



CASE REPORT

Bilateral anterior cerebral infarcts from a depressed anterior base of skull fracture: Case report and review of the literature

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Case report

On the 24th May 2003, the Aeromedical Retrieval Service Careflight International[®], Australia was tasked to retrieve and transfer a patient from Noumea, New Caledonia, in the Pacific Ocean, to a neurosurgical unit in Sydney, Australia. The patient was a 6-year old native Noumean girl who had sustained a blunt head injury after a fall of approximately 2 m while playing on a building site. On arrival at the hospital, the patient was haemodynamically stable (BP 110/70, pulse: 100) but had a Glasgow Coma Score (GCS) of 6/15. She was intubated and ventilated in the Emergency Department. The pupils were normal and reactive and the only other abnormal sign noted was rhinorrhea.

CT scanning carried out at the hospital in Noumea revealed an anterior base of skull fracture with disruption of the sella turcica (Fig. 1) with depression of the anterior fragment. There was a small intracerebral haematoma and pneumocephalus in

the left frontal lobe (Fig. 2). At this stage there were no clinical or radiological signs of raised intracranial pressure. Further images showed dense bilateral frontal lobe infarctions (Fig. 3) possibly from occlusion or disruption of the anterior cerebral arteries bilaterally.

The child remained haemodynamically stable, and was transferred to the intensive care unit (ICU). The neurosurgical unit in Sydney was contacted to accept the patient for further neurosurgical and intensive care management after which Careflight International[®] was tasked for transfer. Management at the hospital in Noumea included sedation (fentanyl (5 $\mu\text{g h}^{-1}$) and propofol infusion (12 $\mu\text{g h}^{-1}$)) and seizure prophylaxis (sodium valproate infusion (3 mg h^{-1})) in preparation for transport. The patient had also been started on prophylactic antibiotics because of the rhinorrhoea and documented base of skull fracture at the request of the accepting neurosurgeon.

Upon the arrival of the Careflight retrieval team (approximately 24 h after the injury), the patient remained haemodynamically stable (BP: 110/60 mmHg and pulse rate 90–110 bpm) and was on pressure support spontaneous ventilation and maintenance intravenous fluids. She was not paralysed

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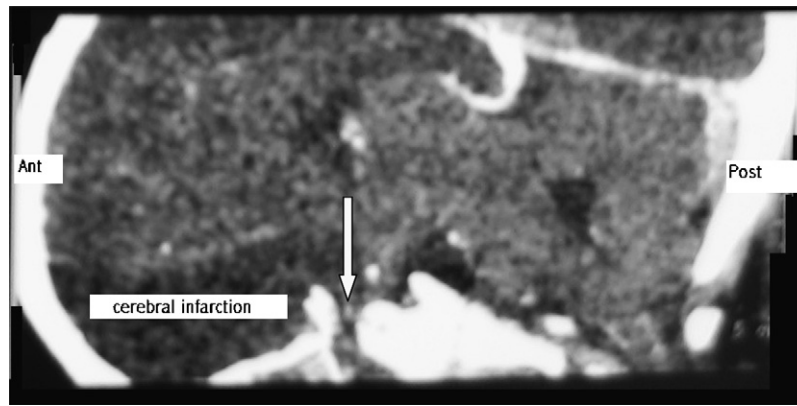


Figure 1 Sagittal section on CT scanning demonstrating fracture through anterior base of skull (arrow), involving the sella turcica. The section of infarction in the anterior cerebrum is also shown (ant. = anterior; post. = posterior).

and the GCS score remained at 2/10 t (on sedation) with normal reactive pupils.

Shortly after arrival, it was noted that the right pupil had become fixed and dilated. The left pupil was still reactive to light, but sluggish. A 100 ml of 20% mannitol was started to reduce the intracranial pressure and the patient was then prepared for transfer (3–4 h flight). However, before departure the patient became very hypertensive (260/140) and agitated, but the pulse remained at 90–110 regular. Oxygen saturations on pulse oximetry fell below 95%. The sedation was increased (fentanyl

and propofol) and the patient was paralysed (vecuronium 2 mg bolus) to allow for easier, more controlled ventilation.

