Delayed bilateral internal carotid artery thrombosis following accidental strangulation

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A 24-yr-old male presented after a fishing accident in which he was pulled underwater by a rope attached to a crayfish pot. He was winched out of the water with the rope still around his neck, sustaining serious neck injuries that ultimately led to his death. After initial resuscitation, he remained fully conscious for approximately 8 h, after which there was a rapid and sudden deterioration in his level of consciousness. The presentation, investigation, management and subsequent postmortem findings are presented and discussed.

Br J Anaesth 2000; 84: 521–4

Keywords: arteries, internal carotid; complications, trauma

Accepted for publication: November 5, 1999

Bifurcation of the common carotid arteries in the neck leads to the formation of the internal and external carotid vessels. The two internal carotid arteries, along with the vertebral arteries, are responsible for the blood supply to the brain, uniting on its inferior surface to form the circulus arteriosus. During their short course in the neck, the internal carotid arteries are thus vulnerable to both penetrating and blunt trauma.

This report illustrates an unusual form of blunt trauma to the neck leading to the rare occurrence of bilateral internal carotid artery occlusion and delayed onset of neurological signs.

Case report

A previously fit 24-yr-old male was transferred to our emergency department by an air ambulance from a peripheral hospital. Eight hours earlier, he had been involved in an accident on a fishing boat in which he was dragged overboard and underwater by a rope around his neck attached to a heavy crayfish pot (weighing approximately 100 kg). He was submerged for more than 60 s at a depth of around 40 feet. His colleagues hoisted him out of the sea and onto the deck using the same rope. On surfacing he was found to be apnoeic, cyanosed and unresponsive. He was given expired air resuscitation for about 10 min by the crew, which led to resumption of spontaneous respiration. A peripheral pulse was present throughout the resuscitation.

On assessment at the peripheral hospital (3 h later), he was fully conscious and orientated, with a core temperature of 35°C. He had vomited once during the journey. Examination revealed bilateral conjunctival haemorrhages; reactive pupils with 1-mm inequality; engorged and tender ears; a swollen neck with a horizontal abrasion marking the position of the rope (at the level of the thyroid cartilage); and mild left arm weakness. Initial haematological investigations were unremarkable, and chest and cervical spine radiographs revealed no abnormalities.

In view of the limited medical resources at the peripheral hospital, he was transferred by air ambulance to a larger facility. Throughout the journey he remained fully conscious and haemodynamically stable. He arrived approximately 8 h after the accident.

In the emergency department, he was noted to have an initial Glasgow Coma Score of 15 but within 20 min he lost consciousness. The trachea was rapidly intubated. At laryngoscopy, the larynx was found to be deviated to the left and the oesophageal opening to the right. Nevertheless, the trachea was intubated with ease.

In view of the sudden deterioration, associated neck injury and history of submersion, urgent CT imaging of the head and neck and carotid angiography were performed. At this stage, the patient remained haemodynamically stable, and was sedated, paralysed and artificially ventilated to normocarbia.

CT imaging revealed gross cerebral oedema and soft tissue oedema of the neck. Carotid angiography revealed bilateral occlusion of the internal carotid arteries (Fig. 1A and B). Urgent bilateral carotid revascularization was therefore performed using saphenous vein grafts. Carotid arterial shunts were used to bypass the occlusion. Monitoring
Fig 1 Carotid angiograms illustrating (A) left and (B) right internal carotid artery thrombosis.

of the cerebral circulation during the operation (e.g. by transcranial Doppler) was not available. The patient was admitted to the intensive care unit after the operation. Check angiography during the operation revealed satisfactory blood flow in the right internal carotid and good flow on the left. There were no problems during the operation apart from hypertension (mean arterial pressure more than 110 mm Hg), which was not treated by the anaesthetist in order to maintain cerebral blood flow. Mannitol and artificial ventilation to normocarbia were used in view of the known cerebral oedema.

In the intensive care unit, a parenchymal intracranial pressure monitor was sited. Initial intracranial pressure was grossly elevated (up to 70 mm Hg) and refractory to maximal osmotherapy, intermittent hyperventilation for exacerbations of intracranial hypertension, and sedation and analgesia with large doses of midazolam and morphine. Cerebral perfusion pressure was maintained at >75 mm Hg with inotropic support using norepinephrine and epinephrine. Focal neurological signs were also present, with an unreactive left pupil. A thiopental loading dose followed by infusion was started to treat the uncontrollable intracranial pressure. This remained high, however, peaking at 80 mm Hg over the next 12 h and requiring numerous bolus doses of thiopental, regular mannitol and hypertonic saline in order to keep it below 40 mm Hg.

Over the course of the next 72 h there was no improvement. The pupils became bilaterally fixed and dilated and brainstem reflexes were absent on clinical examination. Technetium scintigraphy and contrast angiography of the cerebral circulation were performed on two occasions 24 h apart in an attempt to ascertain whether perfusion to the brain was present, as the clinical diagnosis of brain death was made impossible by the thiopental infusion. Both investigations revealed some posterior circulation perfusion, but no supratentorial flow, suggesting re-thrombosis. In view of these findings, continuing support was futile and, after discussion with the family, treatment was withdrawn.

A post-mortem investigation concluded that the cause of death was consistent with cerebral ischaemia following vascular neck injury secondary to neck compression.

Discussion
This case presented an unusual combination of potential problems resulting from accidental near-drowning and near-hanging. The near-drowning aspect of the presentation did not appear to contribute significantly to the outcome, as no
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Water was aspirated from the lungs and at no time was lung function impaired. Although expired air resuscitation was performed initially, the only other finding that could be attributed to the submersion was mild hypothermia. One may argue that the elevated intracranial pressure and cerebral oedema could have been attributable to cerebral hypoxaemia resulting from the initial submersion, but the patient was fully conscious and orientated for a significant length of time after the incident and the submersion time was short.

