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Case report and literature review

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ABSTRACT

Dermoid cysts are rare, congenital cystic tumours that compose < 0.5% of primary intracranial tumours. They derive from embryonic ectodermal tissue during neural tube closure, and are usually located in the midline or parasagittal region of the skull, although their most common location is the spinal canal.

Rupture into the subarachnoid spaces and ventricles is extremely rare, which may occur spontaneously or after closed-head trauma. Tumour rupture may be asymptomatic or give rise to recurrent chemical meningitis due to spillage of its contents into the subarachnoid space and ventricular system, manifesting with seizures, vasospasm with cerebral infarction and death.

We report a 47-year-old male patient who presented with a sudden onset of severe headache, accompanied by nausea and vomiting without disturbed consciousness. The patient was progressively worsening. On admission, body temperature was 37.8 C, and the patient's vital signs were within normal limits. A CT scan demonstrated a hypodense lesion, and T1-weighted MRI scans revealed a round hyperintense lesion in the left frontal horn, with dilated ventricles, compatible with an intraventricular rupture of a left frontal horn dermoid cyst. There was also significant hydrocephalus. Successful surgical intervention followed, resulting in the complete removal of the cyst and the patient's subsequent full recovery with the resolution of symptoms. In this case, we are reporting on the management of a case of intraventricular ruptured dermoid cyst and reviewing the literature.

INTRODUCTION

Dermoid cysts are non-neoplastic primary tumor lesions, frequently congenital and slow-growing. They are rare lesions that represent < 0.5% of intracranial tumours (1). The cysts occur mostly near the midline, at either end of the neuraxis, and are found in the sellar, parasellar region, frontonasal region, and posterior fossa (2). They derive from the incorporation of ectodermal cells, due to defective separation of the neuroectoderm at the time of neural tube closure. This process usually occurs during the 3rd to 5th week of embryogenesis (3, 4, 5).

The cyst wall is composed of a capsule, characterized by a thick, stratified squamous epithelium containing dermal elements such as sebaceous, apocrine or sweat glands; hair follicles; and even sometimes teeth (6, 7). The cyst contents are due to the secretion of desquamated

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epithelium, and sebaceous secretions (5). Enlargement of the cysts is due to the accumulation of the above products. The presence of such elements in the wall distinguishes dermoid cysts from epidermoid cysts. (8) being dermoid tumors 4 to 9 times less frequent than epidermoid tumors.

While often discovered incidentally, they may present with a spectrum of symptoms, including headaches, seizures, and neurological deficits, depending on their size, location, and potential for rupture (2). They usually occur in childhood and are more common in males.

These lesions are often asymptomatic unless they rupture, the dermoid cyst rupture is quite rare, around 0.18%, reported by a single independent study (8). Which may occur spontaneously, or in relation to (often minor) trauma, resulting in dissemination of the intracystic contents into the subarachnoid space and ventricles. But the probable hypothesis behind rupture is spontaneous onset due to accumulation of glandular secretions, brain pulsations and head movement. (9, 10).

Dissemination of the cholesterol debris from the cyst contents into the subarachnoid and intraventricular spaces, may cause chemical meningitis (4, 5). This can be further complicated by obstructive hydrocephalus, vasospasm, infarction, and even death. It is debatable whether these lesions acutely clinically manifest at the time of rupture or if acute symptoms present long after a chronic process of dissemination of dermoid contents.

We review the clinical, radiographic, and surgical features of ruptured intraventricular dermoid cysts.

CASE REPORT

We report a 47 years old male patient with unremarkable medical history. Who presented to the emergency department with a sudden onset of severe headache, accompanied by nausea and vomiting. Patient was progressively worsening. The patient reported a history of intermittent headaches over the past few months but denied any significant neurological symptoms.

During the physical examination, the patient complained of a persistent throbbing headache localized to the right frontal region. No neurological deficits were revealed during clinical examination, body temperature was 37.8 C, and the patient's vital signs were within normal limits. However, the severity and persistence of the headache raised

concerns, prompting a non-contrast computed tomography scan of the head.

A CT scan of the brain demonstrated significant hydrocephalus and progression of the volume of a fatty mass towards the horn of the left lateral ventricle, a round hypo-dense lesion, consistent with air or fat in left frontal horn measuring 53×24×31cm. The imaging findings were suggestive of a ruptured dermoid cyst.

