

## LESSON OF THE MONTH

## Delayed symptoms and death after minor head trauma with occult vertebral artery injury

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### Abstract

**Head injury without loss of consciousness is seldom accompanied by grave complications. We report the case of an 18 year old cyclist who was struck by a car in a minor road traffic accident, suffered minor head injury without loss of consciousness, and died unexpectedly seven weeks later with vomiting and coma. Necropsy revealed an expanding cerebellar infarct and vertebral artery thrombosis, superimposed on an old dissecting intramural haematoma of the right vertebral artery in the atlantoaxial region. Vertebrobasilar occlusion after minor head trauma, hyperextending or rotating neck injury, or neck manipulation is commonest in young people. Occult ligamentous injury to the cervical spine after trauma may be a contributing factor to the pathogenesis of vertebral artery damage after injury to the neck.**

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Head injury does not usually cause death unless there are indicators of severe head trauma, such as depression or loss of consciousness. The torsion or hyperextension of the neck accompanying even minor head injury, however, may cause damage to the vertebral artery, as indicated by the following case.

### Case report

An athletic 18 year old was involved in a motor vehicle accident on 2 February 1991. While riding his bicycle, he was struck by a car. He was thrown on to the bonnet of the vehicle and rolled up to the windscreen. There was no loss of consciousness. After assessment at an emergency room 20 minutes later, he was discharged with a diagnosis of fracture of the tibia, below the tibial plateau. No head trauma could be recalled from the accident, but in view of a minor laceration on his head, he and his mother were given a head injury protocol to take home. There was no depression of consciousness over the next 24 hours.

The boy continued to be heavily involved in body building, cycling, and athletics. Three days after the accident he began to complain of headaches to his school friends. Episodes of

transient global amnesia occurred, during which he would suddenly forget where he was, and what he was doing. These memory lapses were never accompanied by collapse or weakness. He complained of a persistent sore neck.

On 28 March 1991, he was difficult to arouse, and awoke with a frontal headache. He needed help dressing; his mother describing this as "dressing a baby". He stayed away from school, slept most of the day, and vomited increasingly. By early evening, alteration in affect was noted. He was taken to hospital in a drowsy but rousable state. Over the next hour he became progressively rigid and cyanotic, then unresponsive to verbal stimuli or urinary catheter insertion. Brain stem reflexes disappeared. Cardiac arrhythmias appeared, consisting of supraventricular tachycardia and ventricular bigeminy. A CT scan revealed a density in the cerebellum and secondary hydrocephalus. He was apnoeic to a PCO<sub>2</sub> of 62 mmHg after three minutes and was declared brain dead the following morning, 29 March 1991.

### NECROPSY

Necropsy revealed a recent haemorrhagic infarct in the right cerebellar hemisphere, with oedema, tonsillar herniation, and medullary compression. A clot was present in the right vertebral artery. The cerebral hemispheres showed no gross abnormality other than acute dilatation of all ventricles. The neck was extensively dissected. No frank haemorrhages, either recent or old, were seen on visual inspection of the ligaments of the atlas or axis.

Neuropathological examination of the brain showed numerous microscopic infarcts symmetrically distributed in the vertebrobasilar territory of supply, including the occipital lobes, hippocampi, thalamus, and hypothalamus. Their sizes ranged from tens of microns to a few hundred microns in diameter, with cyst formation in the larger infarcts. Vessels showing evidence of old occlusions and residual thrombi were numerous; occlusive fibrointimal hyperplasia and deposition of intraluminal and perivascular iron were seen.

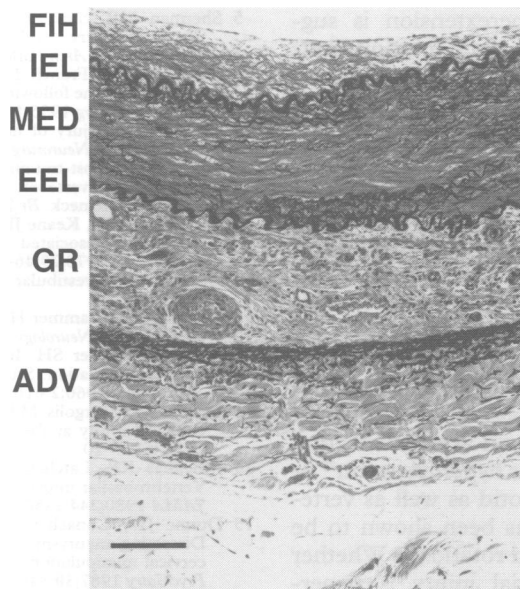
The right vertebral artery was subserially sectioned from its location in the foramen transversarium of C2 to the vertebrobasilar junction. Microscopical examination revealed old intramural dissection in the portion of the vessel in the atlantoaxial region. The artery

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**Figure 1** Magnification of the wall of the injured artery, with the lumen at the top. Progressing outwards concentrically, the photograph shows fibrointimal hyperplasia (FIH), the internal elastic lamina (IEL), the smooth muscle and elastic lamellae of the tunica media (MED), the external elastic lamina (EEL), lighter staining granulation tissue (GR), and the adventitia (ADV). The granulation tissue, in addition to fibroblasts and capillaries, contains iron in macrophages, variably distributed along the circumference and the length of the dissection. Trichrome-elasticin stain; bar = 200  $\mu\text{m}$ .



showed fibrointimal hyperplasia, but stenosis approached maximally 20%–30% of the lumen. The outer third of the tunica media contained fibrovascular granulation tissue, splitting the external elastic lamina from inner concentric laminae and smooth muscle (fig 1).

