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Case Report

Intracerebral Dermoid Cyst: Case Report

İntraserebral Dermoid Kist: Olgu Sunumu

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Abstract
Intracranial dermoid cysts account for less than 0.7% of all intracranial tumours. These are cystic masses that develop due to the ectopic placement of congenital tissues during early embryonic development. They typically present in childhood or early adulthood with symptoms related to mass effect, such as headache, epilepsy, and other neurological signs. The cyst contents generally consist of tissues derived from both ectodermal and mesodermal origins. Early detection and total excision prior to rupture can provide a complete cure. Herein, we present a case of a patient who was admitted with epileptic seizures and was diagnosed with an intracerebral dermoid cyst located in the left temporal region. The case is discussed in the context of current literature.
Keywords: Dermoid cyst, Epilepsy, Intracerebral.

Özet
İntrakranial dermoid kistler, tüm intrakraniyal tümörlerin yaklaşık %0.7'sinden azını oluşturmaktadır. Erken embriyonik hayatta konjenital dokuların ektopik yerleşimi nedeniyle ortaya çıkan kistik kitlelerdir. Genellikle çocukluk ve erken yetişkinlik döneminde, kitle etkisi nedeniyle ortaya çıkan baş ağrısı epilepsi ve diğer nörolojik belirtiler nedeniyle karşımıza çıkar. İçeriği genellikle ektoderm ve mezoderm yapılarından gelişen dokulardan oluşmaktadır. Kist rüptüre olmadan gelişmeden erken fark edilmesi ve total olarak çıkartılması tam kür sağlayabilmektedir. Bu yazıda, epileptik nöbet yakınması ile gelen, tetkiklerinde sol temporal bölgede yerleşimli intraserebral dermoid kist tanısı konulan hastamızı literatür eşliğinde tartışmayı amaçladık.
Anahtar Kelimeler: Dermoid kist, Epilepsi, İntraserebral.

Introduction

Intracranial dermoid cysts are rare, benign congenital tumours that arise (delete) develop (add) due to the abnormal placement of ectodermal tissue between the third and fifth weeks of embryonic development. They account for approximately 0.1–0.7% of all intracranial tumours. These cysts are typically diagnosed during the first three decades of life when symptoms begin to manifest. Dermoid cysts contain skin appendages such as hair, sebaceous glands, and adipose tissue, and are lined with keratinised stratified squamous epithelium (1-4).

They are usually located along the midline in intracerebral regions such as the suprasellar area, cerebellopontine angle, and parasellar region. Clinical symptoms depend on the size, location, and compression of adjacent structures, and may include headache, epileptic seizures, visual disturbances, and cranial nerve palsies. In rare cases, the rupture of cyst contents into the subarachnoid space may lead to serious complications such as aseptic meningitis, hydrocephalus, vasospasm, and cerebral ischaemia (2-5).

In this article, we present a rare case of an intracranial dermoid cyst located in the left temporal region and discuss it in light of the existing literature.

Case

A 79-year-old male patient with a known A 46-year-old female presented to the emergency department in the postictal phase following a generalized epileptic seizure. The patient had been receiving sodium valproate for approximately five years due to recurrent seizures, which had gradually increased in both frequency and severity over time. Clinically, the seizures exhibited features consistent with complex partial (focal impaired awareness) seizures, but did not meet the criteria for drug-resistant epilepsy. The semiology of the episodes was concordant with the left temporal lobe**,** corresponding to the lesion's localization. Interictal electroencephalography (EEG) revealed epileptiform discharges in the left temporal region, suggesting that the seizures were likely related to the lesion. Her past medical history included a 15-year history of migraine. Neurological examination and laboratory findings were otherwise unremarkable.

