PENETRATIVE INJURY TO THE FACE RESULTING IN DELAYED DEATH FOLLOWING RUPTURE OF A CAVERNOUS SINUS ANEURYSM ON THE CONTRALATERAL SIDE

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Introduction

Delayed deaths following assault give rise to medico-legal issues such as whether the assault was directly responsible, did it contribute or was it totally unrelated to the death. We present the case of a patient who sustained penetrative trauma to the face resulting in the formation of a contra-lateral, carotid-cavernous fistula, which resulted in aneurysm formation, delayed rupture and death. A literature search failed to find a similar case.

Case history

A 40 year old intoxicated male was assaulted with an unknown weapon onto his face. On admission to hospital the only external injury was a 2cm long penetrative injury just below the **left** eye, which was sutured. Examination revealed nasal bleeding and total left subconjunctival haemorrhage. Visual acuity was 6/6. The left pupil was 2mm and sluggishly reactive while the right pupil was 5mm and not reactive. No other neurological lesions were present. Two days later he developed chemosis, complete ptosis, non-reactive 6mm pupil and IIIrd, IVth & VIth nerve palsies on the **right** side. Axial Computerised Tomography (CT) images of the brain showed a 3x2cm haemorrhagic contusion of right temporal lobe with a high density area within it and fracture of ethmoidal sinus was queried (Fig 1). He was managed conservatively and discharged six days later and followed up in the neurosurgical clinic. Two weeks later he was again admitted with epistaxis, hematemesis and melena. The right ophthalmoplegia was still present with a dilated non reactive pupil. The left eye was normal. Eye referral, another CT brain and CT para nasal sinuses was done, but the patient left the hospital against medical advice. One week later, which was one month after the assault, he was again brought to the hospital with a severe headache and hematemesis and died soon after admission.

At autopsy, 1000ml of blood was within the stomach and the small intestine contained blood. The cause of death had been given as haemorrhagic shock following upper gastro intestinal bleeding. However, as the relatives were not satisfied with the cause of death, a second autopsy was ordered by the magistrate. Two days later a second autopsy was carried out by the first author on an embalmed body with already dissected organs.

A 2cm long transversely placed healing scar was present under the lateral end of the left eye (Fig 2). Intra cranially an organized contusion on the anterior pole of the right temporal lobe was seen. A 2cm long linear basilar fracture was in close proximity to the pituitary fossa. There was fragmentation and erosion of bone around the pituitary fossa with blood clots within the cavity (Fig. 3). Middle ear cavities contained blood bilaterally. Hospital notes and CT scans of each admission were obtained following the autopsy. Review of the axial CT images of the brain taken on the second admission showed an area of high density in the right temporal region measuring 2x2.5cm merging with the cavernous sinus medially giving the appearance of an expanded right cavernous sinus. An 8x3mm very high density area suggestive of a bony

fragment was noted within the above mentioned area (Fig 4). There was evidence of brain oedema with mild mass effect in the temporal region. On review of the coronal images a complex blow out fracture of the left orbit with opacification within the left maxillary antrum, ethmoidal air cells and sphenoid sinus was observed (Fig 5). Findings at the second autopsy, together with a review of the hospital notes and CT scans, indicated that the penetration under the left eye had extended into the cranial cavity causing a traumatic, direct carotid-cavernous fistula on the contra lateral side, which resulted in aneurysm formation, delayed rupture and death. The police were informed that the death was directly related to the assault which took place one month previously and that a pointed, thin, long, narrow weapon was used. A search of the assailant's house revealed an umbrella with a thin, pointed metal tip. He was arrested and arraigned on a charge of homicide.

