Case Report

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Subdural haemorrhage secondary to arachnoid cyst rupture from scuba diving

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ABSTRACT

We describe a case of intracranial arachnoid cyst rupture leading to ipsilateral subacute subdural haemorrhage secondary to scuba diving in a previously healthy adult male. Our patient required urgent trephination of the cranium to evacuate the subdural haemorrhage and recovered well post operatively. To our knowledge this is the first case reported in the literature of arachnoid cyst rupture with concomitant subdural haemorrhage from scuba diving. This case may have implications for information provided to patients with arachnoid cysts.

Keywords: Arachnoid cyst, Subdural haemorrhage, Scuba diving

INTRODUCTION

Arachnoid cysts are congenital lesions, generally considered a benign pathology which develop from splitting or duplication of the arachnoid membrane. These cysts have a predilection to occur in the middle cranial fossa, favour the left side and are more common in males with a prevalence of approximately 1.4%. 2-4

In adults, the majority of arachnoid cysts are asymptomatic and usually represent an incidental finding.^{1,5} Although rare, there are published reports of subdural haemorrhage secondary to arachnoid cyst rupture in adults. On review of these cases, minor head trauma predominated as a precipitating factor for cyst rupture and subsequent subdural haemorrhage. In several cases arachnoid cyst rupture appeared spontaneous.⁵ There are no published cases of arachnoid cyst rupture and subdural haemorrhage caused by scuba diving in the literature.

CASE REPORT

A 19-year-old male with no significant past medical history presented to hospital via ambulance after experiencing 8 weeks of headaches. Prior to admission the headaches had intensified over the past 4 days, he briefly collapsed one day prior to admission and his symptoms prevented usual daily activities. He denied any history of head trauma and had not participated in contact sports. He had been open water scuba diving 8 weeks prior and again four days prior to admission; on both occasions this corresponded to development of headaches. Systems review and general examination was Neurological examination was normal. documented as unremarkable; pupils were 4mm equal and reactive to light, there was no motor deficit and GCS was 15. A non- contrast CT brain was performed showing a large left fronto-parietal convexity subacute subdural haemorrhage (SDH) with maximal depth of 25mm (Figure 1). This was associated with midline shift of 12mm and significant mass effect with partial effacement of the left lateral ventricle and evidence of subfalcine and early uncal herniation. In addition, there was an arachnoid cyst in the region of the cisterna magna and a left middle cranial fossa (MCF) hyperdensity measuring 43x32x26mm in communication with the SDH. A CT angiogram was performed to exclude aneurysmal pathology, which was normal.

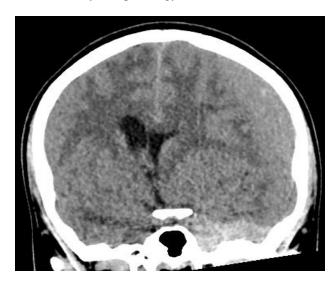


Figure 1: Non contrast CT brain- Coronal plane CT demonstrates thick isodense left SDH in communication with the hyperdense blood in the MCF floor. Note the midline shift and mass effect causing partial effacement of the lateral ventricle.

The patient was transferred via helicopter to our tertiary Neurosurgery centre. He was mildly hypertensive with intermittent bradycardia on arrival, potentially due to raised intracranial pressure. Although the previous neurological examination was reported as normal, examination now revealed a left sided hemiparesis (Kernohan's phenomenon), subtle left lower motor neuron facial nerve palsy and partial right abducens nerve palsy signalling deterioration. He was therefore taken to the operating theatre for left frontal and parietal burr hole evacuation of the subacute SDH.

He recovered well post procedure with no new neurological deficits. Complete resolution of the left hemiparesis and left facial nerve palsy was noted several days post operatively. A subtle right abducens nerve palsy remained at discharge and had resolved at 3 month follow up. A full coagulation profile excluded an underlying blood dyscrasia.

Post-surgery an MRI was performed to further characterise the left MCF hyperdense lesion. This was consistent with an arachnoid cyst (AC) with evidence of intracystic haemorrhage with layered haemorrhagic products (Figure 2). The patient was informed about the arachnoid cyst and management options were discussed at follow up.

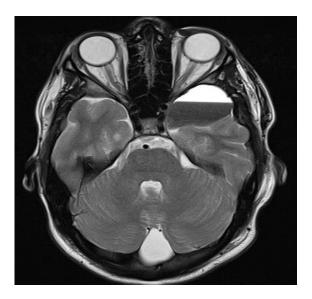


Figure 2: MRI brain- Axial T2 weighted MRI brain demonstrates a left MCF AC, with a fluid level interface demonstrating haemorrhage product and CSF. The posterior fossa AC is also appreciated.

DISCUSSION

Although considered a benign pathology, the literature demonstrates that patients with arachnoid cysts may have a predisposition for haemorrhagic manifestations. Studies suggest the pathogenesis of haemorrhage from ruptured arachnoid cysts caused by minor trauma results from disruption of the vascular arachnoid cyst membrane and tearing of the bridging veins which traverse the cyst.^{6,7} The event of arachnoid cyst rupture and associated subdural haemorrhage is nonetheless a rare event.² The aetiology of spontaneous arachnoid cyst rupture remains to be elucidated. In our case we suspect barotrauma sustained during open water scuba diving was significant enough to cause cyst rupture and subsequent haemorrhage. Scuba diving exposes the patient to changes in atmospheric pressure and can result in barotrauma.8

The vast majority of arachnoid cysts are asymptomatic and represent an incidental finding.² As intracranial imaging becomes more frequent in clinical practice there will be an increased frequency in diagnosis of arachnoid cysts. For this reason it is imperative that we are able to adequately inform patients of the risks of this pathology. Previous large natural history studies of adult patients with arachnoid cysts found incidental arachnoid cysts were unlikely to become symptomatic or change and further, found a generally benign natural history also occurred in the paediatric population.^{2,9}

While these large natural history studies suggest limited risk of complications associated with incidental arachnoid cysts, the literature demonstrates this risk is not zero. Our case highlights a subset of patients who will need appropriate advice in regards to potential risk associated with participating in sports such as scuba diving or other activities in which barotrauma may factor with an arachnoid cyst. Although this is the first reported case of subdural haemorrhage secondary to arachnoid cyst rupture from scuba diving, it is unlikely to be an isolated event. Despite the lack of published cases, we are of the opinion that scuba diving with an arachnoid cyst poses a risk for haemorrhage.

This case highlights the need for doctors to be aware of the risk of arachnoid cyst rupture and subsequent haemorrhage. This should prompt appropriate counselling and education for patients to allow them to risk stratify their participation in this sport or other activities in which barotrauma may result. Our patient intends to return to open water scuba diving, he was offered fenestration of the MCF arachnoid cyst to reduce the risk of further haemorrhagic events.

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