The dissection tapered in extent proceeding superiorly from the atlantoaxial region. At the origin of the posterior inferior cerebellar artery, however, the vessel was encased in dense fibrous tissue, enclosing the nervi vasorum. Sections through this region revealed subperineurial haemorrhage in the nervi vasorum (fig 2) of the vertebral artery. At this level acute neutrophilic inflammation was also seen within the tunica media and the areas of fibrointimal hyperplasia, extending up to defects in the internal elastic lamina and endothelium. Special stains for bacteria or fungi were negative.



**Figure 2** Posterior inferior cerebellar artery near the site of origin and two nervi vasorum. One of the nervi vasorum shows recent hemorrhage (arrow) beneath the perineurium and there is thrombosis of the vasa vasorum (right centre). The intraluminal thrombus has been displaced by postmortem angiography. Trichrome-elasticin stain; bar = 100  $\mu\text{m}$ .

## Discussion

The patient died seven weeks after a minor head injury and showed evidence of both early and delayed injury to the vertebral artery. The delayed death was due to thrombosis of the vertebral artery and cerebellar infarction, and oedema and brainstem compression. Evidence of remote trauma consisted of a healing intramural dissecting haematoma of the tunica media of the right vertebral artery, fibrointimal hyperplasia, and numerous old, resorbing thromboemboli and cystic infarcts in the vertebrobasilar territory. Evidence of recent trauma to the vertebral artery included subperineurial haemorrhage, acute neutrophilic inflammation in the tunica media near the origin of the posterior inferior cerebellar artery, and recent thrombosis of the vertebral artery itself and its vasa vasorum.

The notion of occult ligamentous injury to the neck is supported by the boy's complaints of neck pain, and by the finding in a large series that such occult trauma to the cervical spine is more common than is generally appreciated in traffic accident victims.<sup>1</sup> A series of 12 patients with either unilateral or bilateral facet joint dislocations after motor vehicle accidents revealed vertebral artery occlusions in nine patients.<sup>2</sup> The average age was 38.8 years and no patient in the series was older than 50. Even without occult ligamentous trauma the young neck, with its greater mobility, is prone to vertebral artery injury when manipulated, indicated by the young age of most chiropractic and exercise induced injuries to the vertebral artery.<sup>3–5</sup> It was verified that the boy did not visit a chiropractor after his injury despite the complaints of a sore neck. The final mechanism of death in injury to the vertebral artery is often oedematous cerebellar infarction with brainstem compression.<sup>6–8</sup>

At the microscopic level, the mechanism of the delayed thrombosis of the vertebral artery in the present case was likely endothelial injury superimposed on a severely injured vertebral artery with extensive fibrointimal hyperplasia and old medial dissection. The added complication of lax, injured ligaments in the neck may have played an additional part. An injured vertebral artery with a circumferential, healing dissection is likely to be prone to damage by even normal neck rotation and extension movements, in addition to the athletic movements made by this young man during the course of pursuing his varied sports. Case 1 of Devereaux *et al*<sup>9</sup> showed an old vertebral artery dissection eight years after a road traffic accident, with death due to recent cerebellar and brainstem infarction.

At the gross level, the mechanism of injury to the vertebral artery in the present case may involve compression of the artery due to either lateral rotation or hyperextension. Lateral rotation of the head has been shown to cause vertebral artery obstruction,<sup>10–13</sup> with subsequent dissection<sup>14–17</sup> of the artery and occasionally pseudoaneurysm formation.<sup>18,19</sup> Hyperextension may also lead to dissection of the vertebral artery.<sup>20–22</sup> The mechanism of

rotation rather than hyperextension is suggested in the present case, neck rotation likely occurring as the boy rolled over the bonnet of the car up on to the windscreen.

Local anatomical features unique to the atlantoaxial region<sup>3</sup> are often invoked to explain vertebral artery injury after head or neck trauma involving neck hyperextension or rotation. It is true that the local anatomy of the sharply turning vertebral arteries emerging from the foramina transversaria of the cervical vertebrae makes them vulnerable to occlusion with head rotation, as has been demonstrated at necropsy<sup>10-12</sup> and angiographically.<sup>13</sup> This cannot explain all cases of such damage, however, as carotid artery dissection may also occur after either hyperextension<sup>23</sup> or rotation.<sup>24</sup> Moreover, carotid as well as vertebral artery blood flow has been shown to be sharply reduced with head rotation.<sup>12</sup> Whether the mechanism of arterial injury is hyperextension or lateral rotation, the present case illustrates the vulnerability of the young neck and especially the vertebral arteries to injury, even in cases of minor head trauma. Death may occur a considerable time after the injury due to occult vertebral artery damage in the form of a dissecting haematoma and its consequences.

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