Discussion

Abnormal communications between the cavernous sinus (CS) and the carotid artery can be classified by aetiology (traumatic; spontaneous; iatrogenic), by flow dynamics (low flow; high flow) and by anatomy (direct or dural, internal carotid artery (ICA) or external carotid artery). In direct carotid-cavernous fistulas (CCF) a communication occurs between the ICA and the CS. In this case, a traumatic, high flow, direct fistula between the ICA and the CS was caused. i.e. a Barrow type A fistula[1]. CCF are seen in only 0.2% - 0.3% of craniofacial trauma[2], and of these, seventy- five percent of direct CCF are due to head injury following road traffic accidents or falls[3]. In a series of 100 cases of direct CCF' only one was due to a knife stab to the orbit.[4]. Traumatic CCF are almost always direct and caused by laceration of the ICA within the CS. The high percentage of traumatic CCF is due to the anatomical relationship between the ICA, the CS and the skull base. Since the cavernous ICA is fixed to the dura of the skull base, thus limiting its mobility, it is exposed to shearing forces. Further, since it is the only anatomic location in the body in which an artery is completely surrounded by a venous structure, the shearing forces causes a laceration of the siphon resulting in a traumatic direct CCF which is a rare life-threatening disorder.

Traumatic CCF leads to arteriovenous shunting which usually causes ocular signs[3]. The manifestations are usually unilateral and ipsilateral, but may be bilateral or even contralateral because of the connections between the two cavernous sinuses[5]. Symptoms are often abrupt in onset and rapidly progressive. However, the severity of ocular symptoms can vary widely and does not always correlate with the fistula gradient[6].

A bruit occurs in 80%, proptosis in 72%[4] and diplopia in 60-70% of the cases[5]. However these have not been noted in this case. Retro-orbital pain and pain in the eye which occur in 50-60% and abducens palsy which occurs in 49-85% of patients[5] was detected, as well as the palsies of the oculomotor and trochlear nerves. Due to the medial location in close proximity to the ICA of the VIth nerve within the cavernous sinus it is affected more than the IIIrd and IVth nerves which are on the lateral wall of the cavernous sinus. In the case under discussion, in addition to the cranial nerve palsies, parasympathetic fibres carried in the IIIrd nerve was also affected resulting in a dilated pupil.

Haemorrhage is a devastating complication of a CCF. This may be internal (intracerebral or subarachnoid) or external (epistaxis or ottorrhoea) Massive epsitaxis is rare[7] and in a study of 127 cases of direct CCF there were only four (3.1%) who manifested epistaxis[8]. Of these, two

were fatal. In all four cases angiography showed a pseudoaneurysm or venous pouch had entered the sphenoid sinus via a communication through a basal skull fracture. In this case too, a basal skull fracture was detected at autopsy.

Although it is recommended that patients with head trauma who present with sphenoid sinus fractures and massive epistaxis should be evaluated for the development of traumatic aneurysms as soon as possible[9], this was not done in this case, probably since the facial trauma was considered minor. If the patient exhibits fractures without epistaxis, angiography should be deferred for 2 to 3 weeks; if the first angiographic evaluation reveals normal findings; repeated epistaxis should prompt a second angiographic evaluation. Current treatment of traumatic aneurysms involves occlusion of the main artery through the use of detachable balloons or platinum coils[10].

The probable sequence of events that led to death was arrived at from the clinical, CT scan and autopsy findings. The penetrative trauma under the left eye fractured the medial left orbital wall, penetrated through the left ethmoidal air cells into the contralateral middle cranial fossa causing a bony fragment to lodge in the right temporal lobe. This penetration must have damaged the right cavernous sinus and also the right carotid artery, with the formation of a carotid cavernous fistula. Gradual dilatation and subsequent rupture of the right cavernous sinus took place leading to massive haemorrhage through the basal skull fracture, manifesting as epistaxis and resulting in death. The swallowed blood seen in the stomach at the time of the 1st autopsy resulted in the error in the cause of death.

In this case, the low index of suspicion due to the minor trauma (2cm laceration) sustained on the contra lateral side of the face to the ophthalmoplegia, the patient leaving the hospital against medical advice prior to the 2nd CT being reported and the extreme rarity, contributed to the death.

