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Intrasellar arachnoid cyst: A case report and review of the literature

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ABSTRACT

INTRODUCTION: Arachnoid cysts (ACs) are frequently found on intracranial imaging studies but intrasellar arachnoid cysts are rarely encountered.
PRESENTATION OF CASE: We present a 49-year old patient who had headaches for 6 months and cystic sellar mass was found in his cranial imaging. We operated him by transnasal transsphenoidal route. Our intraoperative diagnosis was an arachnoid cyst and pathologic studies verified our observation. He did well postoperatively and after a 1 year follow-up he was left free from future follow-ups.
DISCUSSION: As common cystic lesions occupying the sellar region can simulate ACs both clinically and radiologically, neurosurgeon can fail to include ACs in making the initial diagnosis preoperatively.
CONCLUSION: Although a rare entity, arachnoid cysts should be considered in the differential diagnosis of sellar region.

1. Introduction

Intracranial arachnoid cysts (AC) are benign lesions of the arachnoid, forming nearly 1% of all intracranial space-occupying lesions [1]. Compared to other cystic lesions of the sellar region namely the cystic adenomas, craniopharyngiomas and Rathke's cleft cysts, they are rarely seen in this localization with few reports available in the English literature [2–14]. ACs are most commonly found in the Sylvian fissure followed by the cerebellopontine angle, the supracollicular area and the vermian area. Intrasellar arachnoid cysts (IAC) constitute about 3% of all intracranial arachnoid cysts [15]. The discrimination of IACs from the cystic lesions of the sellar region remains a difficult preoperative problem because symptoms, signs and imaging characteristics can mimic each other [9,14]. We describe a case of intrasellar arachnoid cyst, treated through transnasal transsphenoidal approach.

2. Case report

A 49-year-old man presented with 6 month history of headache. The results of the physical, neurologic and ophthalmologic examinations including visual acuity, fundoscopy and visual field studies were unremarkable. Cranial magnetic resonance imaging (MRI) revealed a sellar cystic lesion, and a hypophysis MRI including dynamic contrast enhanced studies were performed.

On the MRI a cystic lesion with a dimension of 18 × 14 × 14 mm, extending from the suprasellar cistern, traversing the diaphragma sellae and reaching the level of the dorsum sellae was seen. The optic chiasm was compressed. The lesion was hypointense in T1-weighted images and hyperintense in T2-weighted images. In contrast studies rim like enhancement of the periphery of the lesion was noted. The normal pituitary and pituitary stalk demonstrated a typical enhancement pattern, and were displaced laterally to the right by the lesion (Fig. 1). A computed tomography (CT) scan dedicated to the sellar region, with and without contrast, confirmed a cystic lesion, hypodense with no contrast enhancement (Fig. 1).

His blood studies including complete blood count, routine biochemistry and pituitary hormone levels, and urinalysis were within normal limits.

A presumptive diagnosis of Rathke's cleft cyst was made based on the MRI findings.

The patient was operated by transnasal transsphenoidal route. Intraoperatively, after the dural incision, cerebrospinal fluid began to leak and when the opening was widened the lesion was found to have an arachnoidal membrane and when the membrane was opened watery, colorless, CSF like liquid was seen. The membrane was excised totally and normal pituitary, pituitary stalk and the sellar diaphragm were observed.

Postoperative 24-hour hypophysis MRI revealed that cyst was decompressed and optic chiasm and hypophysis were in normal location with no compression (Fig. 2).

On pathologic examination, monolayered and flattened meningothelial cap cells were observed in the fibrous cyst wall (Fig. 3A). The subepithelial stroma was composed of a thin, non-vascular connective tissue rich in reticulin fibers (Fig. 3B).

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Perilesional anterior hypophyseal cells were present. Immunohistochemically overlying cells revealed, EMA positivity (Fig. 3C), while GFAP, Ki-67, synaptophysin, S-100 were negative. The anterior hypophyseal cells were observed Pan-ck (5/6/8/18) and synaptophysin (Fig. 3D) positive. Thus, the histological examination established a diagnosis of arachnoid cyst.

The patient was discharged on the postoperative fourth day with no complications and morbidity. In his neurosurgical and endocrinological follow-up of 1 year, no abnormality was observed.

3. Discussion

The pathophysiology of the development of IACs remains controversial. It has been suggested that IACs result from a defective

