

## Case Report

### A case of intracranial epidermoid cyst at basal cisterns

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#### Abstract

The radiological features of intracranial epidermoid cyst involving basal cistern in a young woman on Computed tomography (CT) and Magnetic resonance imaging (MRI) are discussed. Extensions of the tumor and its mass effect on adjacent structures and cranial nerves in the patients are discussed. Role of imaging in diagnosis of intracranial epidermoid cyst and differentiating points from other similar intracranial cysts are discussed.

**Keywords:** Intracranial epidermoid cyst, Computed tomography, Magnetic resonance imaging.

#### Introduction

Epidermoid cysts are congenital inclusion cysts which are benign and slow growing extra-axial tumors. These account for ~1% of all central nervous system tumours.<sup>1,2,3</sup> They develop due to desquamation of epithelial cells at the time of closure of neural tube from 3<sup>rd</sup> to 5<sup>th</sup> week of embryogenesis. Therefore, these lesions are composed of ectodermal elements like cholesterol crystals and keratin.<sup>1</sup>

Intracranially, these cysts are most commonly seen in the cerebellopontine angle (40-50%), followed by fourth ventricle and parasellar

region. Other rare locations include interhemispheric fissure, calvarium and intraparenchymal regions.<sup>2,3</sup>

On radiological imaging, they are seen as well defined, lobulated, cystic lesions of cerebrospinal fluid (CSF) density appearing hypodense on computed tomography (CT); hypointense on T1-weighted images and hyperintense on T2/FLAIR-weighted images on magnetic resonance (MR) imaging.

Here, we report a case of intracranial epidermoid cyst and discuss its salient radiological features to diagnose the condition.

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#### Case History

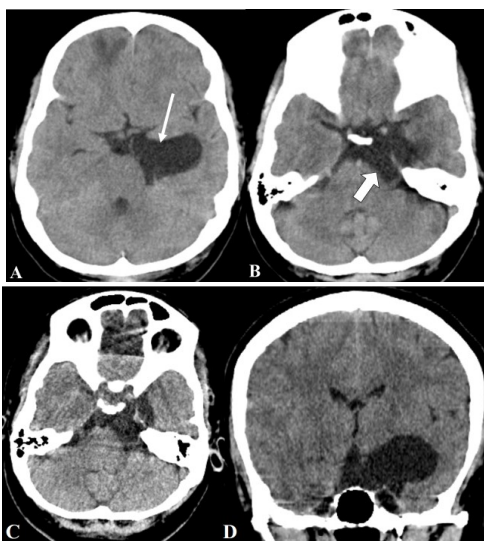
A 38-year-old female patient was referred to radiology department for CT scan of brain following road traffic accident to rule out for any post-traumatic bleeds and injuries. Incidentally a cystic lesion arising from basal cisterns on the left side was noted. There was past history of intermittent type of headache and nausea for last 4 months. No previous history of seizures, trauma or surgery.

## Imaging findings

### Plain CT Brain

On plain CT scan of brain there was a fairly well defined extra-axial cystic lesion of CSF density arising from left crural cistern (Figure 1A) with extension upto suprasellar, peri-mesencephalic, cerebellopontine angle and prepontine cisterns (Figure 1B, C & D).

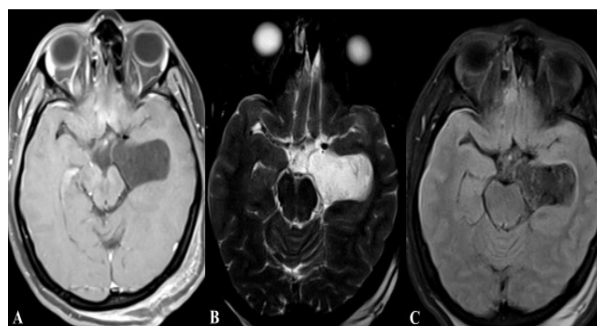
The differential diagnosis of epidermoid cyst, arachnoid cyst or porencephalic cyst was considered. Contrast enhanced MRI brain was suggested for further evaluation.