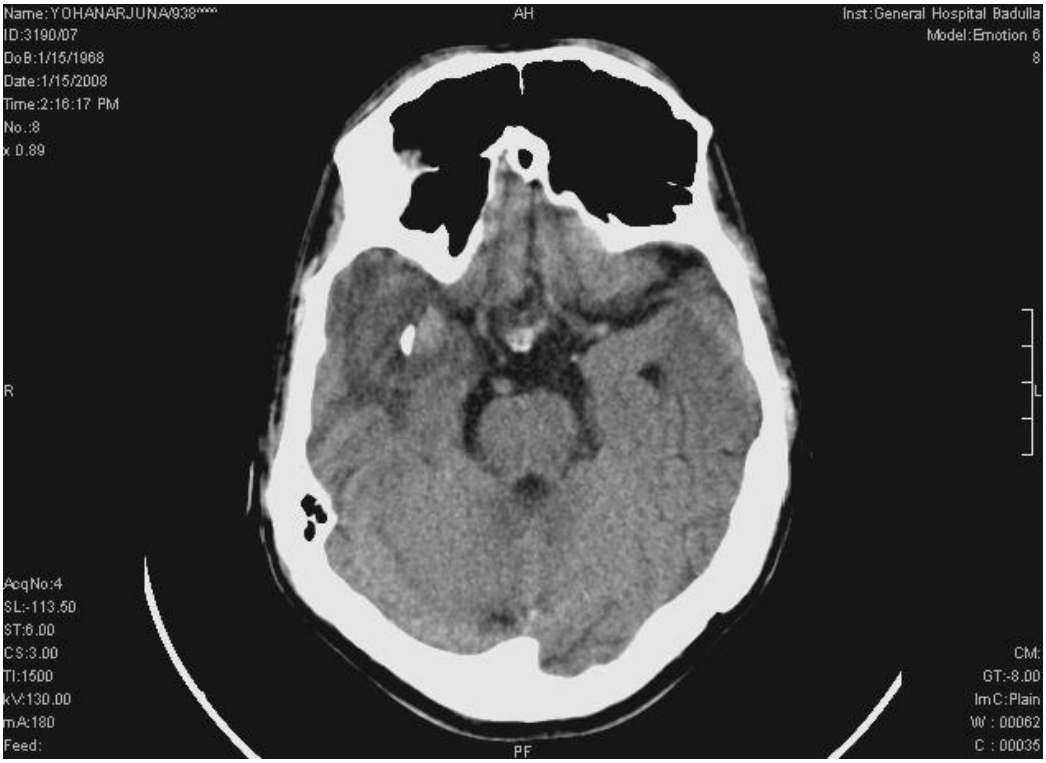

Fig 1 - Axial CT Brain on first admission

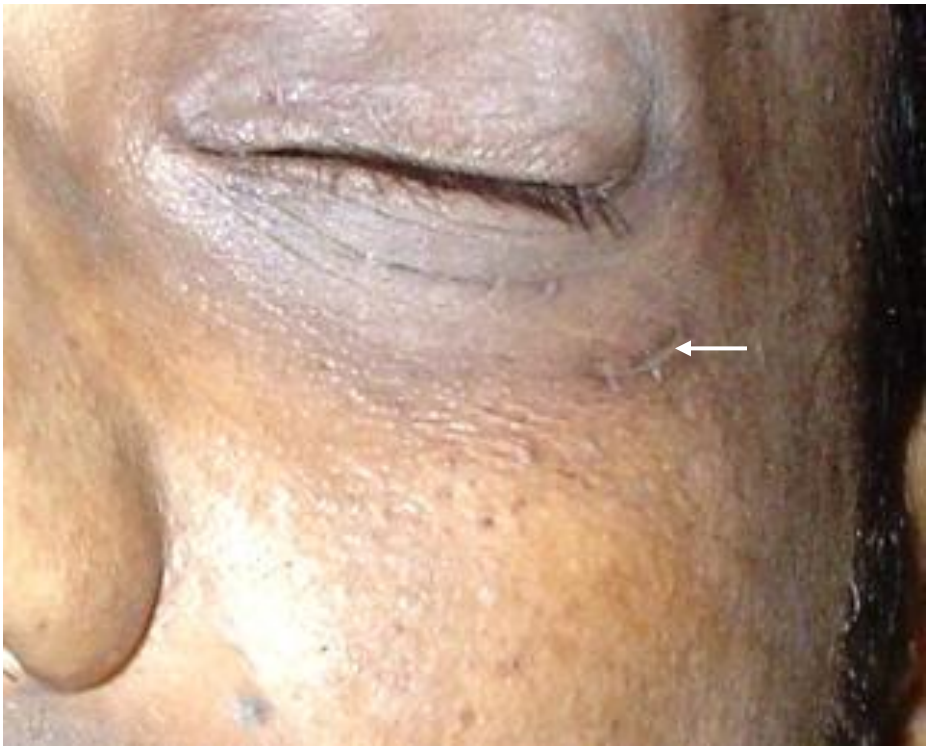

