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CASE REPORT

Post-traumatic rupture of an intracranial dermoid cyst: A case report

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ABSTRACT

Dermoid cysts are rare benign tumors with even rarer intracranial locations. We report the case of a post-traumatic rupture of a left temporal dermoid cyst in a 17-year-old patient who was admitted to the emergency department following a road traffic accident. A CT scan fortuitously revealed a left internal temporal extra-axial lesion with fat density, communicating with material of the same characteristics in the subarachnoid spaces and along the falx cerebri, suggesting a ruptured dermoid cyst. An MRI confirmed the diagnosis by clearly identifying the fatty component of the cyst extending to the subarachnoid spaces. This case illustrates the role of imaging, particularly MRI, in the diagnosis of dermoid cysts.

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Introduction

Dermoid cysts are benign tumor-like lesions resulting from the aberrant inclusion of ectodermal elements during embryonic development. Although they are rare, these cysts can occur in various locations, including intracranially. Their rupture, which may be spontaneous or due to trauma, can lead to serious complications. This article presents a case of post-traumatic rupture of a left temporal dermoid cyst, suspected via CT and confirmed by MRI.

Case Presentation

This is a 17-year-old patient with no significant past medical history who presented to the emergency department following a head trauma caused by a road traffic accident. The trauma was associated with an initial brief loss of consciousness without vomiting.

The initial CT scan (Fig. 1) revealed no contusions or fractures but identified a fairly well-defined extra-axial formation in the left internal temporal region. This lesion showed fat density and measured 25 × 19 mm. In addition, dissemination of similar density material was noted in the basal cisterns, extending to some parieto-temporal and occipital cortical sulci bilaterally, more pronounced on the left side.

An MRI (Fig. 2) was performed, which revealed the same extra-axial left temporal lesion. It appeared heterogeneous, predominantly hyperintense on T1, T2, and FLAIR sequences, with areas of intermediate signal. The lesion exhibited a more marked peripheral T1 hyperintensity, corresponding to a chemical shift artifact at the interface with cerebrospinal fluid. The lesion also showed a signal void on T2* sequences with blooming artifacts, and its signal was suppressed on fat-saturation sequences. It showed no diffusion restriction on DWI sequences. Dissemination of material with similar characteristics was observed, filling the basal cisterns and extending into the occipital and parieto-temporal cortical sulci bilaterally. A few droplets were also noted in the inter- and periventricular regions, as well as along the falx cerebri.

Based on the MRI findings, the diagnosis of a ruptured intracranial dermoid cyst was established.

Given that the patient was asymptomatic, a decision was made to pursue clinical and radiological surveillance. To date, the patient remains asymptomatic, and imaging findings are stable.

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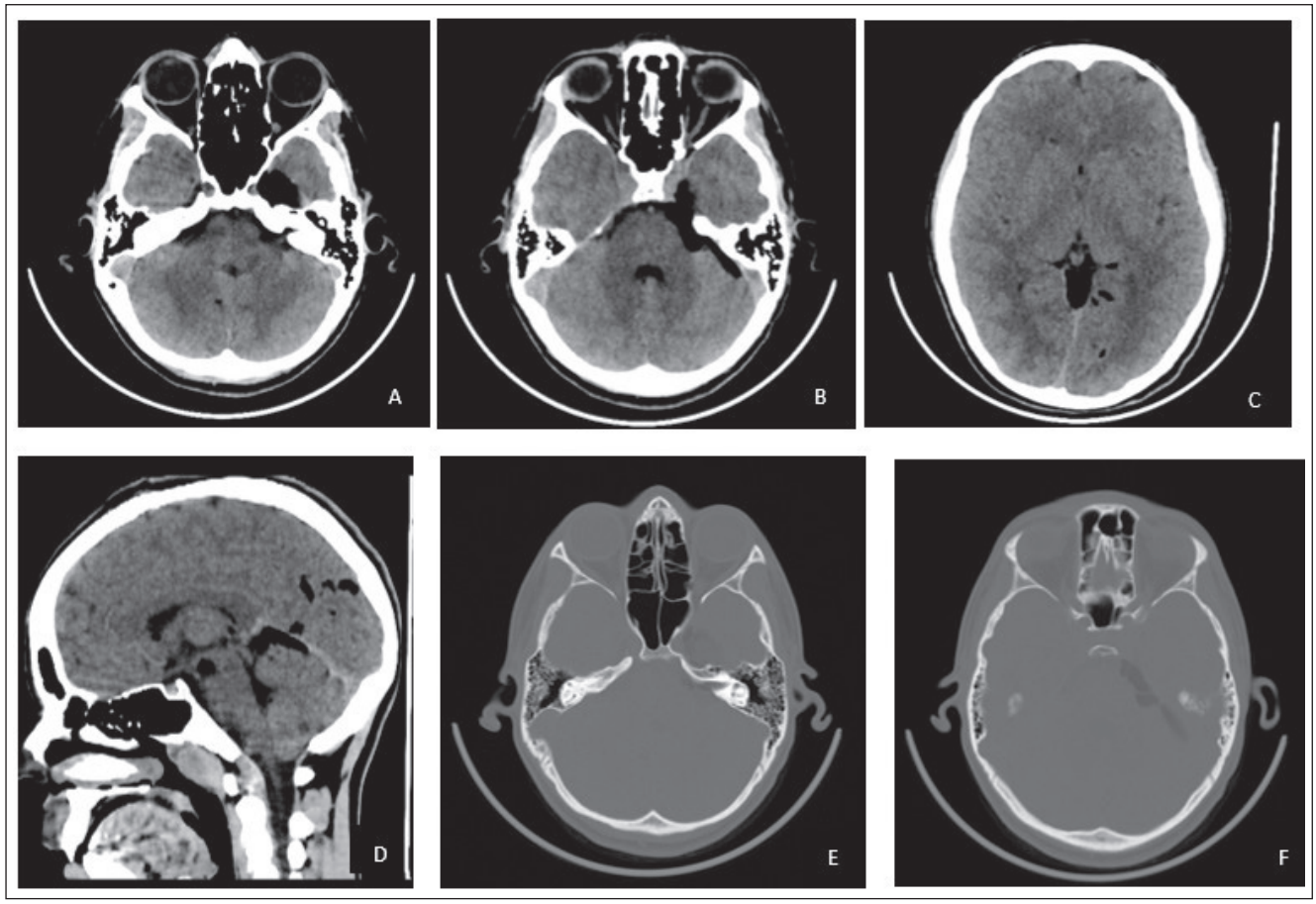


