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CASE REPORT

Brain stem infarction complicating a traumatic carotid cavernous fistula

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Introduction

Life-threatening complications associated with carotid cavernous fistulae (CCF), such as intracerebral hemorrhage, increased intracranial pressure, cerebral ischaemic injury, blindness, and massive epistaxis, have been reported previously.^{4,5,7–9} Rare reports are available regarding fatal brain stem infarction as a complication of CCF bleeding. We report a rare patient who suffered from a fatal brain stem infarction due to traumatic CCF bleeding and basilar artery vasospasm.

Case report

A 24-year-old man sustained a head injury in a motorcycle accident 10 days prior to presentation at our hospital. Initially, the patient was admitted to a community hospital for supportive treatment. The diagnoses were a right zygomatic arch fracture,

right frontal bone fracture, a small right frontal intracerebral haemorrhage, and traumatic subarachnoid haemorrhage. Deterioration of consciousness developed in a few days, and the patient was transferred to our hospital for further evaluation and management.

On admission, the patient's Glasgow Coma Scale (GCS) revealed E2V2M5. High fever and neck stiffness were noted. The physical examination demonstrated mild bilateral proptosis and "red eye". No bruit was noted on the neck area. Traumatic subarachnoid haemorrhage, right zygomatic arch fracture, right frontal bone fracture, a right frontal intracerebral haemorrhage, and hydrocephalus were evident on the brain computed tomography (CT) scan (Fig. 1). With a diagnosis of hydrocephalus and a suspicion of meningitis, the patient underwent an emergent external ventricular drainage. The intra-operative finding was raised cerebrospinal fluid (CSF) pressure (more than 15 cm H₂O). Clear and colorless CSF was noted. The average amount of CSF from the external ventricular tube was 300 ml per day. The study of CSF revealed no abnormality and the fever subsided on the next day.

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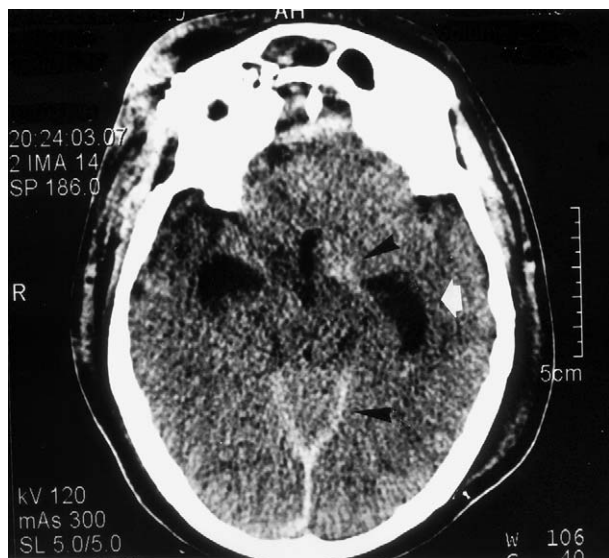


Figure 1 Brain CT scan showed diffuse subarachnoid haemorrhage (arrowheads) with hydrocephalus (arrows).

On the 6th day after admission, a brain CT scan was arranged since there appeared to be no obvious improvement of the patient's neurological condition. The CT scan showed a suspicion of new subarachnoid haemorrhage and right CCF reflected by an engorged right ophthalmic vein (Fig. 2). Cerebral angiography revealed a right carotid-cavernous fistula, a right carotid-ophthalmic vein fistula, and poor opacity of the basilar artery with vasospasm (Fig. 3). Cerebral angiography with embolization was arranged, and successful embolization of the carotid-cavernous fistula was achieved (Fig. 3D). However, the condition of this patient did not improve following successful embolization. A decreased GCS was apparent on the 8th day after admission (GCS:E1V1M4). Brain CT scan



Figure 2 Brain CT scan 6 days after ventriculostomy showed an engorged right ophthalmic vein (arrowheads).

showed low attenuation over the brain stem and cerebellar area (Fig. 4). The patient entered a deep coma on the following day and expired on the 10th day after admission.

