

Prepontine and Meckel's Cave Dermoid Cyst: MR and CT Findings with Literature Review

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ABSTRACT

Dermoid cysts (DCs) are benign, congenital tumors that comprise 0.04-0.6% of all intracranial tumors. DC rupture is a rare complication and usually occurs spontaneously. The most common localisations of intracranial DCs are the posterior fossa, and suprasellar and parasellar regions. The presentations of DCs are highly variable. They are often detected incidentally on computed tomography (CT) or magnetic resonance imaging (MRI) scans while investigating the cause of seizure or headache. Prepontine cistern is a rare localisation for intracranial DCs. To the best of our knowledge, only four cases have been reported in the literature so far. We present MRI and CT findings of a patient with DC, which ruptured into the subarachnoid space extending from the right Meckel's cave to the prepontine cistern.

Key Words: *Dermoid cysts, Meckel's cave, Prepontine cistern, Rupture.*

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INTRODUCTION

Dermoid cysts (DCs) are benign, congenital tumors that comprise 0.04-0.6% of all intracranial tumors. Dermoid and epidermoid cysts are both thought to originate from ectodermal inclusions due to the closing defect of the neural tube between the third and fifth weeks of embryonic life.¹ Accordingly, they are located either at the caudal part of the neuroaxis or close to the midline.² Intracranial DCs are most commonly located in the posterior fossa, suprasellar and parasellar regions.

DCs are considered to have a spectrum ranging from the epidermoid cysts, which contain only squamous epithelium to teratomas that contain all three layers of the embryonic tissue. Since they originate from the epidermis and dermis, they contain sebaceous glands, sweat glands, hair follicles, and dermal elements such as teeth and nails.³ These lesions grow slowly depending on the hair and oil production of the internal dermal elements.⁴

The presentations of DCs are highly variable. They are often detected incidentally on computed tomography (CT) or magnetic resonance imaging (MRI) scans, while investigating the cause of a seizure or headache.

DC rupture is rare and usually occurs spontaneously. As the cyst contents spread to the subarachnoid space because of rupture, aseptic chemical meningitis, temporary cerebral ischemia due to vasospasm, hemiparesis, hydrocephalus; and even death may occur.³

In this case report, we present MRI and CT findings of a patient with DC, which ruptured into the subarachnoid space extending from the right Meckel's cave to the prepontine cistern.

CASE REPORT

A 37-year male patient was admitted to the Neurology Clinic with a headache complaint that has been ongoing for 15 days. There was no neurological deficit on physical examination. The patient's CT revealed a bilobular, hypodense lesion with a tiny calcific focus filling the Meckel's cave and extending to the prepontine cistern (Figure 1a). Moreover, low-density areas compatible with scattered oil droplets were present in subarachnoid space, in the bilateral frontal and occipital lobes, right posterior fossa, and right quadrigeminal cistern (Figure 1b). The lesion's average attenuation was measured between -13 and -88 Hounsfield units (Figure 1c).

MRI scan was performed for a more detailed lesion evaluation, and the lesion was seen to extend from the Meckel's cave to the prepontine cistern with a slight right anterolateral indentation to the pons. It was hyperintense on T1-weighted images (Figure 2a) and heterogeneously hyperintense on T2-weighted images with chemical shift artifact (Figure 2b). The lesion did not enhance after intravenous gadolinium injection and did not show restricted diffusion. Small hyperintense foci were detected in subarachnoid space, compatible with free oil droplets seen in ruptured DCs, in the T1-weighted images. Although the lesion showed proximity to the cavernous segment of the right internal carotid artery and right

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superior cerebellar artery, there was no stenosis or compression effect. The cisternal part of the right trigeminal nerve was pushed laterally in the prepontine cistern by the lesion, but the trigeminal ganglion could not be visualised separately in the Meckel's cave because of the mass effect. However, the patient did not have any complaints indicating trigeminal nerve function.

In this particular case, conservative treatment was chosen instead of surgery because of the patient's mild clinical condition and DC's location. The patient had mild headache during the last two years. The patient's follow-up interval was three months in the beginning, and the follow-up interval was extended to six months because there was no change in physical and radiological examinations (Figure 2c).

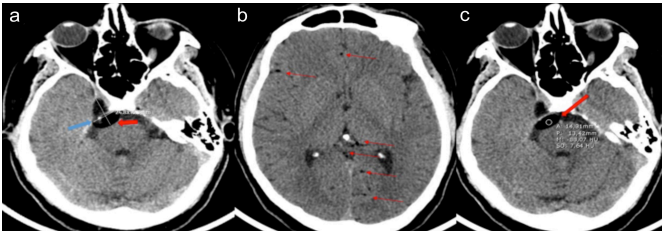


Figure 1: Non-contrast axial CT image (a) demonstrates the bilobular hypodense lesion (red arrow), which fills the Meckel's cave and extends to the prepontine cistern with a tiny calcification in the centre (blue arrow), (b) shows low-density areas compatible with scattered oil droplets observed in the subarachnoid space, in the right frontal, left occipital, quadrigeminal cistern level and interhemispheric fissure (thin red arrows), (c) shows the average attenuation of the lesion (red arrow) measuring between -13 and -88 Hounsfield units.