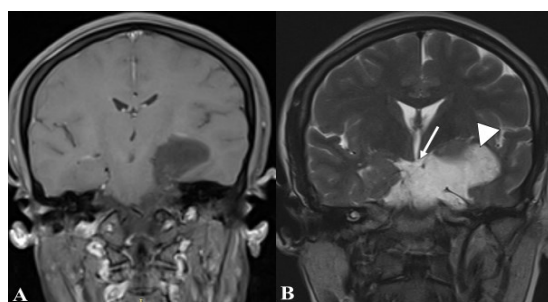
**Figure 1:** (A) Plain CT scan brain, axial section at the level of temporal horn of lateral ventricle shows a fairly well defined extra-axial (white line arrow) CSF density lesion noted arising from left crural cistern. (B, C) Plain CT scan brain, axial section at basal cisterns show an extension of the lesion into suprasellar, peri-mesencephalic region, cerebellopontine angle and prepontine cisterns (Thick white arrow). Laterally, the lesion is causing mass effect in the form of compression of temporal horn of lateral ventricle and medial temporal lobe. (D) CT brain, coronal section extension of the lesion to suprasellar cistern.

### MRI brain (Plain and contrast enhanced)

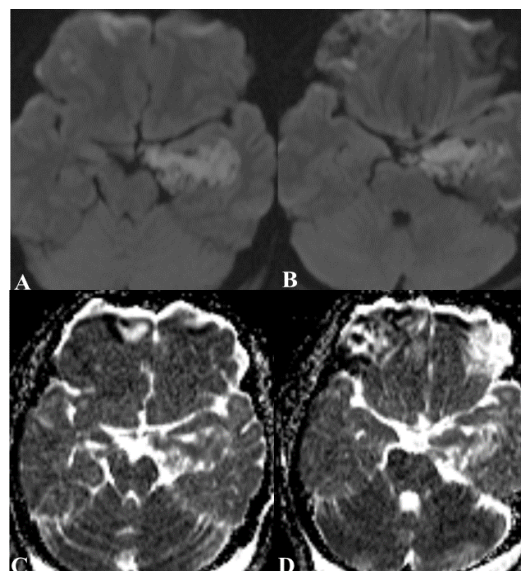
MRI brain showed a well-defined non-enhancing T1 hypointense and T2 heterogeneously hyperintense extra-axial lesion involving basal cisterns (crural) on left side (Figure 2). The lesion is seen extending into suprasellar cistern and sella (Figure 3). The lesion demonstrated restricted diffusion on DWI (Figure 4). No blooming on SWI was noted (Figure 5).



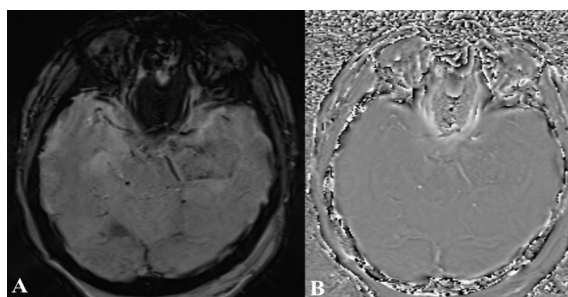
**Figure 2:** (A) A well-defined T1 hypointense and (B) T2 hyperintense extra-axial lesion involving basal cisterns on left side (C) The lesion is hypointense on FLAIR image.



**Figure 3:** (A) Coronal T1 & (B) T2 weighted images showing extension of the lesion to suprasellar cistern and sella displacing optic chiasma medially (line arrow).

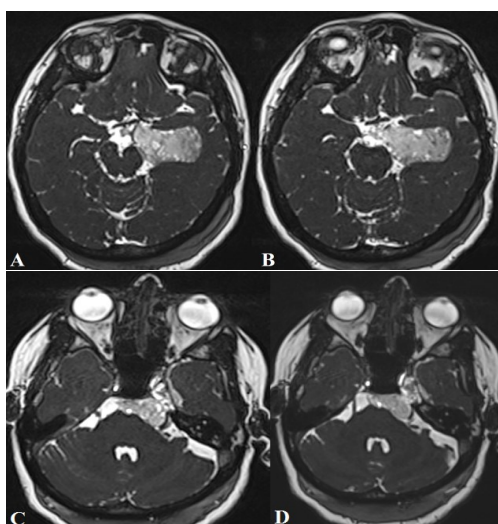


**Figure 4:** (A, B) The lesion appears hyperintense on DWI. (C, D) Corresponding ADC sequences show restricted diffusion.



