

Life-threatening carotid haemorrhage following blunt trauma

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Abstract

Introduction: We report a case of internal carotid arterial damage following blunt neck trauma. This rare mechanism of injury demands a high index of suspicion to enable prompt specialist management.

Case report: A 22-year-old man presented to hospital after sustaining blunt neck trauma. Rapid onset of stridor necessitated an emergency tracheostomy. Computed tomography angiography demonstrated a tear of the right internal carotid artery, which was repaired surgically.

Discussion: Blunt carotid vessel injury, although rare, has a high mortality rate. Mechanisms of injury include hyperextension and contralateral neck rotation, a direct blow to the vessel, and laceration by adjacent bony structures. The 'gold standard' investigation for suspected blunt carotid vessel injury is catheter angiography, although this carries a small risk of stroke. Computed tomography angiography is a less invasive, alternative investigation which has almost equivalent accuracy. The extent of damage to the vessel wall will dictate treatment. In our literature review, we discuss the presentation, investigation and different treatment modalities available.

Conclusion: This case highlights an unusual mechanism of carotid artery injury, with a delayed, potentially fatal presentation. Such injury demands a high index of suspicion, and confirmation with specific investigations. Management is hazardous and requires experienced personnel in all aspects of care.

Key words: Internal Carotid Artery; Neck; Trauma; Angiography

Introduction

We report a case of internal carotid artery damage following blunt neck trauma. This rare mechanism of injury demands a high index of suspicion to enable prompt management and to avoid significant morbidity and mortality.^{1,2}

Case report

A 22-year-old man walked into the emergency department having sustained a laceration to his left pinna. He had been allegedly punched on the side of his head and neck 12 hours earlier. Initially, there were no signs of respiratory distress, but within minutes he became stridorous and comatose. Severe pharyngeal and laryngeal swelling precluded endotracheal intubation and the patient therefore underwent an emergency tracheostomy via a midline vertical incision to secure his airway. His coagulation screen was normal. A chest X-ray demonstrated a large left pneumothorax. A chest drain was inserted and he was nursed in intensive care.

In the subsequent 24 hours, the patient's neck became increasingly swollen. A contrast-enhanced computed tomography (CT) scan revealed rapid contrast extravasation from the proximal right internal carotid artery (ICA), indicative of rupture of the vessel wall. No bony injury to the cervical spine was visible on the CT scan; thus, a sharp bony fragment could not have caused the tear (Figure 1).

The local vascular surgical team performed open neck exploration. A 5-mm tear was found on the posteromedial aspect of the ICA, 1 cm beyond the bifurcation of the common carotid artery. In view of its location, there was no possibility of inadvertent damage to the vessel at the time of tracheostomy. The torn segment was resected and a saphenous vein graft was sutured end-to-side to bridge the gap between the common carotid artery and the right ICA.

Ten days after the initial assault, the patient left the hospital fit and well, after successful decannulation of the tracheostomy.

Discussion

Blunt carotid vessel injury accounts for only 3 to 10 per cent of carotid arterial injury, and less than 1 per cent of all blunt trauma. Although rare, it has high rates of morbidity (25–58 per cent stroke rate) and mortality (31–59 per cent).³ Poor prognostic factors include stroke and coma at presentation.

Blunt carotid vessel injury is thought to occur by three basic mechanisms: (1) extreme hyperextension and contralateral rotation of the neck, placing the vessels in proximity to the lateral articular processes of C1–C3 vertebrae near the skull base; (2) a direct blow to the vessel; and (3) laceration by adjacent bone fractures.⁴

A combination of extreme hyperextension and a direct blow probably accounted for our case.

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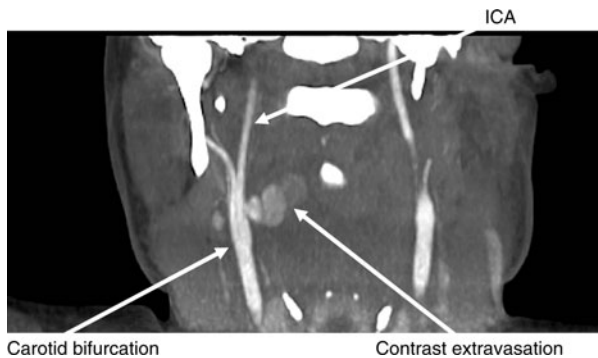


FIG. 1

Coronal reformat of contrast-enhanced computed tomography neck scan, demonstrating rupture of right internal carotid artery (ICA).