Fig 2 – healing scar beneath left eye

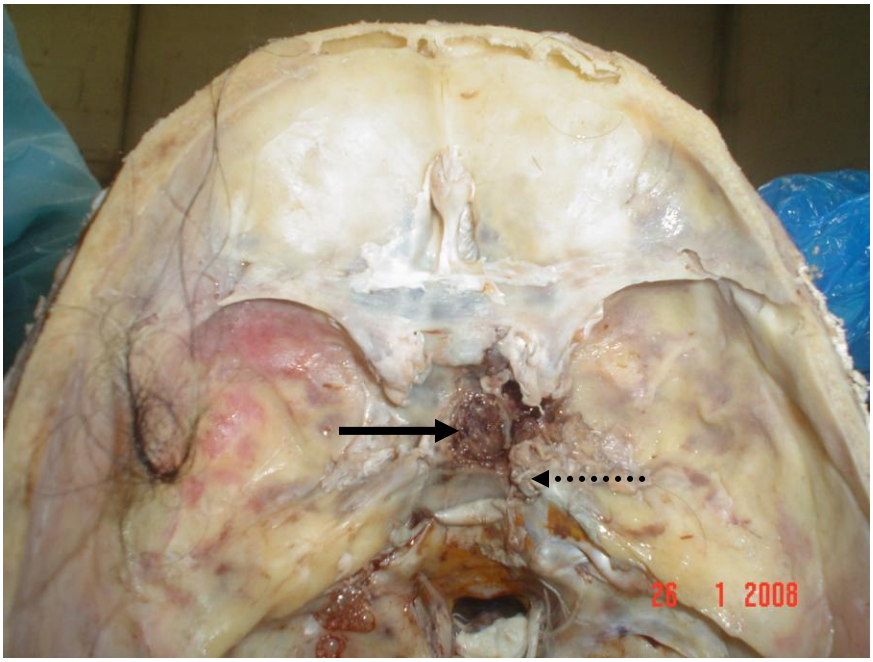

Fig 3 – linear fracture (dotted arrow) and erosion of pituitary fossa (thick arrow)