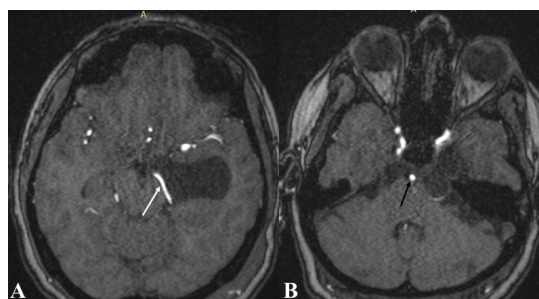
**Figure 5: (A) SWI & (B) corresponding Phase shows no blooming focus within the lesion.**

Anteriorly, the lesion was extending to suprasellar cistern and sella displacing optic chiasma medially (Figure 6A). It is indenting C6 & C7 segment of left internal carotid artery with arc of contact ~ 90°; however show normal flow (Figure 6A & B). It was extending to cavernous sinus and Meckel's cave and displacing the trigeminal nerve laterally. Medially, there was extension into peri-mesencephalic cistern, cerebello-pontine angle and pre-pontine cisterns and was causing compression and displacement of left cerebral peduncle, midbrain & pons; however no signal intensity changes was noted (Figure 6C & D). Oculomotor nerve and trochlear nerves were displaced medially and abducent nerve was seen displaced laterally. No obvious thickening or enhancement of the cranial nerves was noted. Laterally, the lesion was causing mass effect in the form of compression of temporal horn of lateral ventricle and medial temporal lobe (Figure 1A, 3 & 6A-B). Posteriorly, it was abutting basilar artery and left posterior communicating artery (Figure 7A-B).



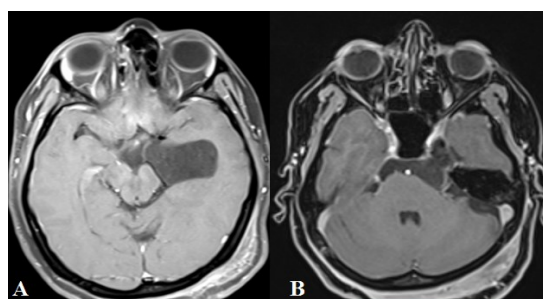
**Figure 6: (A-B) Extension to perimesencephalic cistern and is displacing Oculomotor nerve and trochlear nerves are displaced medially. (C-D) It is**

extending to cerebello-pontine angle cistern, pre-pontine cistern, cavernous sinus and Meckel's cave causing displacement of the trigeminal nerve and abducent nerves laterally. The lesion is abutting the basilar artery in prepontine cistern.



**Figure 7: The lesion is abutting left posterior communicating artery (white arrow) and basilar artery (black arrow) posteriorly.**

On contrast enhanced MRI, the lesion was showing no enhancement (Figure 8A-B)



**Figure 8: (A & B) The lesion is not enhancing on T1 fat saturated post-contrast images.**

## Diagnosis

The above features on contrast enhanced MRI brain are suggestive of intracranial epidermoid cyst with its local extensions and mass effect on adjacent cranial nerves and vessels.

Patient was operated and the sample was sent for histopathological examination.

Specimen was sent for histopathological examination and the diagnosis of epidermoid cyst was made on cytology; hence proving CT and MRI findings as discussed above.

## Discussion

Intracranial epidermoid cysts are congenital tumors and typically slow growing. Most patients with epidermoid cysts are asymptomatic at clinical presentation, although patient can rarely have headache, cranial nerve defects or cerebellar symptoms. The current patient has past history of few

episodes of headache. However she was diagnosed of intracranial epidermoid cyst incidentally after performing a CT brain following road traffic accident.

The anatomical locations of the epidermoid cysts are epidermis, intracranium, spleen, spinal cord and testis. Of all the intracranial tumors, epidermoid cysts constitute ~ 1%.

These lesions are usually well-circumscribed, congenital inclusion cystic tumors of CSF density as we seen in this case. They are also known as “pearly tumors”. The patients usually become symptomatic after second decade of their life, most commonly between 4<sup>th</sup>- 5<sup>th</sup> decade of the life. These lesions have male predominance. These cysts are rarely acquired secondary to surgery or post-traumatic implantation.<sup>1,2,3</sup>

Sellar/ suprasellar lesions usually clinically present with visual disturbances and nonspecific headache. Symptoms are mainly due to mass effect or compression of cranial nerves. Hydrocephalus is a very rare and late manifestation.

