CASE REPORT

Delayed presentation of carotid artery dissection following major orthopaedic trauma resulting in dense hemiparesis

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We report a 30-year-old patient who was involved in a high-velocity road traffic accident and developed a left-sided hemiparesis, which was noted in the post-operative period following bilateral femoral intramedullary nailing. CT scanning of the brain revealed infarcts in the right frontal and parietal lobes in the distribution of the right middle cerebral artery. CT angiography showed occlusion of the right internal carotid artery consistent with internal carotid artery dissection. He was anticoagulated and nine months later was able to walk independently.

An awareness of this injury is needed to diagnose blunt trauma to the internal carotid artery. Even in the absence of obvious neck trauma, carotid artery dissection should be suspected in patients with a neurological deficit in the peri-operative period.

Carotid artery dissection following blunt injury is a significant cause of morbidity in all age groups. It can occur spontaneously or following major trauma, but a high level of awareness is required to make this difficult diagnosis. Only 0.08% to 0.67% of patients admitted to hospital after a road traffic accident have blunt carotid injury; however, the signs and symptoms of carotid artery dissection may be delayed for up to six days after injury. We report a case of carotid artery dissection in a previously healthy man who was involved in a high-velocity motor vehicle accident and developed a dense left-sided hemiparesis.

Case report

A 30-year-old right-handed foreign national, who did not speak English, was involved in a high-impact road traffic accident. He was the restrained driver of a car in a head-on collision with another car, requiring extraction from the scene by the emergency services. There was no loss of consciousness and he was assessed and resuscitated according to advanced trauma life support protocols. His injuries included right-sided rib fractures with underlying lung contusion, a left-sided pneumothorax requiring insertion of a thoracostomy tube, an open right femoral fracture, a closed right fracture of the femoral neck, and a left diaphyseal femoral fracture. He did not have early CT imaging of his head as there was no neurological deficit. He underwent fixation of both femora within eight hours of injury with left cephalomedullary nailing on the left and retrograde intramedullary nailing on the right (Fig. 2).

Six hours post-operatively it was noted that he had a complete left-sided hemiparesis and left-sided facial weakness. Blood tests revealed anaemia with a haemoglobin concentration 8.7 g/dl, and slight hyponatraemia with a sodium 131 mmol/l. Electrocardiography showed a normal sinus rhythm with no post-operative changes, and arterial blood gases were normal. A CT scan of the brain showed wedge-shaped infarcts in the right frontal and parietal lobes (Fig. 3) and further scans at five days showed more pronounced, multiple low-density areas in the watershed areas in the right frontal and parietal lobes.

Trans tho racic echocardiography showed an atrial septal defect with normal left and right ventricular size and function. A paradoxical fat embolus was postulated at this stage, and transthoracic echocardiography revealed a large patent foramen ovale with evidence of a left-to-right shunt but no right-to-left shunt, thus making a paradoxical embolus unlikely.

Subsequent CT angiography of the vasculature neck showed occlusion of the right internal carotid artery 1.5 cm from its origin (Fig. 4). The left internal carotid and both common carotid arteries were normal. Based on these findings, the patient was diagnosed as having carotid artery dissection. He was
treated with warfarin (dosage adjusted to maintain an international normalised ratio between 2.0 and 3.0), but three months later there was no evidence of improvement of the left hemiparesis. However, at nine months he had recovered enough to walk independently.

**Discussion**

Carotid artery dissection following a road traffic accident is rare and difficult to diagnose as symptoms can be delayed. It occurs when a small tear forms in the tunica intima, enabling blood to enter the vessel wall, raising the intima and causing stenosis. This in turn leads to a thrombus, which can occlude the vessel with haemodynamic compromise, or produce effects via an embolic mechanism. One proposed mechanism of injury to the internal carotid artery is rapid deceleration, with resultant hyperextension and rotation of the neck which stretches the artery over the upper cervical vertebrae, producing an intimal tear. Another proposed mechanism of injury is direct trauma. The increased use of shoulder-strap seatbelts due to statutory requirement may be producing a changing pattern of injury. Prior to 1980 only 96 cases of blunt trauma to the carotid artery were reported; however, there are now a total of 480 reported cases in the literature, with 242 during the last five years.

The patient who we describe was driving a right-hand-drive car, with the strap passing across his right shoulder, and the injury was to his right internal carotid artery, in keeping with this mechanism of injury.
with this theory. Four previous case reports have shown carotid artery dissection on the same side as the seatbelt, assuming the cars being driven were not imported.\textsuperscript{9-12} Other case reports have been of bilateral carotid artery dissection following a road traffic accident, but the brain ischaemia was only found on the same side as the seatbelt.\textsuperscript{13,14}

This injury can be asymptomatic and may go undiagnosed. It may present with ipsilateral headache, neck pain, transient episodic blindness, ptosis with miosis, neck swelling, a reduction in the sensation of taste, pulsatile tinnitus, and focal weakness.\textsuperscript{15} Physical signs are important, as a detailed history is often difficult following high-velocity trauma. These include focal neurological deficit, hemiparesis, partial Horner’s syndrome, cranial nerve palsy, cervical bruit, haematoma formation in the neck and massive epistaxis.\textsuperscript{9,15} Mortality from blunt carotid injury is between 20% and 40%, and permanent neurological deficit is seen in 40% to 80% of cases.\textsuperscript{16,17}

Angiography remains the standard for identification of carotid injuries but carries risks, including bleeding, contrast nephropathy, embolus, and further vascular injury.\textsuperscript{18} Magnetic resonance angiography, duplex ultrasound scanning and CT angiography are other less invasive techniques may be used.\textsuperscript{19,21} The advantage of CT angiography as used in this case is that it is readily available and is not operator dependent.\textsuperscript{22}

There is a wide differential diagnosis for patients presenting with stroke following acute trauma (Table I). Emboli originating in the heart should be considered, and paradoxical embolism is possible if the patient has a congenital heart defect.\textsuperscript{23} Thromboemboli may develop in the

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**Table I. Differential diagnosis for delayed-onset hemiparesis following trauma**

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<th>Differential diagnosis</th>
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<td>Intracranial lesion</td>
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<td>Extradural haematoma</td>
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<td>Subdural haematoma</td>
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<td>Subarachnoid haemorrhage</td>
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<td>Brain contusion</td>
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<td>Embolus</td>
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post-operative period, and fat emboli may be produced as a result of long bone fractures and intramedullary nailing. Diagnosis in this case could have been delayed owing to the difficulty of assessing a multiply injured patient who spoke no English.

This case highlights the serious consequence of carotid artery dissection following a high-velocity vehicular accident.

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References