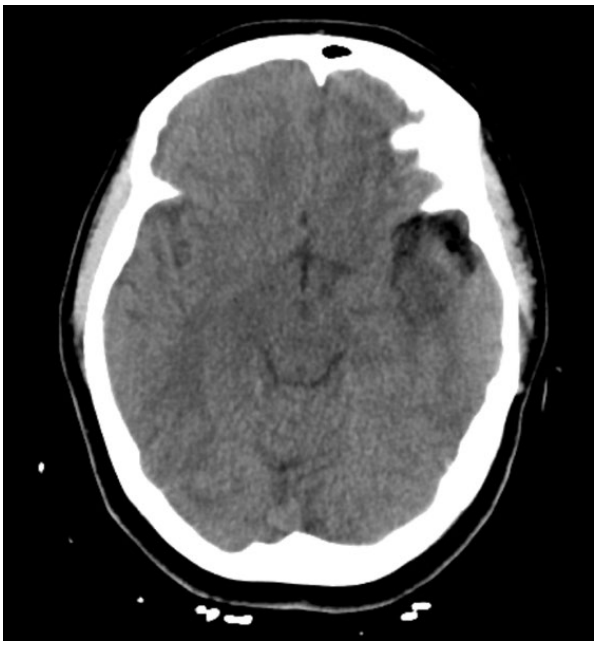

Figure 1. On brain CT, a 31 mm heterogeneously cystic mass was observed in the left temporal region. The lesion appeared isodense in its content with a surrounding hypodense area.

Cranial computed tomography (CT) revealed a 31mm intra-axial cystic lesion in the left temporal lobe with minimal perilesional vasogenic edema (Figure 1). Magnetic resonance imaging (MRI) demonstrated a well-defined, lobulated cystic lesion measuring 31 × 30 mm in the left anterior temporal region. The lesion appeared hyperintense on T2-weighted sequences**,** showed peripheral contrast enhancement after intravenous contrast administration, and was surrounded by mild vasogenic edema (Figures 2A, 2B).

Detailed MRI evaluation of the hippocampal formation and mesial temporal structures revealed no evidence of hippocampal sclerosis. There were no signs of hippocampal volume loss, increased T2 signal intensity, or disruption of the internal hippocampal architecture. Therefore, the epileptic focus was considered to be secondary to the dermoid cyst rather than a mesial temporal pathology.

A left pterional craniotomy was performed to access the lesion. Upon entering the cyst cavity, a large amount of hair, keratinized epithelial debris, and adipose tissue was evacuated. The cyst wall was then completely excised by blunt dissection**,** and the resection cavity was thoroughly irrigated with saline to ensure removal of any residual material (Figure 3).

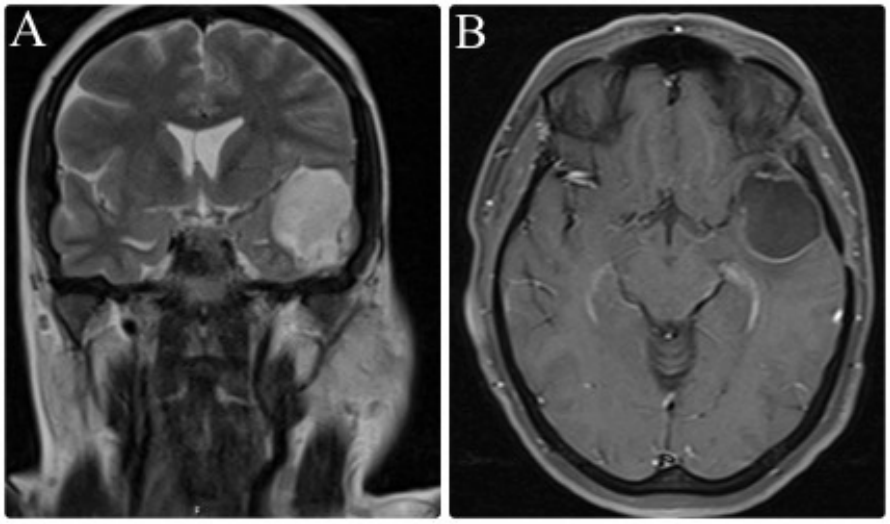

Figure 2. A. Coronal T2-weighted MRI revealed a cystic lesion measuring approximately 31 × 30 mm in the left anterior temporal region, which appeared hyperintense. B. Axial T1-weighted MRI following intravenous contrast administration demonstrated a lobulated cystic lesion with peripheral contrast enhancement and mild surrounding vasogenic oedema.

