Short report

Arachnoid cyst with rupture into the subdural space

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SUMMARY Arachnoid cysts which develop in relation to the cerebral hemispheres are usually found in the middle cranial fossa. These cysts are usually asymptomatic but can produce symptoms if there is haemorrhage into the cyst or the development of an associated subdural hematoma. Recent publications have emphasised the association of arachnoid cysts of the middle fossa with subdural haematomas. This report describes a case of an asymptomatic arachnoid cyst which ruptured into the subdural space. This event was followed by the development of symptoms despite the lack of haemorrhage.

The advent of computed tomography (CT scan) has increased the recognition and facilitated the evaluation of arachnoid cysts of the middle cranial fossa.¹ It has been suggested that the presence of an excessive contralateral shift of the midline structures and/or collapse of the ipsilateral ventricular system in a CT scan showing an arachnoid cyst of the middle cranial fossa is compatible with the presence of an associated subdural hematoma.² This complication is believed to result from tearing of blood vessels draped over the cyst following minor head trauma or rupture of the cyst.³

Case report

An 11-year-old boy was admitted to hospital for evaluation of headaches of one month's duration. The headaches started after he had been swimming, but there was no history of head trauma. The first headache was bilateral, diffuse and throbbing in nature and lasted about 24 hours. Subsequent headaches were left-sided, lasted several hours, and occurred at least once a day. They were often present upon awakening and occasionally awoke him from sleep. Their onset was sometimes heralded by a brief flash of red light which was not localised to a particular visual field. There was usually no nausea or vomiting, however, one episode of projectile vomiting, without nausea, occurred just after admission. There was no photophobia. The headaches were relieved by paracetamol and codeine. The

Fig 1A CT scan in the horizontal plane showing a lucency in the subdural space and shift of the midline structures from left to right.

patient took no other medication. Past medical history was entirely negative. There was a family history of migraine. The patient was otherwise well and did very well in school. General physical and neurological examination revealed no abnormalities except for funduscopy which showed a capillary blush and blurred disk margins. Laboratory investigations were within normal limits except for an erythrocyte sedimentation rate of 23 mm/h (Westgren). An electroencephalogram (EEG) showed focal slowing over the left temporoparietal region. A CT scan showed a large rounded lucency inferolateral to the left temporal lobe filling the floor of the left middle cranial fossa. There was a

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Fig 1B High resolution CT scan in the coronal plane showing the confluence of the subdural and infratemporal lucencies and thinning of the bone in the left middle cranial fossa.

rim of lucency which was believed to be a distended subdural space over the left frontal and parietal lobes. The midline structures showed a moderate shift to the right (fig 1A). These radiologic findings, and the acute onset of headaches, suggested the presence of an arachnoid cyst and associated chronic subdural haematoma. Coronal sections were obtained using a high resolution CT scan which confirmed the fact that the lucency was displacing the left temporal lobe superiorly and appeared to show thinning of the bone in the left middle fossa, and also the confluence of the two lucencies already noted on the horizontal scan (fig 1B). A craniotomy confirmed the presence of an arachnoid cyst communicating with the subdural space. The fluid, under increased pressure, was indistinguishable from CSF and there was no sign of bleeding. Histopathologic examination of the wall of the cyst was consistent with arachnoid membrane.

Discussion

It has been suggested in the past that many cases reported as arachnoid cysts are actually chronic subdural hygromas.⁴ The same author suggested that many cases diagnosed as arachnoid cysts with

chronic subdural hygroma really represented external hydrocephalus *ex vacuo*, the CSF filling unoccupied space following agenesis of the temporal lobe. He called this temporal lobe agenesis syndrome. The findings in the present case are not consistent with this hypothesis. If the accumulation of CSF in the middle fossa represented external hydrocephalus *ex vacuo* then the fluid would not have been under increased pressure, could not have ruptured into the subdural space and could not have caused a shift of midline structures.⁵ Two possible mechanisms can be considered to explain the development of a communication between the cyst and the subdural space. First, it is possible that a communication developed between the cyst and the subarachnoid space. This may have resulted from a tear in the cyst wall due to minor head trauma, with a flap-valve effect, allowing passage of CSF into the cyst whenever there was an increase in intracranial pressure. Under these circumstances the cyst would gradually increase in size and there would be a shift of the midline structures and compression of the ipsilateral ventricular system. An enlarged cyst of this type, under increased pressure, could rupture into the subdural space. Alternatively, the rupture of the arachnoid cyst into the subdural space may have been an acute event, since the development of headache was sudden in this case. This mechanism would require rupture of the cyst following a transient increase in intracranial pressure during swimming, which is often associated with a Valsalva manoeuvre.

Because of acute onset of symptoms, the recent emphasis on the association between arachnoid cysts and subdural hematoma, and the CT scan appearance, the initial diagnosis of arachnoid cyst and subdural haematoma was made in this case and was only refuted by the operative findings. However, this patient is possibly unique in that the arachnoid cyst produced symptoms by rupture into the subdural space without associated haemorrhage. This case demonstrates that findings on CT scan consistent with an arachnoid cyst of the middle fossa, in the presence of excessive contralateral shift of the midline structures and/or collapse of the ipsilateral ventricular system, does not necessarily indicate the presence of an associated subdural hematoma. Consequently, the demonstration of an arachnoid cyst of the middle fossa by CT scan, associated with a lucency in the subdural space, suggests that the cyst may have ruptured into the subdural space.

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