Figure 1. CT scan without contrast injection in axial slices with parenchymal window (axial slices A, B, C) and sagittal (D); axial slices window (E and F): left temporal formation of fat density ruptured into the subarachnoid spaces with dissemination of fat density material in the cisterns and cortical sulci.

Discussion

Dermoid cysts are congenital formations of ectodermal origin, representing less than 1% of intracranial expansive lesions [1]. They are primarily located in the supra- and parasellar regions, the basifrontal area, and the posterior cranial fossa, although rarer temporal localizations have been reported, as in our case [2].

Histologically, these cysts are characterized by a thick wall composed of keratinizing epithelium, which may include various skin appendages [3]. This structure explains the potential for rupture during growth, due to the development of these appendages [2].

The rupture of an intracranial dermoid cyst is a rare but potentially severe event [4], sometimes spontaneous [5], and often caused by direct trauma or an increase in intracranial pressure. In the case presented, mild head trauma likely led to an increase in pressure, resulting in cyst rupture. Clinically, this rupture may manifest with a variety of symptoms, ranging from simple headaches to meningeal syndrome [6].

Imaging allows diagnosis by identifying on CT a well-defined lobulated extra-axial structure with fat density [7], which may contain parietal calcifications without significant contrast enhancement [8].

Very rarely, a dermoid cyst may exhibit high density, thought to be due to a combination of saponification, microcalcification, and blood products. This most commonly occurs in cysts located in the posterior fossa [9].

MRI provides a more detailed visualization of dermoid cysts. Typically, these lesions appear as well-defined masses with hyperintensity on T1 and T2 sequences, due to their fat content [7]. This fat may also cause chemical shift artifacts at fat-fluid interfaces. Diffusion-weighted sequences do not show significant restriction, and there is generally no enhancement after gadolinium injection [10]. Parietal calcifications, when present, appear hypointense on T1 and T2 sequences and are usually better visualized on CT [11].