No postoperative complications were observed. Histopathological examination confirmed the diagnosis of a dermoid cyst, showing keratinized stratified squamous epithelium, hair follicles, and sebaceous glands**,** consistent with ectodermal differentiation (Figures 4A, 4B). During follow-up, the patient remained seizure free**.** Sodium valproate (1000 mg/day) was continued for six months postoperatively and was then gradually tapered and discontinued under neurological supervision

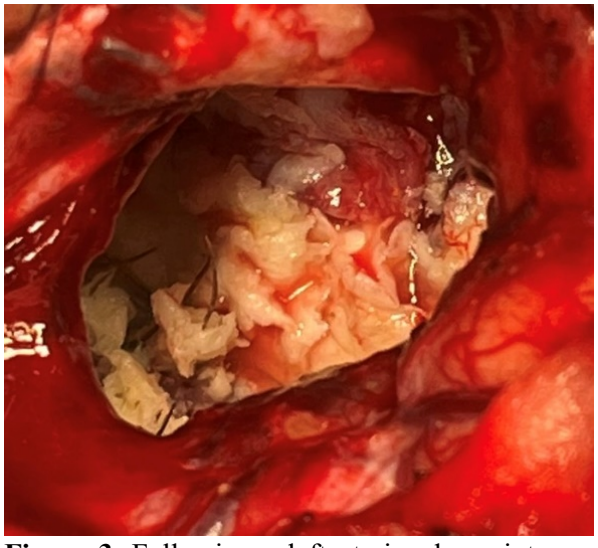

Figure 3. Following a left pterional craniotomy, the cyst cavity was accessed, revealing a large amount of hair, keratinised epithelial debris, and adipose tissue.

Discussion

Intracranial dermoid cysts account for less than 1% of all dermoid cysts in the body. They are believed to originate from embryonic remnants trapped along suture lines during early embryogenesis, particularly during neuroectodermal closure. These cysts are encapsulated by stratified epithelial tissue and contain elements such as desquamated squamous epithelial cells, hair follicles, adipose tissue, sweat glands, sebaceous glands, and keratin debris representing both ectodermal and mesodermal origins (1,2). Although dermoid cysts share certain histological similarities with mature teratomas, they are composed exclusively of ectodermal derivatives and therefore represent a distinct pathological entity.

Intracerebral dermoid cysts typically exhibit slow growth and may remain asymptomatic for prolonged periods. Clinical symptoms usually arise due to mass effect depending on the size and location of the cyst. According to previous reports, intracerebral dermoid cysts account for less than 0.7% of all intracranial dermoid cysts, and only a limited number of cases have been described in the literature (1,6–9). The most frequently reported localisations include the suprasellar, parasellar, and posterior fossa regions, whereas pure intraparenchymal temporal lobe involvement is extremely rare. Reported cases are often presented with headache or epileptic seizures, reflecting the mass effect or cortical irritation caused by the lesion (6–8). Surgical indications generally include symptomatic mass effect, progressive

neurological symptoms, or seizure control, and complete excision of both the cyst wall and its contents is associated with an excellent prognosis and low recurrence rates (2,3,8,9). In our case, the patient was presented with epileptic seizures that had progressively worsened over approximately five years despite medical therapy.

On CT, dermoid cysts commonly appear as hypodense cystic lesions, often accompanied by calcifications or fat density. On MRI, they are typically hyperintense on T1-weighted sequences due to their fat content and show hyperintense or heterogeneous signal characteristics on T2-weighted images. MRI also provides detailed assessment of the cyst's relationship to adjacent structures (7,8). In our case, contrast-enhanced MRI demonstrated a hyperintense cystic lesion on T2-weighted imaging, with peripheral contrast enhancement and mild surrounding vasogenic oedema.

