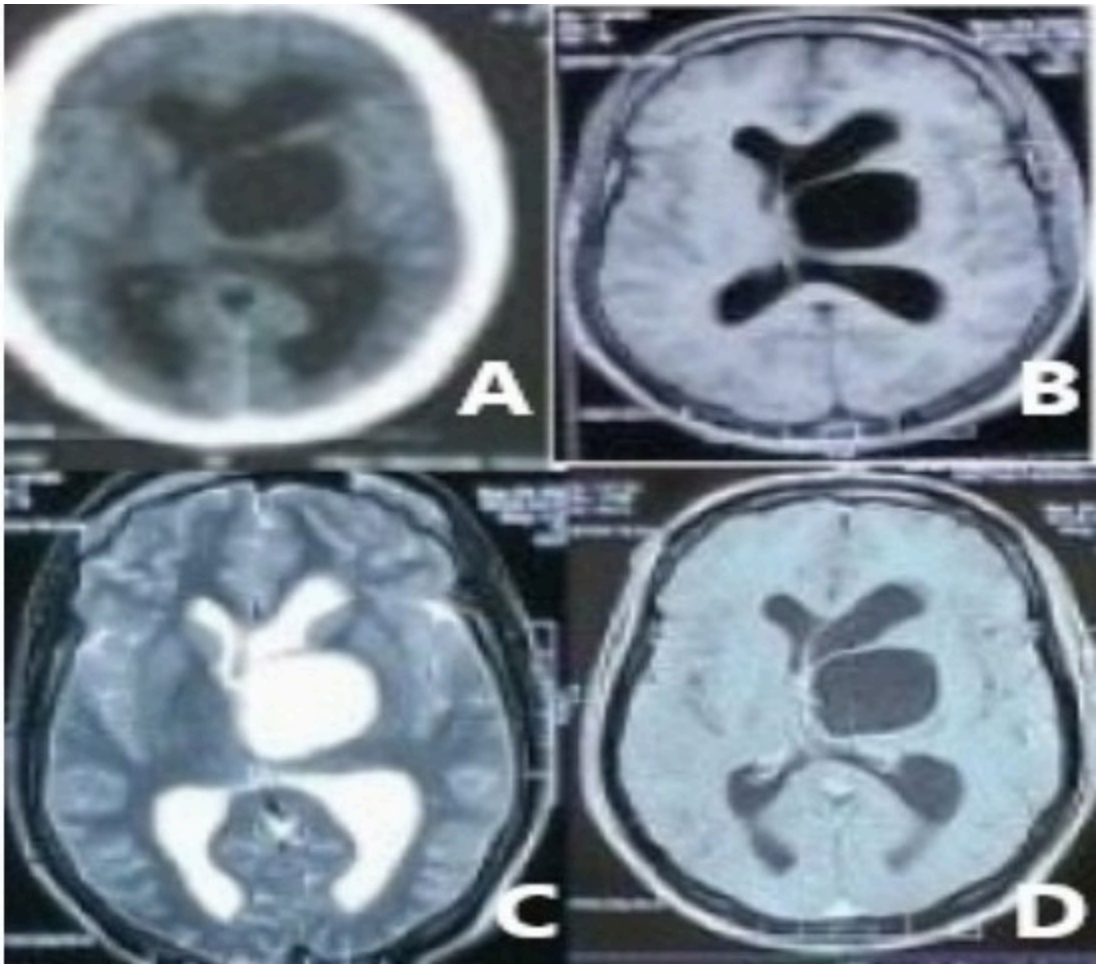


Figure 2

2A—The NCCT head reveals a well-defined hypodense cystic mass in the left thalamic region abutting the wall of the body of the left lateral ventricle, resulting in a mass effect.

Figure 2B – axial T1W image showing well-defined interaxial hypointense lesion in the left thalamus.

Figure 2C—The axial T2W MRI brain image reveals a hyperintense, well-defined cystic mass in the left thalamus without perilesional edema.

Figure 2D – axial MRI brain with gadolinium-enhanced image showing well-defined cystic mass lesion in left thalamus without any peripheral contrast enhancement of wall.