Discussion

Carotid cavernous fistulae typically arise following trauma or from a spontaneous source such as a ruptured aneurysm that results in a direct shunt between the internal carotid artery and the cavernous sinus.¹ Traumatic CCF usually occurs in young men. The high-flow pattern was classified by anatomical and angiographic characteristics as a direct or Type A CCF.¹ The symptoms often have an acute onset and progress rapidly.¹ The complications of CCF include haemorrhage, increased intracerebral pressure, cortical venous hypertension, decreasing visual function, progressive proptosis, and cerebral ischaemia.^{4,5,7-9} A CCF with the unusual complication of brain stem dysfunction caused by poor extracranial venous drainage and obstruction to venous drainage from the brain stem has been reported.^{7,9} Our patient's angiography demonstrated a right CCF, multiple false aneurysms of the internal carotid artery, and vasospasm of the basilar artery (Fig. 3). Presumably one of the false aneurysms of the internal carotid artery ruptured into subarachnoid space and caused subarachnoid haemorrhage. The basilar artery vasospasm might have been induced by the subarachnoid haemorrhage. These findings were compatible with vasospasm of the basilar artery and subsequent brain stem and cerebellar infarction.

The goals of treatment are to eliminate the fistula and to maintain the patency of the internal carotid artery.^{1,3} The earliest successful treatment for CCF apparently occurred in 1809 when Travers successfully occluded a CCF by ligating the common carotid artery.⁶ The modern era of endovascular surgery commenced with Prolo and Hanberry. They successfully occluded CCFs with non-detachable balloons in 1971.⁶ By 1974, Serbinenko had developed detachable balloons that occluded the fistula and preserved the ICA. Later, Debrun used latex detachable balloons to occlude direct CCFs and achieved an ICA patency rate of 59%.^{2,6} At present, transarterial detachable balloon embolization is considered the initial treatment modality for direct CCFs.^{2,6,10} Direct type fistulas rarely resolve spontaneously; they almost always require urgent and immediate treatment if progressive visual loss, corneal exposure, intolerable retro-orbital pain or bruit, cortical venous drainage, or massive epistaxis occur.^{3,4,7} Our patient underwent an urgent embolization of CCF after the diagnosis was made.



Figure 3 Cerebral angiography (A, B: pre-embolization; C, D: post-embolization) demonstrated a direct type right carotid cavernous fistula. (A) The anteroposterior projection of right carotid artery angiography revealed a large vascular pouch over the cavernous sinus. Pseudoaneurysms (arrows) were evident on this angiogram. (B) Lateral projection of vertebrobasilar arteries showed long segment narrowing of the basilar artery (arrows). (C) Lateral projection of right carotid artery angiography showed four detachable balloons occluding the carotid cavernous fistula (arrows). (D) Lateral projection of right carotid artery angiography showed occluded carotid cavernous fistula and a residual fistula to the ophthalmic vein (arrowhead).

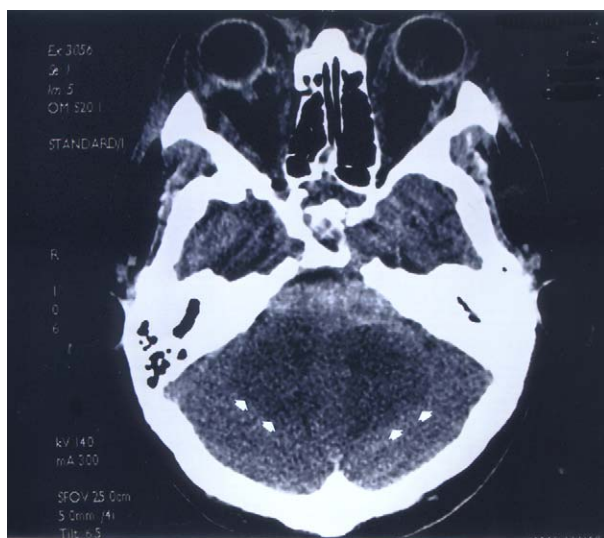


Figure 4 The brain CT scan revealed subarachnoid haemorrhage ventral to the brain stem, and low attenuation on the brain stem and cerebellar area (white arrows).

Although the embolization was successful, the fatal brain stem infarction could not be avoided. The patient entered a deep coma on the following day and expired. The vasospasm in the basilar artery was probably secondary to subarachnoid haemorrhage. The combination of subarachnoid haemorrhage causing spasm and the relative venous obstruction or raised venous pressure due to the CCF might cause the brain stem and cerebellar infarction.

Conclusion

We have reported a unique case of traumatic CCF in a patient who suffered a fatal brain stem infarct resulting from basilar artery vasospasm. Although this type of fatal complication is rare in traumatic CCF, doctors should keep in mind the possibility of this condition, especially in light of the high mortality and morbidity with CCF bleeding. We suggest

that traumatic CCF should be diagnosed early and treated aggressively.

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