The primary treatment modality is complete surgical excision of the cyst with preservation of its integrity. Total removal of both cyst contents and capsule significantly reduces recurrence rates. However, total excision may be challenging due to the cyst's potential adherence to neural and vascular structures. In our case, surgical treatment was limited to lesionectomy**,** as the epileptic seizures were semeiologically concordant with the lesion located in the left temporal lobe, and no radiological or electrophysiological evidence of mesial temporal sclerosis was identified. According to the current criteria for temporal lobe epilepsy surgery, additional resection of mesial temporal structures is only indicated in the presence of drug-resistant epilepsy or discordance between lesion location and seizure semiology. Therefore, performing lesionectomy alone was considered an appropriate surgical approach in this patient.

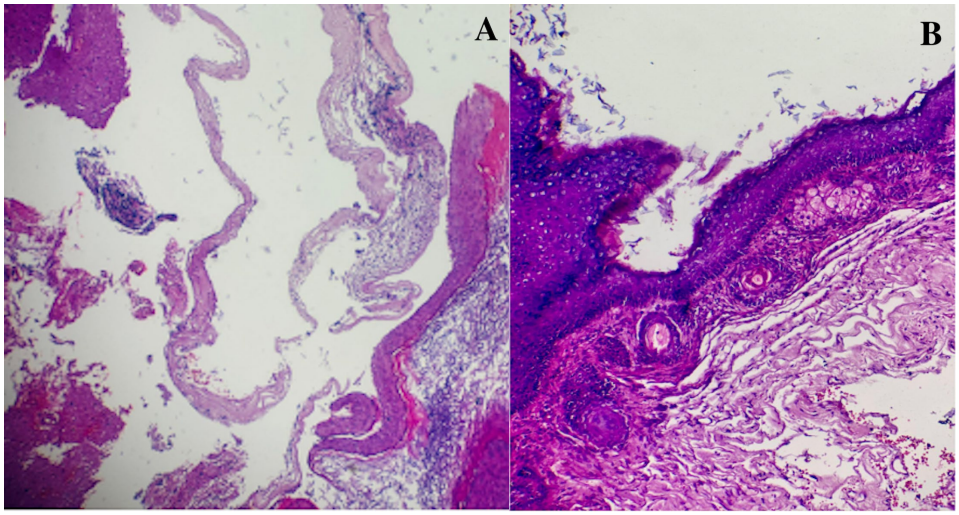

Figure 4. A. Brain left temporal lobe, dermoid cyst; Hematoxylin and eosin stain, ×40. Areas of mature glial tissue with fibrillary background and keratinized squamous epithelium, representing ectodermal components of the cyst. B. Brain left temporal lobe, dermoid cyst; Hematoxylin and eosin stain, ×100. Stratified squamous epithelium with underlying dermal structures, including hair follicles and sebaceous glands, confirming ectodermal differentiation.

Subtotal excision carries a high risk of recurrence. To prevent postoperative complications such as aseptic meningitis and delayed ischaemic events, thorough irrigation of the resection cavity and subarachnoid cisterns with saline is essential (3). In recent years, endoscopic surgical techniques have been introduced alongside conventional microsurgical approaches in the management of dermoid cysts, whether ruptured or not (9). In our case, after complete excision of the lesion, we performed thorough irrigation of the resection cavity and the Sylvian cistern with saline to remove any residual material.

In ruptured cases, the risk of chemical meningitis and other neurological complications is heightened due to inflammatory processes. Surgical management becomes more complex, and additional postoperative interventions may be required, such as corticosteroids for inflammation control or ventriculoperitoneal shunting for hydrocephalus. Dermoid cyst rupture is reported to occur in approximately 0.18% of cases. When rupture does occur, it may result in serious intracranial complications such as chemical meningitis and hydrocephalus (3,10).

Conclusion

Intracranial dermoid cysts are rare but clinically significant congenital lesions. Clinical symptoms may be non-specific, and imaging techniques play a crucial role in diagnosis. Surgical intervention generally provides favourable outcomes; however, careful preoperative assessment and surgical planning are essential due to the risk of potential complications.

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Conflict of interest statement

The authors have no conflicts of interest to declare.

Written consent

There is an informed consent form for the patient (14/11/2024).

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