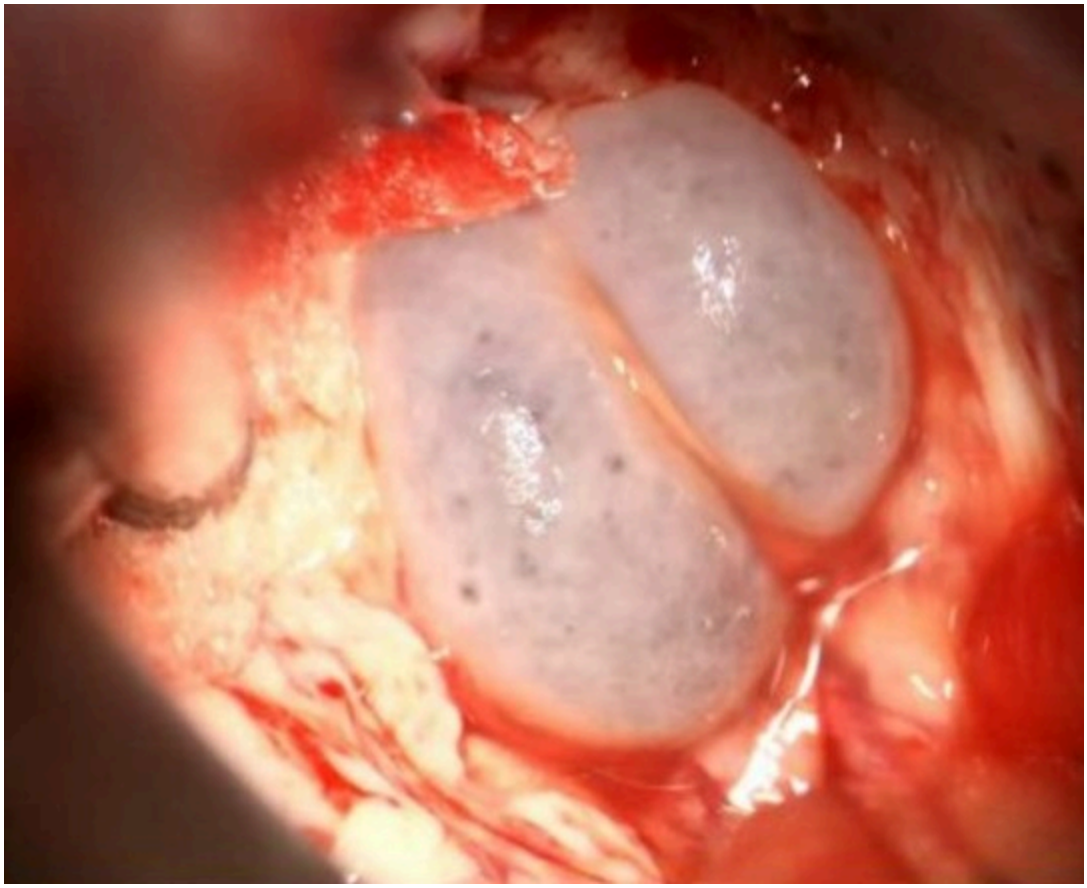


Figure 3

Intraoperative image of case 2 showing an interaxial cystic lesion in the left thalamus surrounded by cottonoids with a thin rim of brain parenchyma over the lesion.