Magnetic resonance imaging MRI brain confirmed the findings (Figure 1). showing dermoid cyst in the different sequences. (A) T1 MRI brain, axial cut, showing hyperintense round lesion in left frontal horn, with dilated ventricles, (B) T2 axial cuts showing hyperintense lesion almost similar to CSF, (C) FLAIR showing hyperintense lesion.

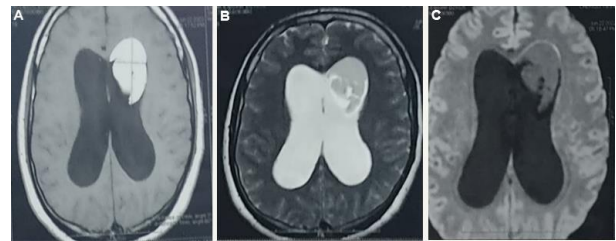


Figure 1. Magnetic resonance imaging showing dermoid cyst in the different sequences. (A) T1 MRI brain, axial cut, showing hyperintense round lesion in left frontal horn, with dilated ventricles, (B) T2 axial cuts showing hyperintense lesion almost similar to CSF, with scattered contents. (C) FLAIR showing hyperintense lesion.

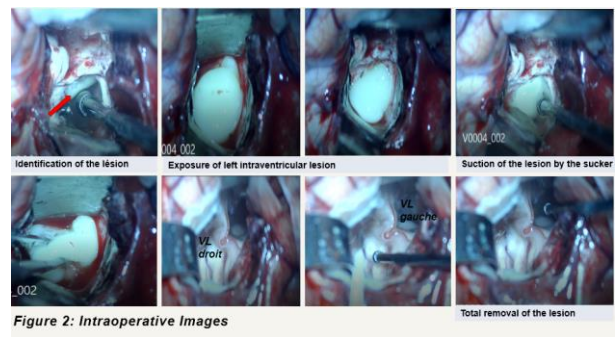


Figure 2: Intraoperative Images

Patient was planned for surgery. Due to large size of lesion (53×24×31cm), we planned for transfrontal transcortical approach via middle frontal gyrus. Procedure – Patient was kept in supine position with head in neutral state. Bicoronal skin incision was given and left paramedian frontal craniotomy (7 cm size) was made. Before opening middle frontal gyrus, we passed ventricular catheter through it to drain CSF and followed its trajectory to open frontal horn. The tumor cyst was containing hair follicles, fat, and sebaceous glands, which were easily removed with suction to allow intracystic debulking and decompression of the cyst. The resection cavity was

thoroughly irrigated with saline, till it became clean, and dispersion of cyst contents was prevented to minimize the risk of chemical meningitis. The cyst was successfully resected (Figure 2).

He presented postoperative hydrocephalus eventually required ventriculoperitoneal shunting. Postoperatively, the patient remained neurologically stable. He had no evidence of recurrence at the time of his 2-year follow-up examination.

DISCUSSION

The primary lesions of intraventricular location are relatively infrequent. The most frequently described tumor lesions located in lateral ventricles are meningiomas, papillomas, astrocytomas and subependymomas, being dermoid cysts infrequent. Clinically they will present a syndrome of endocranial hypertension due to obstructive hydrocephalus.

Dermoid cyst is a rare type of benign mass lesion, formed due to abnormal non-disjunction of all dermal layers (ecto-, meso- and endoderm). It is uncommon in brain. The dermoid cyst rupture is quite rare. Less than 15 cases of ruptured intraventricular dermoid cyst have been reported till now. Dr. Howard Brown and Dr. Douglas Miller first reported about ruptured intraventricular dermoid cyst in 1947 and 1950 respectively. (11).

The rupture is usually spontaneous, but it has also been reported after closed-head trauma (12). The dissemination of intracystic contents after surgical removal of dermoid cysts has also been described. When dermoid cysts spontaneously rupture into the subarachnoid space or ventricular system, patients can present with a variety of symptoms, including headache, seizures, hemiparesis, aseptic meningitis, hydrocephalus, vasospasm, cerebral ischemia, fat embolism, and visual deficit (9, 13, 14, 15, 16, 17).

The clinical presentation of dermoid cysts depends on multiples factors, therefore, is directly related to their location and size. They increase in size due to epithelial desquamation and glandular secretion, generating edema and displacing adjacent structures. (5).