Another mechanism of injury may explain the subsequent course. The presence of the rope around the neck and the subsequent survival allow us to refer to the injury as a near-hanging. The mechanism of the injury is different from most near-hangings, as there would have been no initial sudden axial traction from suspension because of the presence of the surrounding water to support the weight of the body.

In this case, the injuries were more likely sustained after attempts at removal from the water by his colleagues. As they winched him back up to the surface, there would have been a period between exiting the water and reaching the deck when he would have been suspended by his neck, the forces of the winch and the pursuig craypot acting in opposite directions and tightening the rope. This would lead to continual external pressure on his neck, and thus a form of strangulation.

The contents of the anterior neck region are uniquely vulnerable to external pressure because of the number of vital structures in a relatively confined and unprotected space. Posteriorly, the spinal cord is at its most vulnerable, the cervical spine being more mobile but less stable than the thoracic and lumbar regions of the vertebral column.

Structures at risk in hanging include the vasculature of the neck, the carotid bodies, the larynx and its associated cartilages, and the cervical spine. Secondary injury to the brain is the main cause of early death. Pulmonary complications are responsible for delayed mortality, often as a result of the acute respiratory distress syndrome, the exact pathogenesis of which is unclear in this setting, although a centrenurogenic mechanism has been proposed.

No bony or cartilaginous damage was sustained in our patient. Cervical spine injuries are rare in non-judicial hanging and depend on a significant drop length, at least the height of the patient (which was not relevant in this case).

The obvious clinical finding was gross swelling of the neck, which initially raised concerns of airway obstruction, although this was resolved on intubation. Venous obstruction and subsequent stagnant cerebral hypoxia has been proposed as the initial event in most hangings, but arterial obstruction is likely to play a more important role, particularly as loss of consciousness has been reported to occur as early as 15 s after suspension. The force required to obstruct the carotids is only slightly greater than that for the jugular veins: 3.5 kg compared with 2 kg, both of these values would easily be exceeded with only partial suspension of body weight.

Suspicion of vascular damage as a cause of the gross swelling of the neck led to visualization of the arterial vessels using angiography. This demonstrated bilateral internal carotid artery occlusion (Fig. 1A and B). Traction on arterial structures can lead to intimal damage and subsequent thrombosis and obstruction. This was the most likely cause in this case, although it is a rare finding; an autopsy series of 101 hangings failed to demonstrate any intimal tears.

Traumatic thrombosis of the internal carotid artery is reported as being caused by one of four mechanisms: injury to the intrapetrous or cavernous part of the carotid artery during basal skull fracture; injury to the point of emergence of the carotid artery from the cavernous sinus as the result of shearing strains; a direct blow to the neck or trauma to the paratonsillar area by a foreign object carried in the mouth; and stretching of the carotid artery by hyperextension and lateral flexion of the neck (which probably occurred in this case). Secondary symptoms resulting from thrombosis of the carotid artery develop most commonly between 12 and 24 h after the injury. The prognosis in these patients is mostly poor.

By reviewing the history, the natural progression of the pathology can be elaborated upon. After initial resuscitation, the patient remained conscious for about 8 h. During this time, intimal damage of the carotid must have been present and the thrombotic process would have been developing. The time interval before onset of unconsciousness is typical of cases of blunt carotid trauma, as dissection of the intimal layer and progressive thrombosis can have a latency period of up to 24 h. Deterioration after this period was likely to be due to the gross increase in intracranial pressure and the concomitant decrease in cerebral perfusion pressure, leading to loss of consciousness as a result of cerebral anoxia. Unfortunately, during the inevitable delay involved in obtaining the essential diagnostic imaging studies and corrective surgery, the intractable cerebral oedema meant that significant brain damage had already ensued before the patient was admitted to the intensive care unit, rendering futile subsequent attempts at controlling intracranial pressure.

It has been suggested that, after initial resuscitation, appropriate airway control and neurological examination, CT scanning of the head and neck and repeated colour-flow Doppler sonography are indicated in all patients with neck trauma of this nature. If Doppler sonography reveals an intimal lesion, angiography and immediate surgery and reconstruction of the artery have been recommended, even if there is not yet a neurological deficit. Others recommend angiography in all patients with neurological symptoms.

Thrombolysis has been used with success in cases of non-traumatic carotid thrombotic occlusion (stroke) and has been suggested as an alternative to surgical correction in traumatic thrombosis. But there are no reports of bilateral
traumatic carotid thrombosis being treated with systemic or arterially directed thrombolysis. In our case, there was fear of the small retinal and cerebral haemorrhages enlarging with thrombolytic therapy, leading to blindness or significant intracranial bleeding and also interfering with definitive surgical repair.

There is an inevitable delay, as surgical intervention is dependent on accurate localization of the abnormality. Cerebral angiography is a time-consuming event in the presence of acute and profound ischaemic brain injury, delaying restitution of cerebral blood flow with inevitable neurological deterioration. Alternatively, computerized tomographic angiography uses the same technique as head and neck scanning and provides images that may be of adequate quality for emergency, life-saving surgery.

The computerized tomographic angiogram of this patient is presented in Figure 2. Our vascular surgeons consider records such as these to be sufficient to allow the patient to be taken directly to theatre without the delay of cerebral angiography. Timely intervention is vital for the best neurological result, and so where this technology is available it may provide a better outcome.

This case illustrates the need for a high index of suspicion of vascular injury in cases of blunt trauma to the neck, especially when the history suggests a significant degree of trauma from partial or full suspension. A period of normal consciousness and the absence of neurological signs is not uncommon in blunt injuries to the carotid vessels, and thus such cases present a diagnostic challenge.

References