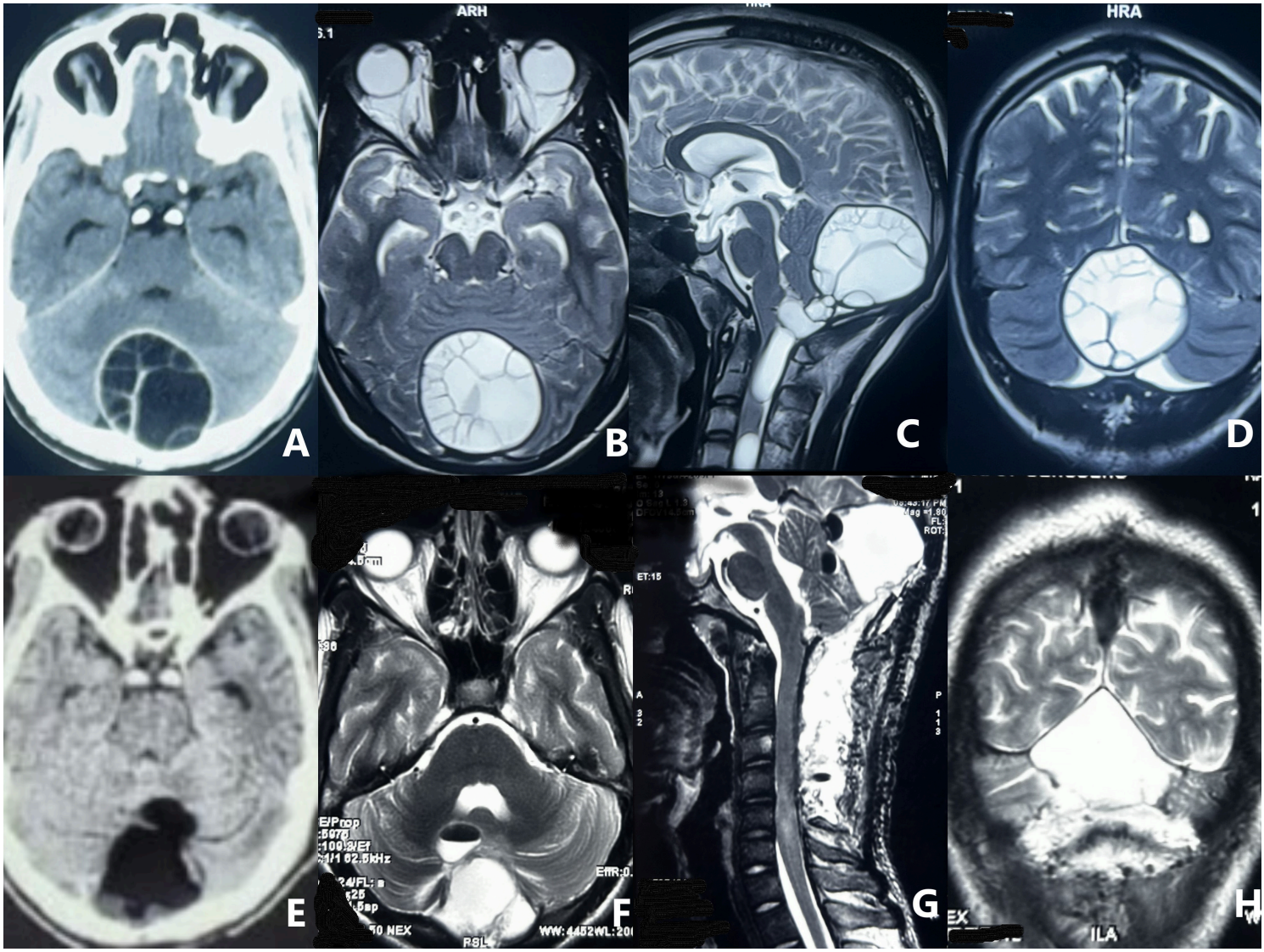


Figure 4

4A- NCCT head image showing well-defined cystic retro cerebellar lesion with multiple isodense to hyperdense thick septations within the lesion with peripheral hyperdense walls

Figure 4B – axial T2W MRI of brain image showing well-defined hyperintense lesion of size 5.1x5 cm in the retro cerebellar region with multiple septations and loculations causing cerebellar hemisphere compression bilaterally

Figure 4C—sagittal T2W MRI brain image showing a well-defined hyperintense lesion in a retrocerebellar location with multiple septations and loculations. The cystic lesion extends inferiorly into the upper cervical spinal cord. The lesion compresses the brainstem and cervical spinal cord anteriorly as well as the distal superior sagittal sinus and torcula herophili, pushing them superiorly.

Figure 4D—coronal T2W MRI brain image showing an infratentorial well-defined hyperintense lesion in the retro cerebellar location cantered in the midline, causing bilateral cerebellar hemisphere compression.

Figure 4E—Post-operative non-contrast computed tomography (NCCT) head axial images showing complete removal of the retrocerebellar lesion with a large air cavity and surrounding hypodensity in the posterior fossa measuring 4.2 X 3.5 cm.

Figure 4F—Postoperative axial T2 brain MRI image showing a postoperative cavity of size 31 x 43 x 45 mm with an air-fluid level, following CSF signal.

Figure 4G—Postoperative sagittal T2W image showing laminectomy up to C4 level and complete removal of cystic lesion of retrocerellar region and cervical spinal cord. Myelomalacic changes can be appreciated in the cervical spinal cord at the C4 and C5 levels.

Figure 4H – Post-operative coronal T2W image showing residual cavity post cyst excision.