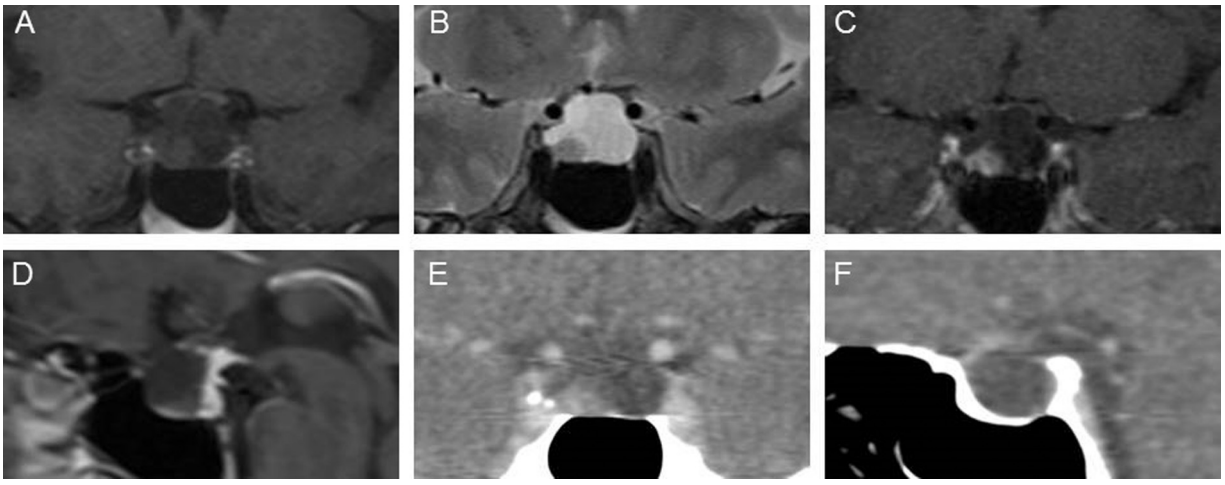

Fig. 1. Preoperative MRI and CT images showing a cystic sellar lesion. A: Coronal T1 weighted MRI image, B: T2 weighted MRI image, C: Coronal Dynamic contrast enhanced T1 weighted MRI image, D: Sagittal dynamic contrast enhanced MRI image; E and F Coronal and sagittal contrast enhanced CT images.

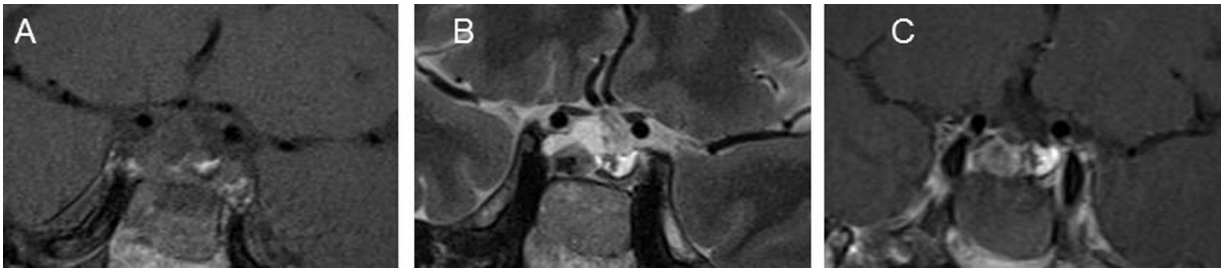

Fig. 2. Postoperative Day 1 coronal MRI images showing cyst excised and the normal hypophysis. A: T1 weighted, B: T2 weighted and C: Dynamic contrast enhanced studies.

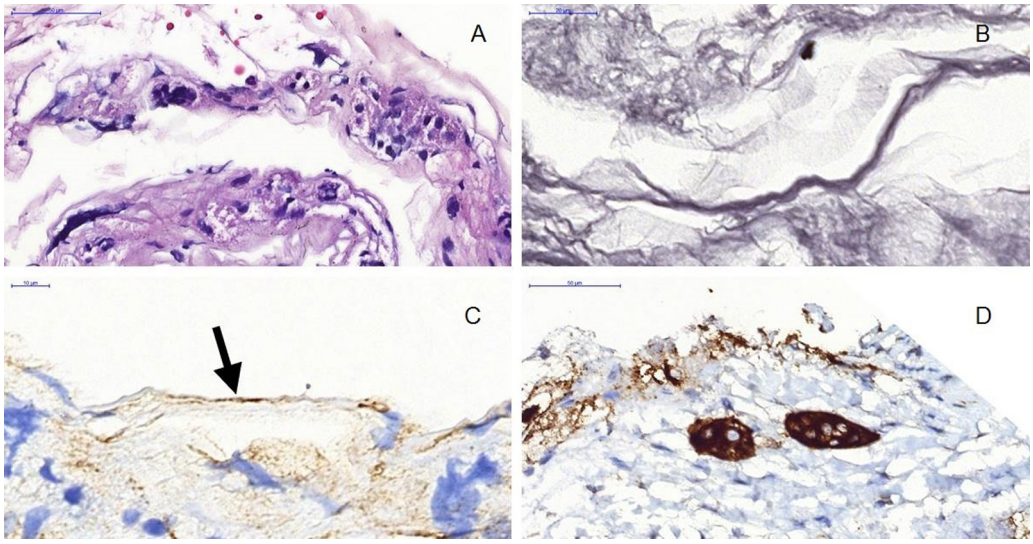

Fig. 3. Histopathology and immunohistochemistry. A: Flattened arachnoid cells lined on thin basement membrane (H&E, x59.7). B: Basement membrane rich in reticulin fibers in cyst wall (Gomori's reticulum stain, x113.2). C: Cyst wall overlined by flattened arachnoidal cells reacting with EMA (arrow) (Biotinylated streptavidin complement, EMA, x127.5) D: Anterior pituitary cell cluster in close neighbourhood of cyst wall (Biotinylated streptavidin complement, Synaptophysin, x61.0).