The cysts can be intradural or extradural in location. Intracranially, they are most commonly seen in cerebellopontine angle constituting for ~ 40-50%. Approximately 10% of the lesions are seen in suprasellar cistern. They can also be seen in ventricular system (most common fourth ventricle), basal cisterns as in our patient. Other locations are brain parenchyma, interhemispheric fissure and pineal region. Extradural lesions are intradiploic which usually causes focal cranial vault thinning or defect.<sup>3</sup>

Lesions have a very thin outer capsule and fluid content within.<sup>2,3</sup> Above 10% cysts show calcifications, most commonly interhemispheric epidermoids.<sup>4</sup> Most of the lesions are subdural in location. Few of the lesions are well-circumscribed round or ovoid lesions seen in the epidural space.<sup>4,5</sup>

Capsule of the cyst is composed of stratified squamous epithelium tissue because of which the cystic fluid is composed of keratin and cholesterol in a solid crystalline state. Large lesions are prone to rupture because of its thin capsule.<sup>1,6,7</sup> Ruptured cysts are prone to spread faster.<sup>6</sup> Recurrence of these ruptured tumors after resection can occur; however their recurrence rate is extremely low.<sup>8</sup>

The super infection of epidermoid cyst is rare. If infected, attribution of its communication with paranasal sinuses and the inflammatory reaction should be considered resulting in its superinfection.

### **CT findings**

Radiologically epidermoid cysts present as hypodense cystic lesions of CSF density and can pose

a diagnostic challenge on CT like in our case.<sup>3</sup> It is sometimes difficult to differentiate epidermoid cyst and radiological imaging remains the sole preoperational diagnostic investigation. However, MRI plays a significant role in diagnosing the condition. Differentials for intracranial epidermoid cysts are arachnoid cysts, mega cisterna magna, dermoid cyst, ratke's cleft cyst, and cystic tumors like acoustic schwannoma, neurenteric cyst and rarely neurocysticercosis.<sup>5</sup>

On imaging intracranial epidermoid cysts are usually capsulated irregular lesions and when large they are seen encasing adjacent neurovascular structures. On CT, these lesions are seen as homogeneously hypodense non-enhancing cystic lesions of cerebrospinal fluid density (CSF) density. The cellular debris along with high cholesterol content of the fluid lowers the density of cysts to approximately 0 Hounsfield units (HU).<sup>1,9</sup>

### **MRI findings**

On MRI, epidermoid cysts are isointense to CSF on T1 & T2 weighted images, heterogenous on FLAIR images and lesions do not restrict on diffusion weighted images. Typical cysts do not enhance on post-contrast study as capsule is avascular.<sup>1,2,3,4</sup> However, few lesions show thin enhancement around the periphery. Enhancement is seen in infected epidermoid cyst and it is pronounced in case of malignant degeneration.<sup>4,9</sup>

### **Differential diagnosis**

A rare phenomenon called “white epidermoid” where the cysts appear hyperdense on CT, hyperintense on T1 weighted images, hyper to hypointense on T2 weighted images, secondary to hemorrhage, high protein content of fluid or saponification within the cyst.<sup>6,10</sup>

Epidermoid cysts are differentiated from other intracranial cysts lesions as follows:

Dermoid cysts are well defined lobulated masses of low attenuation of fat showing occasional wall calcification and thin rim of enhancement is seen.<sup>5,11</sup> No calcifications or enhancement of the wall was seen in our case. Whereas epidermoid cysts show restricted diffusion as in our case.

Similarly, suprasellar arachnoid cysts mimic epidermoid cysts and both being congenital with CSF signal intensity on CT and all sequence of MRI; suprasellar cisterns show no restriction on DWI.<sup>11</sup>

Rathke cleft cysts follow CSF signal intensity on CT and MRI; however they can rarely appear hyperintense on T1 due to high protein content. They



usually show no restriction on diffusion weighted MRI. They are believed to occur due to failure of obliteration of the lumen of Rathke pouch.<sup>6</sup>

Other cystic tumors like acoustic schwannoma or craniopharyngioma may look similar; however they have a solid enhancing component within which is usually identifiable.<sup>4,6,11</sup>

### Treatment

Complete surgical excision is the treatment of choice with consistent good excellent long-term results being reported in various series. However, as portions of the capsule may become densely adherent to surrounding structures, total resection could be extremely difficult and subtotal resection may have to be performed in such cases to prevent neurological damage. Recurrences are uncommon and mostly seen after subtotal resections.

### Conclusion

Imaging plays a vital role in diagnosing intracranial epidermoid cyst pre-operatively. The lesion is a well-defined cystic lesion of cerebrospinal fluid density on CT and all sequences of MRI showing restricted diffusion on DWI. No calcifications/post-contrast enhancement is seen in them.

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