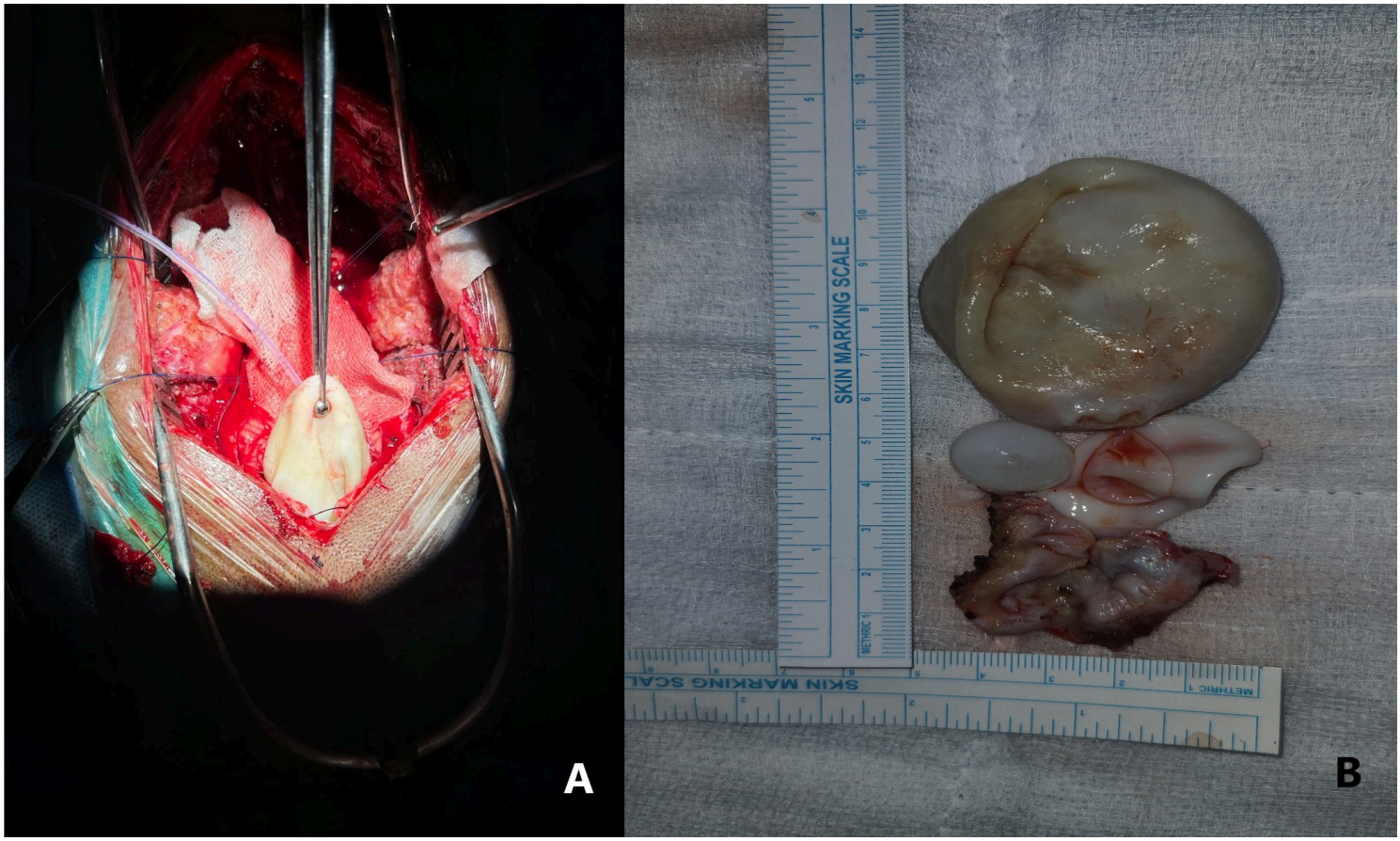


Figure 5

5A—Intraoperative image of case 3 showing manual exenteration of the retro cerebellar cyst with forceps in addition to the Dowling technique of cyst removal.

Figure 5B—Multiple hydatid cysts after removal from the retrocerebellar and intradural extramedullary cervical spinal cord locations, with the largest being a retrocerebellar cyst measuring almost 5x5 cm in size.

