

Non-traumatic haemorrhage in an arachnoid cyst and ipsilateral subdural hematoma

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Abstract

A case of haemorrhage into an arachnoid cyst with no history of injury is reported. The patient had an associated subdural hematoma also. Arachnoid cysts are incidentally detected on neuroimaging. Hemorrhage into the cyst is a rare complication following head trauma. Subdural hygromas and hematomas are also rare associations and less than 30 cases of arachnoid cysts with spontaneous hemorrhage are reported in literature.

Keywords: arachnoid cyst; spontaneous intracystic hemorrhage; subdural hematoma

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Introduction

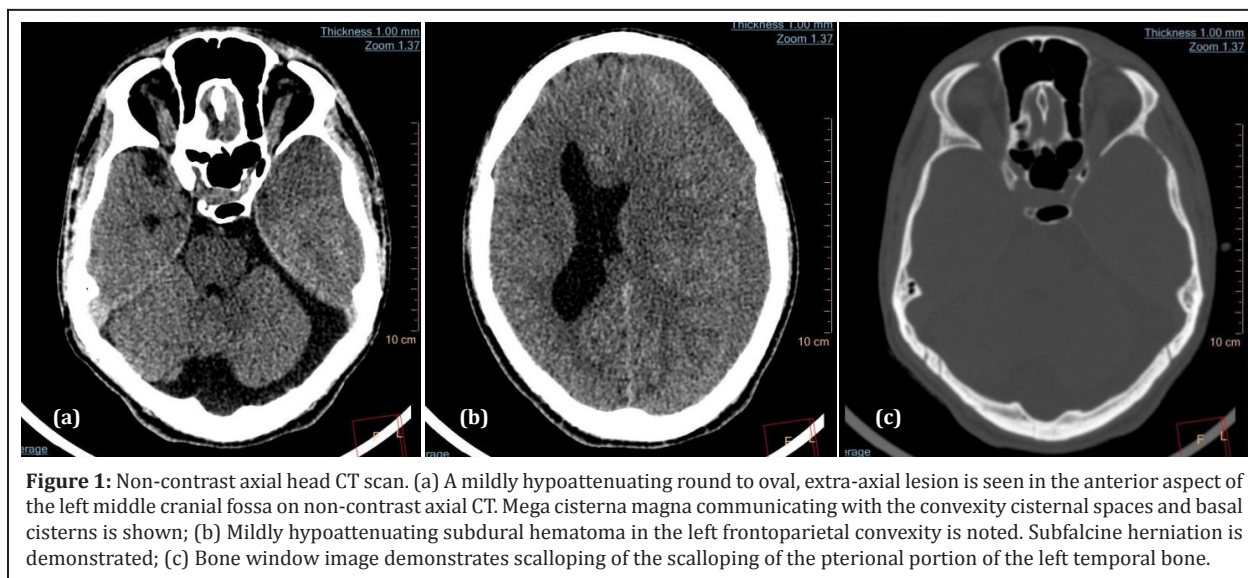
Arachnoid cysts constitute 1% of intracranial space occupying lesions. 50 to 60% are located in the middle cranial fossa [1]. Arachnoid cysts are more common on the left side and males are predominantly affected. Patients are usually asymptomatic but may present with headache, papilledema, hydrocephalus or seizures in instances when the cyst is large. Spontaneous resolution or sudden enlargement may occur [2-5]. Subdural or subarachnoid hemorrhage are rare complications into arachnoid cysts. Survey of literature reveals less than 30 cases of arachnoid cysts associated with both spontaneous intracystic hemorrhage and ipsilateral subdural hematomas [3, 4, 6-8].

Case report

A 23-year-old male presented with history of headache for 2 months at a referring hospital. At the time of presentation there was no history of nausea or vomiting. There was no antecedent head trauma. Non-contrast head CT and MRI demonstrated arachnoid cyst in the left temporal fossa and subdural hygroma along the left fronto-temporo-

parietal cerebral convexities. He was treated conservatively and brought to our hospital after an episode of seizure and vomiting with worsening of the headache. Non-contrast head CT scan showed mildly hypoattenuating round to oval arachnoid

cyst with hemorrhage within it. Left sided mildly hypoattenuating subdural collection was consistent with an early subacute subdural hematoma. Focal thinning with scalloping of the pterional portion of the left temporal bone was noted (Figure 1 a-c).