However, the patient continued to deteriorate with falling oxygen saturations (<80%), and was transferred from mechanical ventilation to bag-valve ventilation with 100% oxygen. The patient quickly developed neurogenic pulmonary oedema with large amounts of clear fluid being suctioned from the endotracheal (ET) tube. The position of the ET tube was satisfactory having been checked with direct laryngoscopy. A further bolus of 20%

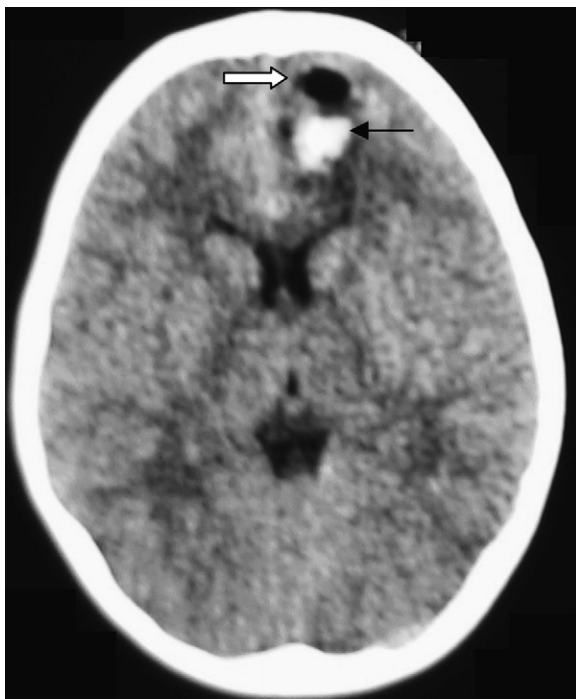


Figure 2 CT of brain showing contusion (black arrow) with pneumocephalus (white arrow) at left frontal lobe.

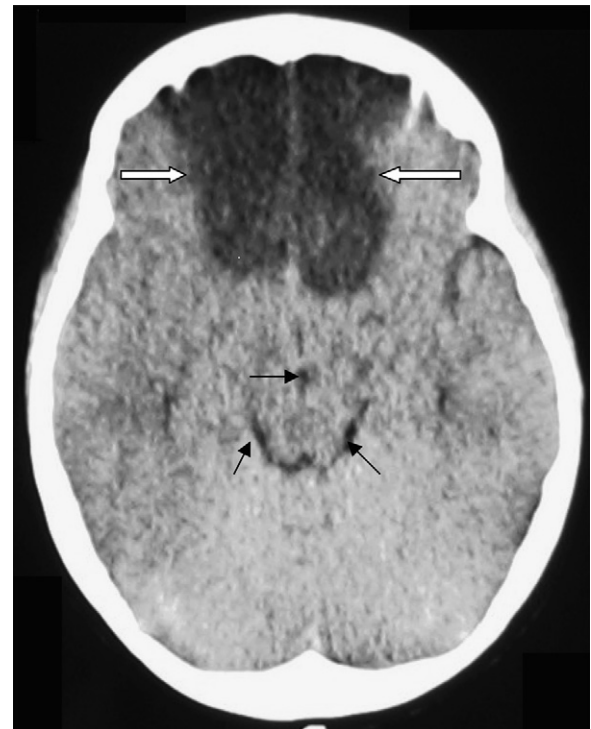


Figure 3 CT of brain showing bilateral frontal infarctions (white block arrows). Basal cisterns and third ventricle is preserved (black arrows).

mannitol (100 ml) and 10 mg of furosemide were administered as emergency treatment of the raised intracranial pressure. Despite these efforts, the patient continued to deteriorate with saturations falling to below 70% and both pupils becoming fixed and dilated. The blood pressure dropped and the patient went into cardiac arrest. Despite advanced cardiac life support (ACLS) (including epinephrine 0.2 mg bolus aliquots to a total of 2 mg) the patient developed asystole and further ACLS was deemed futile. Resuscitation attempts were stopped after 20 min of ACLS and the patient was pronounced dead.