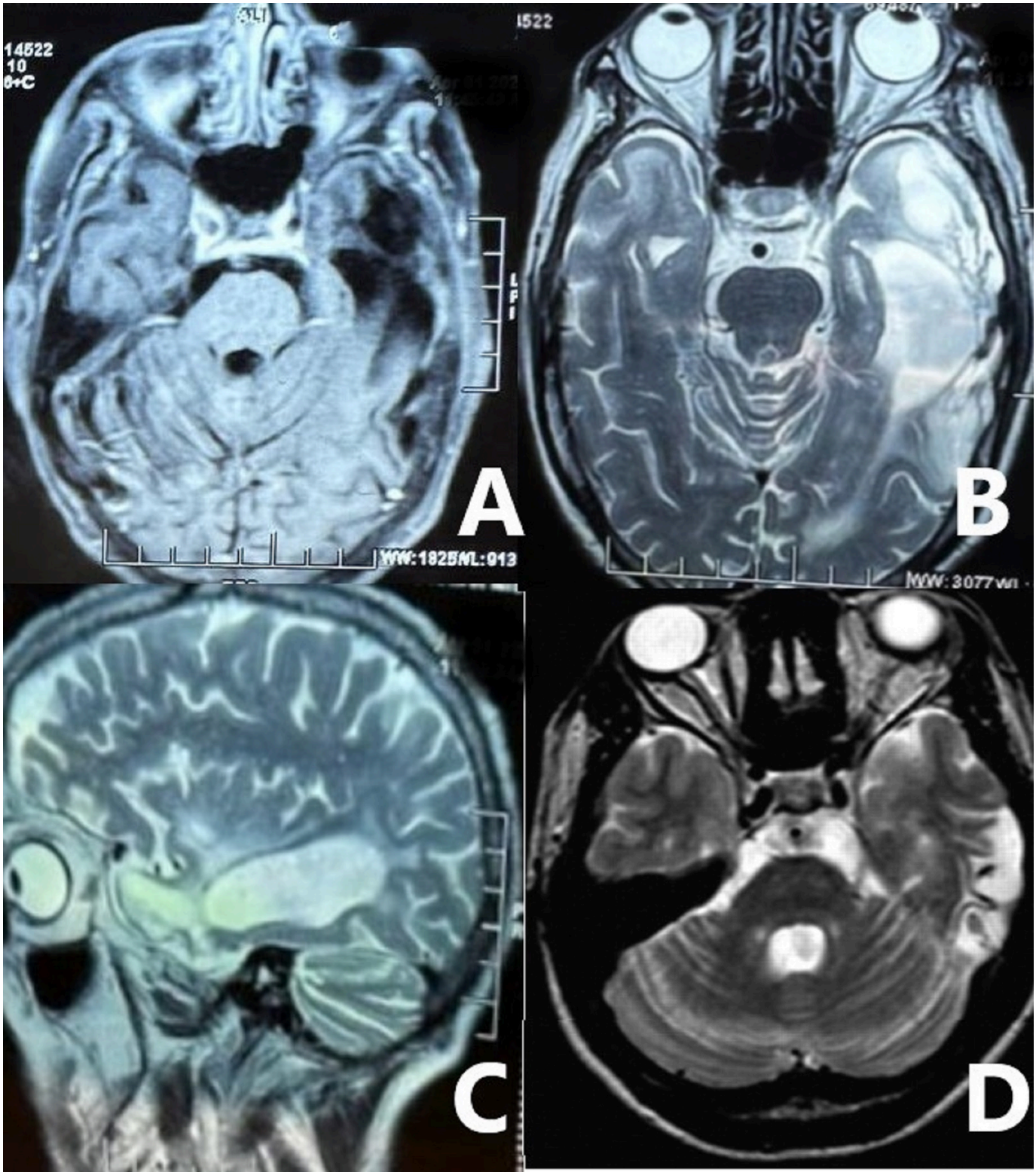


Figure 6

6A – MRI with contrast T1W axial view of brain suggestive of hypointense multiple cystic lesions in left temporal lobe without peripheral rim enhancement with perilesional edema.

Figure 6B – MRI brain T2W axial view suggestive of multiple hyperintense cystic lesions in left temporal lobe with perilesional edema causing effacement of left temporal horn.

Figure 6C - MRI brain T2W sagittal view suggestive of hyperintense cystic lesion in left temporal lobe with perilesional edema measuring 3.5x1.5 cm.

Figure 6D - MRI brain T1W axial view done at 6 months follow-up showing resolution of cysts in left temporal lobe with residual gliosis.