Motor vehicle accidents account for the majority of cases (41–70 per cent), with a direct cervical blow a less common cause (10–20 per cent).⁴

Blunt carotid vessel injury usually occurs in association with other serious injuries (e.g. facial and head injury, cervical spine fracture, or thoracic injury).³ Therefore, compounded by the fact that presentation may be delayed, blunt carotid vessel injury is often missed, or indeed not searched for, in the acute setting.⁵ Combined head and chest injury is associated with a 14-fold increase in incidence.

Carotid vessel injury may also occur in a seemingly innocuous way. There are various case reports in the literature outlining such causes, including: chiropractic manipulation, rapid head turning, baseball injury, boxing, violent coughing, sneezing and neck extension during intubation.⁶ There may be an underlying predisposing condition such as a collagen vascular disorder.

Blunt carotid vessel injury may cause a spectrum of damage. Injury may lead to intimal disruption and subsequent thrombosis, media disruption and pseudoaneurysm formation, dissection, or damage to all three vessel layers with vascular leakage. Transection is nearly always fatal. Our patient was very fortunate to be in hospital when the rupture of his right internal carotid artery (ICA) became symptomatic (i.e. when stridor developed).

Anatomically, the majority of injuries occur in the distal ICA, in the region of the skull base, or within the artery's course in the bony skull.

Clinical presentation may be delayed from one hour to several weeks.⁵ Patients may present with: respiratory obstruction; a diffuse or discrete, pulsatile mass (i.e. pseudoaneurysm); transient ischaemic events; or stroke. An ipsilateral Horner's syndrome, due to sympathetic nerve compression, is suggestive of arterial damage.¹

The recent use of screening protocols (e.g. Memphis and Denver) based on symptoms, signs and associated injuries has enhanced early detection prior to subsequent neurological sequelae. However, the use of such protocols in the UK is not universal.

As most patients are asymptomatic at the time of presentation, there is a narrow window of opportunity to diagnose and commence treatment. Indeed, prior to the advent of screening protocols, Berne *et al.* reported a median time to diagnosis of 12.5 hours for survivors of blunt carotid vessel injury, versus 19.5 hours for non-survivors.⁴

The 'gold standard' investigation for suspected blunt carotid vessel injury is catheter angiography. It has the added advantage that it may be combined with an

interventional procedure. However, the procedure carries a 0.5–2 per cent risk of stroke.

Contrast-enhanced magnetic resonance angiography is a sensitive technique, but it is time-consuming and thus hazardous in the acute setting. In addition, the monitoring of sick patients undergoing magnetic resonance imaging requires specialist non-ferromagnetic equipment, which is not always available. Helical CT angiography is quick, non-invasive and causes minimal deviation from imaging protocols in the trauma setting, which often include CT of the brain, neck and chest.

The primary treatment for intimal tears and stable dissections is anticoagulation therapy, although there is little evidence to support this. Pseudoaneurysms and ongoing bleeding require either endovascular or open repair. The position of the injury, together with the available expertise, determines the nature of the intervention. Distal, inaccessible pseudoaneurysms are better treated with an endoluminal stent graft, whereas proximal ones may be more amenable to open repair.⁷ Stent grafts may also be a relatively less hazardous way of controlling a substantial acute haemorrhage. The long term patency of stent grafts has yet to be demonstrated, and all require antiplatelet agents to be administered long term.⁴ It was therefore felt that, in view of the relatively young age of our patient, direct surgical repair was the best long term option.

- **Blunt carotid vessel injury is rare but has a high mortality rate**
- **Mechanisms of injury include hyperextension and contralateral neck rotation, a direct blow to the vessel, and laceration by adjacent bony structures**
- **Computed tomography angiography can be used to investigate blunt carotid vessel injury**
- **The extent of damage to the vessel wall will dictate treatment**

Open surgical options will again depend on the nature of the injury. They include thrombectomy, intimal flap repair, false lumen repair and interpositional graft placement (as in our case). In rare cases of extensive, uncontrollable distal injuries, carotid artery ligation is performed, with its inherent risk of a neuro-ischaemic event.

Conclusion

This case highlights an unusual mechanism of carotid arterial injury, with a delayed, potentially fatal presentation. This injury demands a high index of suspicion and confirmation with specific investigations. The management is hazardous and requires specialist multidisciplinary care.

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