On T2-weighted MR imaging shows the mildly hyperintense arachnoid cyst in the left temporal fossa and the subdural collection had a mild hypointense signal to adjacent brain parenchyma while on T1-weighted imaging shows marked hyperintense cyst due to hemorrhage in the lesion and the subdural collection demonstrated increased signal intensity (Figure 2 a-f). These findings were consistent with early subacute hemorrhage. Following contrast administration enhancement of only the dural membrane was observed. There was no subarachnoid hemorrhage.

that these cysts form from an aberration in arachnoid development which results in splitting or duplication of the membrane, from a defect in condensation of the mesenchyme, or from abnormalities of CSF flow [6, 9, 10]. The precise cause of arachnoid cyst genesis remains inconclusive. Increasing use of neuroimaging has resulted in incidental detection of arachnoid cysts.

Frontotemporoparietal craniotomy with subdural haematoma evacuation and marsupialisation of the arachnoid cyst was performed. Biopsy of the cyst wall confirmed the presumptive radiologic diagnosis.

On both CT and MRI scans arachnoid cysts are well-defined extra-axial non-enhancing lesions with the same attenuation (on CT) or signal (on all MRI pulse sequences) as that of CSF. They do not communicate with the ventricular system and have no identifiable internal architecture. Bony erosion and remodeling, features suggestive of a longstanding process are seen in 50 % of the cases and are thought to be secondary to chronic fluid accumulation with transmitted pulsations [11]. Depending on the size and location of the cyst, adjacent deformity or hypoplasia of the subjacent brain may be present [6].

Discussion

Arachnoid cysts comprise around 1% of all intracranial space-occupying lesions reported in first two decades of life. Common locations include the middle cranial fossa, cerebral convexity, perisellar, retrocerebellar, cerebellopontine angle and quadrigeminal cisterns [1]. Most arachnoid cysts are thought to be congenital, while some arise from post-inflammatory changes after trauma, intracranial hemorrhage, or infection. It is theorized

Galassi has classified arachnoid cysts into three groups based on their size and relation with the Sylvian fissure. Type 1: Small and spindle shaped cysts which are localized in the anterior temporal pole. CT cisternography usually reveals their connection with the subarachnoid space. Type 2:

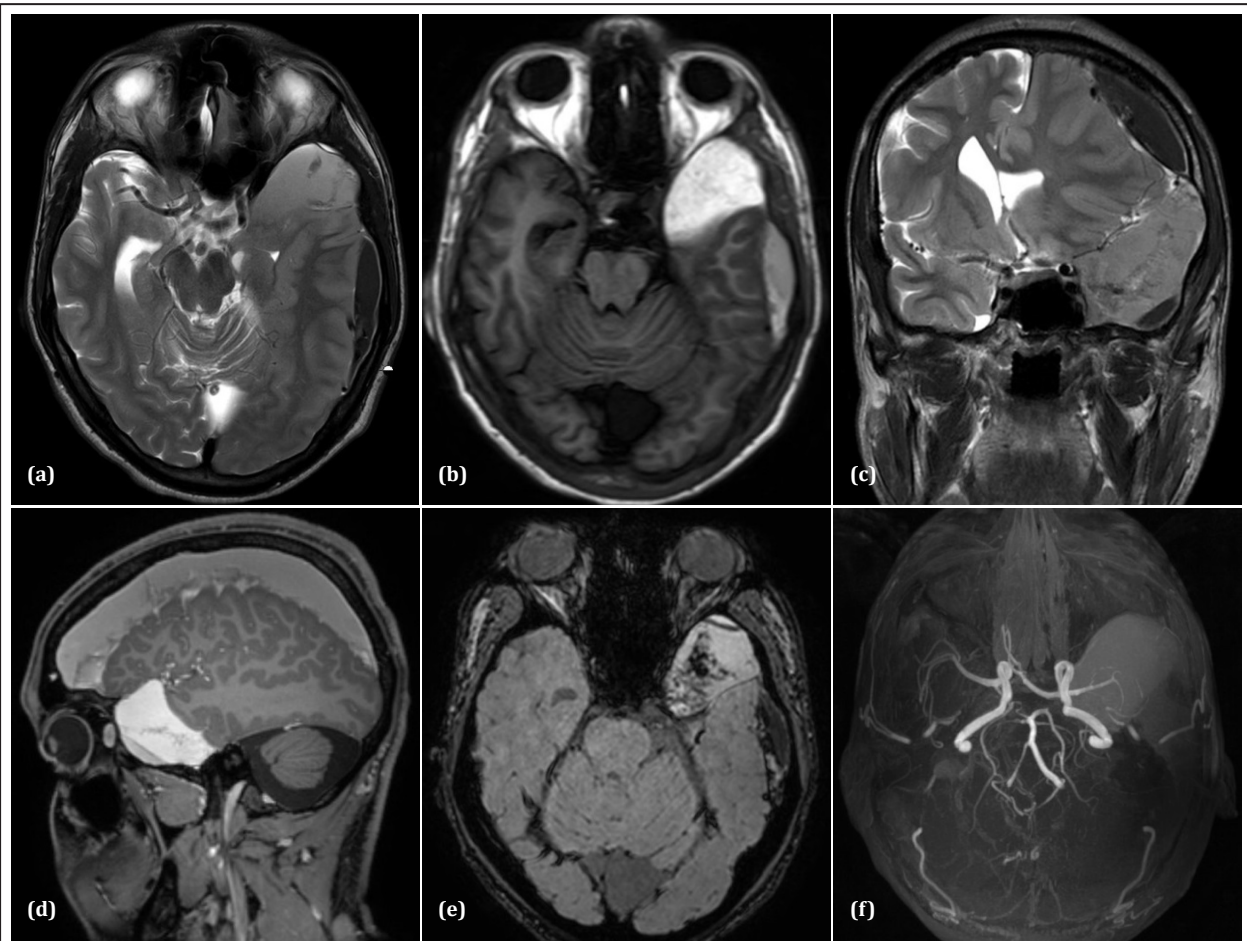


Figure 2: Axial, coronal and sagittal T-1, T2-W, GRE, TOF MRA and contrast enhanced MR images. (a) T2-W axial image shows the mildly hyperintense arachnoid cyst in the left temporal fossa; (b) Axial T1-weighted MR image shows marked hyperintense cyst due to hemorrhage in the lesion. Subdural hematoma in the low temporal convexity is noted; (c, d) Coronal and sagittal T2-W and contrast enhanced images show the hemorrhagic arachnoid cyst and dural enhancement; (e) Multiple punctate and curvilinear foci of blooming indicating haemorrhage are seen on GRE sequence; (f) TOF MRA does not reveal any abnormal vascularity.

Cysts localized in the middle and proximal parts of the Sylvian cistern. The insula is uncovered. Type 3: Cysts covering the entire Sylvian fissure leading to midline shift. They have minimal connection with the subarachnoid space [12, 13].

Trauma, aneurysm, vascular malformation and coagulopathy are common etiologies of subdural haematomas. Certain cystic and solid mass lesions are occasionally associated with intracranial haemorrhage but they are not common. Less than 20 cases of meningiomas with subdural hemorrhage have been reported [11].

A haemorrhagic epidermoid or porencephalic cyst may mimic an arachnoid cyst with intracystic hemorrhage. Most epidermoid cysts are isointense or slightly hyperintense to CSF on both T1 and

T2-weighted MR imaging. Epidermoid cysts are associated with restricted diffusion and thus show high signal intensity on diffusion-weighted imaging. Porencephalic cysts usually communicate with the lateral ventricles. They are smooth-walled cavities within brain parenchyma and isointense to CSF on T1-weighted MRI. Adjacent white matter typically shows hyperintensity on T2-weighted and FLAIR images [6].

Although most arachnoid cysts are without symptoms, an increase of intracystic fluid or more rarely hemorrhage into the cyst itself and/or adjacent subdural space may cause symptoms. Such hemorrhagic events may be post-traumatic or spontaneous. Blood accumulates within the cyst and/or subdural compartment when rupture of outer arachnoid cyst wall and surrounding fragile veins

occurs [3, 14]. Intracystic hemorrhage can also be due to interruption of the highly vascular arachnoid cyst membrane or of the bridging veins traversing the cyst cavity [15]. Spontaneous cyst enlargement and intracystic hemorrhage can be accounted by fluid production by flat arachnoid cells lining the cyst wall if sufficient intracystic pressure is exerted to breach the wall and disrupt the vasculature.

The management of an arachnoid cyst is determined by clinical presentation along with consideration for cyst size and location. Asymptomatic patients may be conservatively managed while symptomatic patients are candidates for surgery. Surgical decompression may be indicated for complicated early subdural hematomas. There is less than 0.1% annual risk for hemorrhage in patients with a middle cranial fossa arachnoid cyst [3]. Since both cyst peritoneal shunting and cyst fenestration have had good outcomes, the surgical procedure of choice is still debated [16, 17].

Conclusion

Although very rare, spontaneous intracystic hemorrhage into an arachnoid cyst may occur and possibility of such a complication should be considered in the evaluation of patients with raised intracranial pressure and associated intracranial hemorrhage elsewhere.

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Conflicts of interest

Authors declare no conflicts of interest.

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