It is postulated that the presence of lipid contents in the subarachnoid space causes chemical irritation of the adjacent neurovascular structures. In one study by El-Bahy et al. (18), headache was the most common symptom, followed by seizures, cerebral ischemia with sensory or motor deficit, and aseptic

meningitis. In patients with unruptured dermoid cysts, the signs or symptoms result from compression of adjacent neurovascular structures; however, because these are slow-growing tumors, some may reach large sizes without causing neurological signs or symptoms (19, 20).

Dermoids have a medial location and are more frequent in the perisellar region and posterior fossa. As for those of intraventricular location, it is debated whether they are primarily intraventricular tumors or if they penetrate the cavities from the cisterns of the base or periventricular sulci.(21).

The CT imaging is considered the test of choice for emergency contexts, due to the ectodermal content of the cysts they will be observed as well-circumscribed, hypodense lesions, with the imaging characteristics of fat, due to the predominant lipid content. Hydrocephalus may be present if there is rupture into the ventricular system and a fat-fluid level may be present. The cyst may have a heterogeneous appearance, if there is a mixture of hair, calcification, and epidermal debris. Dermoid cysts do not demonstrate enhancement with the administration of contrast.

MRI is superior to CT in characterizing the extent of lipid dissemination, and for operative planning. The cysts appear hyperintense on T1-weighted imaging, due to their fat content, occasionally with a liquid-fat level. The presence of lipid droplets in the subarachnoid cisterns, sulci, and ventricles is pathognomic of a ruptured cyst. On T2-weighted imaging, dermoid cysts have heterogeneous signal intensity. Depending on the different components, they may appear hypo- or hyperintense. If the internal fat content is low, the cyst may have fluid signal. If there is associated chemical meningitis, there may be pial and ventricular ependymal enhancement after the administration of contrast.

The differential diagnosis of fat-containing intracranial cysts includes epidermoid cysts, arachnoid cysts, teratomas, and lipomas.

The definitive treatment of dermoid cysts is purely surgical, being of choice the excision of the tumor by open microsurgery guided preferably by intraoperative ultrasonography or excision by neuroendoscopy. Operative treatment consists in radical removal of the tumor without contamination of the cerebrospinal fluid (CSF) by the content of the capsule. Adherence of the capsule to the important structures of the brain can sometimes impede the

radical removal. Also, in these cases cure can be expected, especially in adults.

The open or endoscopic approach will be decided according to the criteria of the medical team, considering mainly the size of the tumor lesion to make the decision. It is important for the neurosurgeon to remove the tumor with a precise safety diameter to avoid possible recurrence, but also trying to generate as little damage as possible to the healthy brain parenchyma.

In this case, microsurgical technique was decided to increase the percentage of tumor resection. The aim of this management is to avoid the development of hydrocephalus associated with the natural evolution of endocranial hypertension syndrome, which would culminate in herniation and the consequent death of the patient.

The contents within the tumor cyst consistently included hair follicles, fat, and sebaceous glands, which were easily removed with suction to allow intracystic debulking and decompression of the cyst. The resection cavities were irrigated with saline, and dispersion of cyst contents was prevented to minimize the risk of chemical meningitis.

The patient's post-procedure evolution was favorable. Currently the patient is discharged with the corresponding care and indications at home.

CONCLUSION

Intracranial dermoid cysts are benign, asymptomatic lesions. But various complications have been reported following rupture of dermoid cyst, like, seizures, hydrocephalus, meningitis, deranged mental functions, focal deficit, death. Even after surgical excision, patient might deteriorate due to meningitis or hydrocephalus. Rupture of intraventricular dermoid cyst is extremely rare, resulting in dissemination of the intracystic contents into the subarachnoid space and ventricles.

In this case, since the patient presented symptoms associated with hydrocephalus, it was decided to perform a microsurgical technique to increase the percentage of tumor resection. The patient fortunately had an uncomplicated recovery.

The treatment of choice for these lesions is neurosurgery. The goal is to aim for complete surgical resection, while weighing against the risk of causing damage to adjacent structures. This will prevent possible complications associated with the tumor lesion that may compromise the patient's life.

Contemporary low rates of mortality in intracranial dermoid cysts reflect earlier and more correct diagnosis and advances in surgical techniques.

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