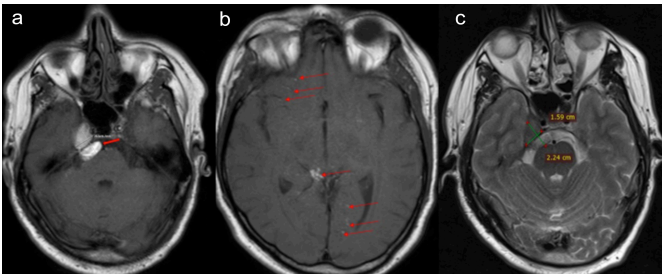


Figure 2: (a) Axial T1-weighted image shows hyperintense lesion from the Meckel's cave to the prepontine cistern, with a slight right anterolateral indentation to the pons (red arrow), (b) Axial T1-weighted image demonstrates tiny hyperintense foci detected in the subarachnoid space compatible with free oil droplets with ruptured dermoid cyst (thin red arrows), (c) Axial T2-weighted follow up image (final examination) shows no difference after two years.

DISCUSSION

DCs consist of 0.04-0.6% of all brain tumors.⁵ These are benign tumors and tend to grow slowly. Due to the compression of neurovascular structures, these may cause focal neurological findings. Although rare, DCs may rupture.⁶ Rupture is usually spontaneous, less frequent and may occur secondary to a head injury.¹ In cases of rupture, headache, seizures, change in consciousness, aseptic chemical meningitis, stroke secondary to vasospasm, hydrocephalus, increased intracranial pressure, or granulomatous arachnoiditis may occur.²

In some patients, progressive neurological symptoms due to growing mass effect of DCs have been reported before the rupture.

The DC originates from the ectopic epithelial cells, which are components of the neural tube, and indicates the characteristic location of the cyst, the sellar-parasellar region, and the posterior fossa.^{1,6} Although the first cases in the reported literature had infratentorial localisations such as cerebellar vermis and the 4th ventricle neighborhood, there is an increased propensity for supratentorial localisation in recent case reports. There are just a few cases in the prepontine cistern, and this case is the 5th case with this rare localisation.⁷

DCs are not true neoplasms. They include a cystic cavity that is filled with accumulation of desquamated products and sebaceous secretions instead of cell division, and these features are essential for their distinction from the epidermoid cysts.⁸

DC is typically seen as a well-defined, low attenuated heterogeneous mass due to the predominance of fat content in CT examination. The lesion's heterogeneous appearance depends on the varying amount of hair, calcification, and epidermal debris. Calcification has been reported in up to 20% of cases. In the presence of rupture, hypodensities of oil droplets in the subarachnoid space are pathognomonic findings in radiological imaging. In the presence of a rupture into the ventricular system, hydrocephalus and oil-fluid levels may occur. If chemical meningitis occurs after a ruptured DC, a leptomeningeal reaction or enhancement may be observed.⁹

In MRI, DCs show high signal intensity in the T1-weighted, fast spin echo (FSE) on T2-weighted and FLAIR sequences. Moreover, chemical shift artifact may be seen in the frequency coding direction in the oil/water interface in T2-weighted images. Contrast enhancement and perilesional edema are not expected features.¹

The presence of enhancement after contrast agent injection may be associated with malignant transformation.¹⁰ The presence of scattered high signal intensities in the subarachnoid space and ventricles on MRI is diagnostic for ruptured DC.¹

Diffusion-weighted imaging is important to differentiate DC from an epidermoid cyst. DCs do not restrict diffusion. Lipoma, arachnoid cyst, and cystic craniopharyngioma are other lesions in the differential diagnosis of DC. Lipomas, although having similar imaging features, appear more homogeneous on imaging, while arachnoid cysts are isointense with cerebrospinal fluid on all MRI sequences. Craniopharyngiomas often have a multicystic appearance that usually extends to sella. Calcification and contrast enhancement are common features for craniopharyngiomas.⁸ MRI is more useful than CT for surgical planning in determining the tumor's relationship with neurovascular structures and the spread of post-rupture oil droplets.

In conclusion, limited literature is available regarding the incidence of ruptured DC. Although it is a benign lesion, rupture may cause clinical symptoms ranging from headaches to different neurological complications and even death. Therefore, it is vital to know the imaging features to make the correct diagnosis and differentiate other cystic lesions.

PATIENT'S CONSENT:

Informed consent was obtained from the patient.

CONFLICT OF INTEREST:

The authors declared no conflict of interest.

AUTHORS' CONTRIBUTION:

BKY: Design, interpretation and analysis of data, writing manuscript, literature search.

NG, TSC, SO: Literature search, obtained images.

RT: Revising it critically for important intellectual content.

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