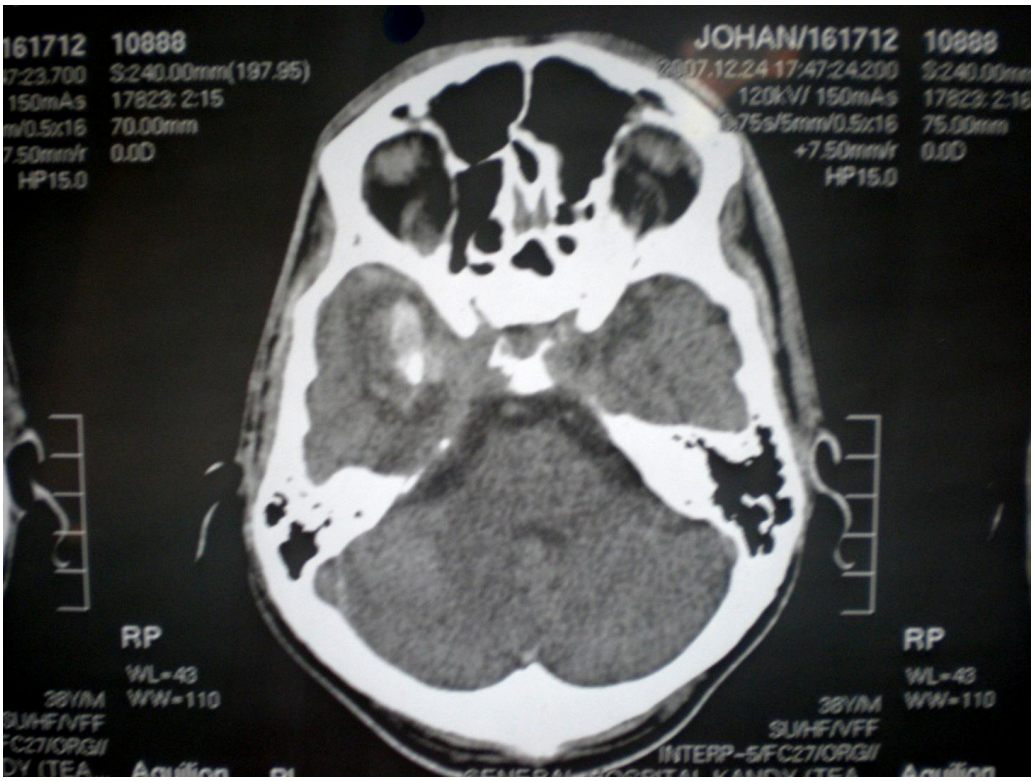

Fig 4 - Axial CT Brain on second admission

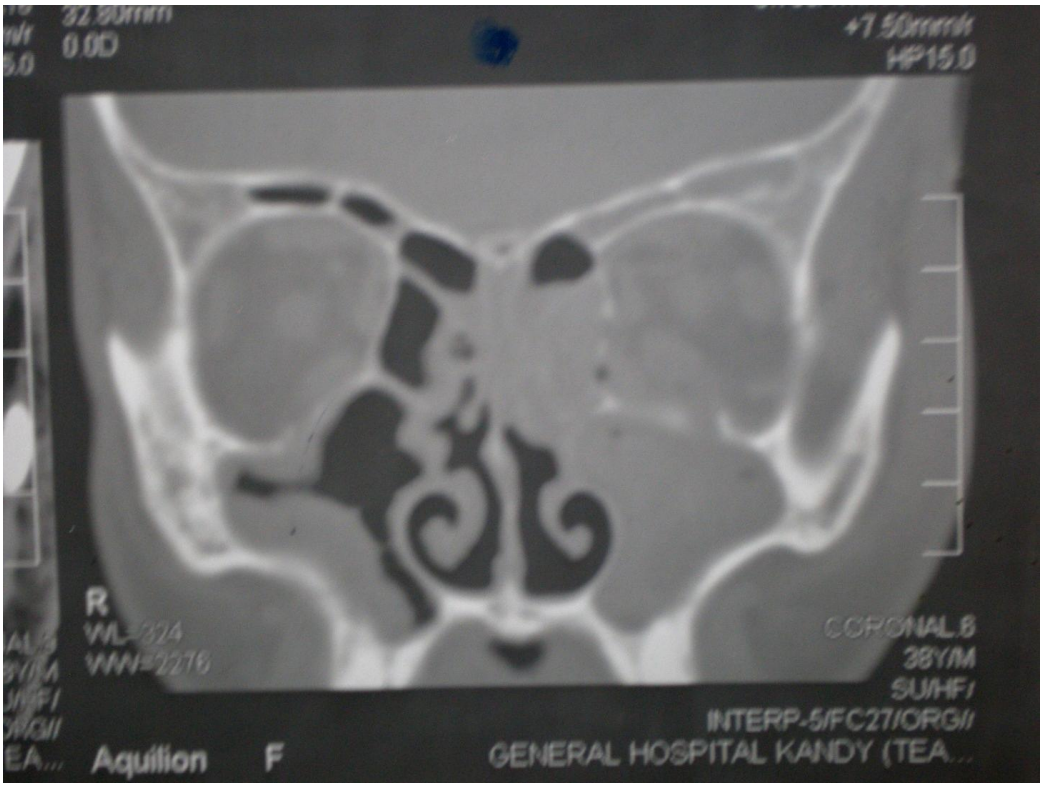

Fig 5 – Coronal CT orbits on second admission

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