Discussion

Basal skull fractures complicate approximately 24% of blunt head injuries but are unusual after penetrating trauma⁴ with the exception of high velocity injuries (e.g. gun shot wound).¹³ Most commonly, the fracture will occur through the floor of the anterior cranial fossa but involvement of the posterior cranial fossa through the temporal bone may also occur.²¹ Signs of a base of skull (BOS) fracture include otorrhoea, bruising of the mastoid bone (Battle's sign), rhinorrhoea and periorbital bruising (raccoon eyes). Less commonly described are trauma to the cavernous sinus and cranial nerves (especially IX, X, XI, XII).^{3,15,19,30}

The management of the BOS fractures is usually conservative (e.g. elevation of the head of the bed, lumbar drainage, etc.),^{4,31} although direct repair of the dura has been advocated with prolonged CSF leakage, evidence of ascending infection and to decompress the cranial nerves if involved.^{4,8,21,26,31} Other relative indications for operative management of BOS fractures include depressed skull fractures and extensive cranio-facial injuries.³¹ The use of prophylactic antibiotics is probably of most value after penetrating injury.^{5,11}

Direct intracranial vascular trauma is a rare manifestation of injury although involvement of the Circle of Willis should be considered in association with fractures through the clivus or when internal carotid artery dissection is suspected.^{14,20,22,25,29} Fractures through the sella turcica may also involve its resident pituitary gland as well as potential optic nerve damage at the level of the optic chiasm. Usually greater forces are involved for this type of damage to occur and therefore more extensive intracranial vascular involvement should be considered.^{7,16}

Blunt head trauma with vascular involvement is a rare injury, with the internal carotid artery within the foramen lacerum being the most com-

monly injured vessel giving rise to a high carotid artery injury.^{6,12} Surgical management of these injuries is difficult, but there have been reports of successful vein bypass grafts being used.²⁸ Involvement of the other more complex intracranial arteries has been even less commonly described,²⁵ with most reports of acute cerebral artery occlusion leading to cerebral infarction being due to spontaneous dissections of the arteries,^{17,18,24} whose management may be very different from traumatic arterial occlusion.³² Only one case of a dissecting aneurysm of the anterior cerebral artery after blunt trauma has been reported.² Other reports of occlusion after trauma have been described either after penetrating injury^{10,23,27} or as a late manifestation after the development of an arterial aneurysm.^{1,9}

In the case we describe, the patient had an acute anterior cerebral artery injury after blunt head injury. Low velocity mechanisms (i.e. 2 m fall), leading to a depressed fracture with compression of the anterior cerebral vessels has rarely been reported, and thus information on the best course of management is lacking. Presently, it is not clear if a BOS fracture in association with intracranial vascular injury is an indication for operative exploration and repair with the exception of internal carotid artery involvement.²⁸ Experience with the management of BOS fractures with cerebral artery involvement is poor, although the management has tended to be non-operative.^{1,14,25} Bilateral occlusion of the anterior cerebral arteries with cerebral infarction is a very rare injury. In the case we describe the mechanism of infarction is likely to be due to external compression on the anterior cerebral arteries from the depressed skull fragment. Unfortunately a post-mortem was not carried out on this patient and so the precise site of vascular compression is not known.

The prognosis for recovery from this type of neurological injury is poor. Our patient arrested after approximately 24 h post-injury, from what can be presumed to be raised intracranial pressure and medullary coning. It is unlikely that the prognosis could be altered even after exploration and repair of the injuries as the potential for recovery of the infarcted tissue is poor.

In conclusion, although this is a rare injury pattern from low velocity the possibility of vascular compromise should be considered with depressed skull fractures at the base of the skull. Expert neurosurgical opinion should be sought regarding further management of such injuries including whether a hopeful outcome can be offered, before a prolonged transfer takes place.

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