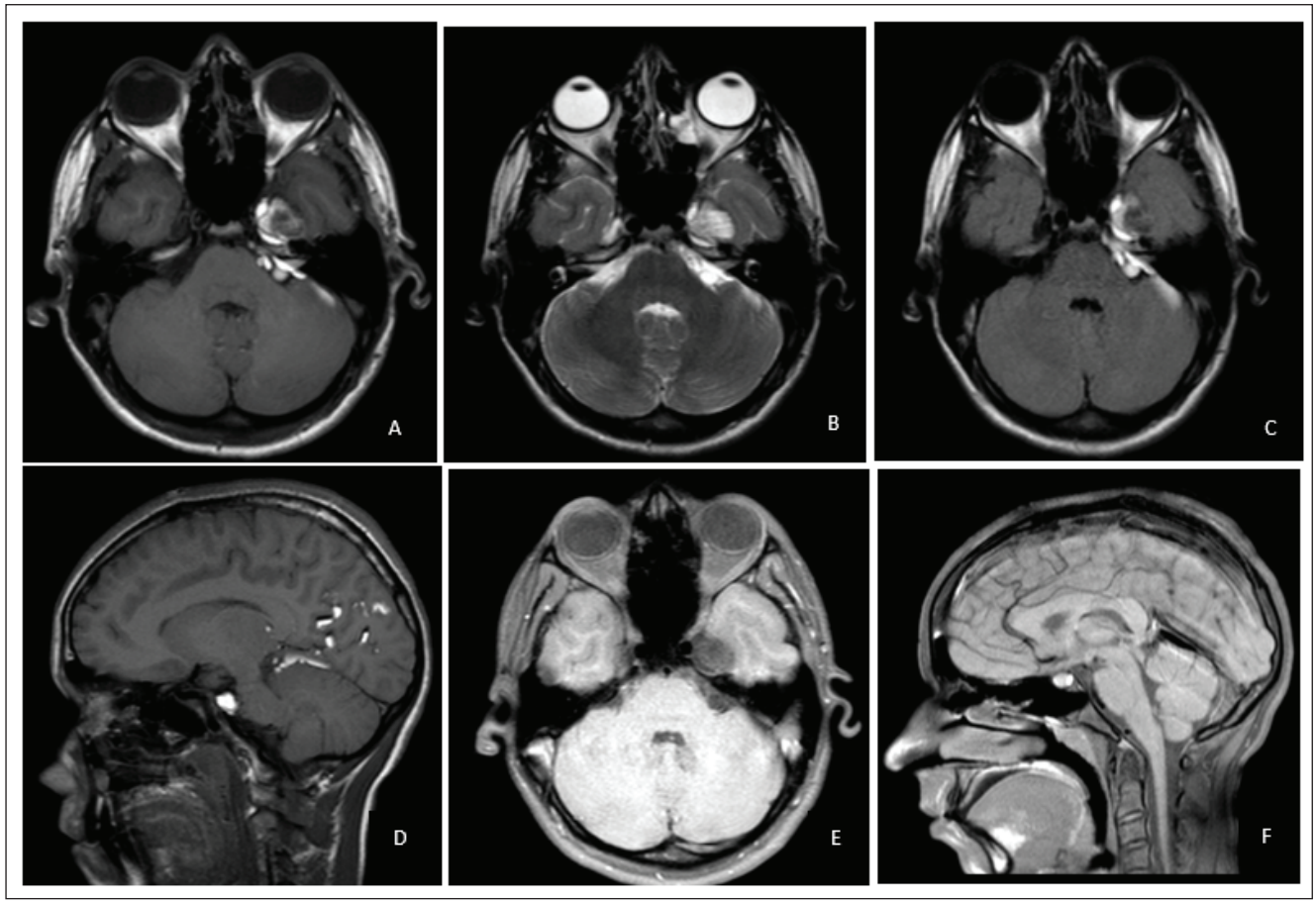


Figure 2. MRI of the same patient with axial sequences T1 (A), T2 (B), FLAIR (C), T1 FS (E), and sagittal T1 (D) and T1 FS (F): left temporal formation of fat density ruptured into the subarachnoid spaces with dissemination of fat density material in the cisterns and cortical sulci. Note the disappearance of the signal from the formation of fat-saturated sequences.

Rupture is identified by the presence of fat-density material disseminated in the subarachnoid spaces, cisterns, and even intraventricularly. Fat-fluid levels may be observed intraventricularly, or fat droplets may be seen in the sulci [9].

This rupture can cause complications such as chemical meningitis, vasospasm, and hydrocephalus, requiring prompt and appropriate management to minimize the risk of neurological sequelae [12].

The main differential diagnosis is an epidermoid cyst, which can be distinguished from a dermoid cyst by its signal, which is similar to cerebrospinal fluid. Diffusion-weighted sequences show marked hyperintensity with a very low apparent diffusion coefficient in epidermoid cysts [10], unlike in our case, which showed no findings on diffusion sequences.

Another differential diagnosis is lipoma, which appears as a homogeneously fatty lesion, unlike the dermoid cyst, which shows heterogeneity due to its histological composition [10], as in our case.

A conservative approach may be considered for ruptured cysts when there is a risk of vascular injury during surgery, but with a risk of recurrent rupture [12]. This approach involves intravenous corticosteroid treatment during the acute phase [5]. Corticosteroid therapy may also be combined with surgical treatment when indicated, with the possibility of injecting corticosteroids directly into the surgical site to reduce inflammation caused by cyst rupture and postoperative changes [13].

Surgical resection of the cyst remains the treatment of choice, complemented by symptomatic management of complications [13].

Conclusion

The rupture of an intracranial dermoid cyst, although rare, represents a medical emergency requiring rapid diagnosis by imaging. Appropriate, multidisciplinary management is essential to prevent serious complications and ensure a favorable prognosis for the patient.

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