



Snakes alive! Caput medusae due to cerebral venous angioma

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The increasing sophistication and availability of modern cranial imaging techniques has resulted in the detection of incidental lesions in greater numbers. Caution needs to be exercised when deciding whether these lesions are responsible for the patient's symptoms and whether treatment is merited. We present a case where two coincident brain lesions led to a referral for possible neurological surgery.

Case report

A 45-year-old man presented to the emergency department of a Welsh district hospital with a 1-week history of persisting headaches and dizziness after hitting the top of the right side of his head forcefully against an overhead lamp. On examination, no abnormal neurological findings were found.

In view of his lingering symptoms, an un-enhanced computer tomographic (CT) scan of his brain was performed. This showed a small, superficial right-frontal haematoma (Figure 1). He underwent a magnetic resonance imaging (MRI) scan which was unremarkable except for the haematoma. The patient subsequently had a digital subtraction cerebral angiogram which revealed a leash of vessels converging onto a dilated central vessel which drained into the superior sagittal sinus (Figure 2).

Figure 1. Un-enhanced computed tomographic scan of brain showing the superficial right-frontal haematoma.



Figure 2. Digital subtraction cerebral angiogram during the venous phase. The arrow points to the 'caput medusa' of the cerebral venous angioma. The 'X' marks the position of the intracerebral haematoma relative to the venous anomaly.



At this stage, the patient was referred to the neurosurgical team for consideration of surgery. When we reviewed the angiograms we found that the vascular lesion was consistent with a cerebral venous angioma. Moreover, this anomaly was distal to the location of the haematoma. There were no other vascular abnormalities. The frontal haematoma was a contusion secondary to the patient's head injury. He was treated conservatively and made a full recovery.

Discussion

The actual prevalence of cerebral venous angiomas (CVA) is probably higher than the quoted 3% of the population¹—as this figure has been derived from brain imaging and autopsy studies.

CVAs are developmental anomalies composed entirely of venous structures interspersed with normal brain parenchyma and are found mainly in the posterior fossa, cerebral cortex, and the deep white matter.² The pathogenesis of CVAs is unknown but may represent attempts at establishing a collateral venous circulation following an ischaemic insult during the development of medullary veins and their tributaries.³ CVAs therefore drain normal brain tissue.

CVAs are usually discovered incidentally during investigations for unrelated neurological disorders. They have a characteristic angiographic appearance which has been likened to the head of the Medusa (caput medusae).² They appear as stellate structures on contrast-enhanced MRI scans unless obscured by the presence of haematoma.² The incidence of second cerebrovascular malformations in patients with known CVAs is as high as 19%.⁴

The majority of these coincident vascular malformations are cavernous haemangiomas and there is a possibility that the development of both these malformations may be related.⁵ CVAs had previously been associated with various neurological symptoms and haemorrhage. However, there is increasing evidence to suggest that venous angiomas rarely, if ever, bleed.^{2,4}

Most recent studies suggest that neurological symptoms attributed to CVAs (especially haemorrhage) are probably due to associated cavernous malformations, arteriovenous fistulas, or unidentified vascular malformations.^{2,4}

Patients found to have a CVA should ideally have at least an MRI to exclude concurrent vascular malformations as these are more likely to give rise to symptoms. Due to the low morbidity associated with bleeding (if it was actually due to a CVA) and the high complication rates when these lesions are treated with either surgery or radiosurgery, the majority of neurosurgeons now treat cerebral venous angiomas conservatively.^{2,4}

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References:

1. Sarwar M, McCormick WF. Intracerebral venous angioma: Case report and review. *Arch Neurol.* 1978;35:323–5.
2. McLaughlin MR, Kondziolka D, Flickinger JC, et al. The prospective natural history of cerebral venous malformations. *Neurosurgery.* 1998;43:195–201.
3. Saito Y, Kobayashi N. Cerebral venous angiomas: Clinical evaluation and possible etiology. *Neuroradiology.* 1981;139:87–94.
4. Rigamonti D, Spetzler RF, Medina M, et al. Cerebral venous malformations. *J Neurosurg* 1990;73:560–4.
5. Wilms G, Belus E, Demaerel P, et al. Simultaneous occurrence of developmental venous anomalies and cavernous angiomas. *AJNR.* 1994;15:1247–54.