Case Report: Floating fat globule within an arachnoid cyst

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Abstract

Intralesional floating fat globules have been reported in cystic lesions such as teratoma of the ovary and dermoid of the head and neck but not within intracranial lesions. Fat globules dispersed within the subarachnoid space are a known imaging finding of ruptured intracranial dermoid. We report a unique case of an intralesional solitary floating fat globule within a multicompartmental arachnoid cyst, with varying locations on serial imaging. We also put forward a hypothesis for the pathogenesis of fat within an arachnoid cyst. To the best of our knowledge, this is the first such report in the literature.

Key words: Arachnoid cyst; intralesional fat; magnetic resonance imaging

Introduction

Intracranial arachnoid cysts are benign CSF filled, congenital, intra-arachnoidal space-occupying lesions that represent 1% of all intracranial lesions. They occasionally become symptomatic with advancing age. On imaging, the cyst typically has the same signal intensity as CSF in all sequences. We report an unusual case of a floating fat globule within a multi-compartmental suprasellar arachnoid cyst and discuss its possible etiopathogenesis.

Case Report

A 29-year-old woman presented with a 1-year history of frontal headache not associated with features of raised intracranial pressure. She had been managed medically at a local hospital. MRI of the brain done at the onset of headache revealed a multicompartmental cystic lesion occupying the suprasellar region and extending to both Sylvian fissures. The lesion was hypointense on T1W and hyperintense on T2W images and fluid-attenuated inversion-recovery (FLAIR) imaging, with no enhancement on gadolinium injection [Figure 1A–C and F]. There was no restriction on diffusion-weighted imaging [Figure 1D and E]. A focal, well-defined intralesional differential signal intensity, showing T1 shortening and intermediate signal on T2W images, was seen in the right Sylvian fissure [Figure 2A and B]. Blooming on susceptibility-weighted imaging (SWI) and suppression on fat-saturated images suggested the possibility of a fat globule [Figure 2C and D]. Because the patient was not willing for any surgical procedure, she was managed symptomatically.

Ten months later, in view of worsening headache, she was re-evaluated by CT scan. Noncontrast CT scan revealed an iso-hyperdense lesion in the same location with the fat globule seen (hypodensity) in the suprasellar cistern [Figure 3]. Repeat MRI after 1 month showed suppression of the cyst contents on FLAIR imaging [Figure 4C] and a shift in the position of the intracystic nodule to the left anterior temporal region [Figure 4A and B]. Due to failure to control the headache with medication, the patient was taken up for decompression of the lesion. At surgery, a cerebrospinal fluid (CSF)-containing cyst with a transparent membrane was seen. The cyst collapsed on incision, revealing a yellowish, soft globule lying free within it [Figure 5]. Histopathological examination revealed flattened arachnoid cells lining a fibrocollagenous membrane, consistent with a diagnosis of arachnoid cyst [Figure 6]. Analysis of the intralesional nodule confirmed the presence of fat globules.
Figure 1 (A-F): Axial T1W MRI image (A) shows a homogenously hypointense cystic lesion (arrow) in the suprasellar cistern, extending posteriorly to the interpeduncular cistern and laterally to both Sylvian fissures. It is hyperintense (arrows) on T2W (B) and FLAIR (C) images. There is no restriction on diffusion (arrows in D and E). The cyst does not show enhancement on a post-contrast T1W image (F).

Figure 2 (A-D): Axial T1W MRI image (A) shows a hyperintense intracystic nodule with intermediate signal intensity on a T2W image (B). Blooming is seen on an axial susceptibility-weighted image (C). The hyperintense signal is suppressed completely on T1W fat-saturated spin-echo image (D). The arrows in the images indicate the nodule.

Figure 3 (A,B): Plain axial CT scans show an iso-hyperdense cystic lesion with suprasellar fat density. Note the change in position of the fat globule (arrow).

Figure 4 (A-C): Axial T1W MRI (A) and T2W MRI (B) images show the changed position of the fat globule (arrow), now situated in the left anterior temporal region. Coronal FLAIR MRI image (C) shows suppression of the cyst contents (arrow).

Discussion

Arachnoid cysts are benign, congenital, intra-arachnoidal space-occupying lesions that are filled with clear CSF. They tend to be unilocular, smoothly marginated expansile lesions and are molded by the surrounding structures. Intracranial arachnoid cysts represent 1% of all intracranial lesions. About 50%–60% are found in the middle cranial fossa; other locations include the suprasellar cistern and posterior fossa (10%), where they occur most commonly in the cerebellopontine angle cistern. Less common locations are within the interhemispheric fissure, over the cerebral convexity, in the cisterna magna, quadrigeminal cistern, and choroidal fissure.

The precise mechanism for the formation of arachnoid cysts is not known. It is possible that they are secondary to “splitting” of the developing arachnoid. A newer concept for the middle fossa arachnoid cyst is failure of the temporal embryonic meninges to merge as the Sylvian fissure forms. These two layers remain separate, forming a duplicate arachnoid. Other mechanisms might include active fluid secretion by the cyst wall, slow distention by CSF pulsations, or one-way ball-valve flow of CSF.

Although most arachnoid cysts remain stable with advancing age, they can sometimes become symptomatic due to cyst enlargement or hemorrhage. Hemorrhage may occur not...
only in the cyst but also in the subdural or extradural spaces. A vascular membrane with bridging veins may explain the propensity for intracystic hemorrhage. Previous/chronic minor intracystic blood leakage may be responsible for the hyperdensity visualized on CT scan.

The classic arachnoid cyst has no identifiable internal architecture and does not enhance. The cyst typically has the same signal intensity as CSF on all sequences. Occasionally, however, hemorrhage, high protein content, or lack of flow within the cyst may complicate the MRI appearance. In our patient, there was a change in the MRI characteristics of the lesion on repeat imaging, suggesting the possibility of a cystic lesion with varying protein content, e.g., neuroepithelial cyst, dermoid cyst, or colloid-like cyst. In view of the lack of restriction on diffusion-weighted imaging, we did not consider the possibility of an epidermoid cyst. The presence of a freely mobile globule within the lesion precluded the diagnosis of an arachnoid cyst. The intralesional nodule demonstrated high signal on T1W and intermediate signal on T2W, which was suggestive of fat/cholesterol or proteinaceous material. In view of the suppression on T1W fat-saturated imaging, the diagnosis of a fat globule was considered preoperatively. This intralesional nodule also showed blooming on SWI. The reason for blooming is not exactly known; however, exaggerated chemical shift artifact appears less likely, as the hypointensity was not uniform and not at the edges. There have been brief reports recently mentioning hypointensity of fat on SWI although the exact reason has not been discussed. The presence of fat within arachnoid cysts has not been reported in the literature. Microscopically, the arachnoid cyst wall is made up of a vascular collagenous membrane lined by flattened arachnoid cells. Arachnoid cysts lack a glial-limiting membrane or an epithelial lining. Rarely, choroid plexus–like tissue has been reported in the walls of arachnoid cysts, which supports their maldevelopmental origin.

In conclusion, we report the first case of an arachnoid cyst with a mobile fat globule within it. This possibly represents a dual congenital maldevelopment resulting in the formation of an arachnoid cyst and intracranial fat globule.

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