

BLUNT TRAUMA TO THE CAROTID ARTERY

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Non-penetrating trauma to the internal carotid artery presenting as an immediate or delayed neurological deficit is an uncommon clinical entity. It has a high reported morbidity and mortality. Three cases are presented with long-term clinical and radiological follow-up. The mechanisms of injury, clinical features and possible treatment modalities are discussed.

Key words: angiography, internal carotid artery, non-penetrating trauma.

Introduction

Blunt trauma to the cervical internal carotid artery may lead to stenosis, dissection, thrombosis, embolism, aneurysm formation or rarely to arterial rupture.¹ Motor vehicle accidents are the most common sources of these injuries, but cases secondary to relatively minor trauma have been reported.² The onset of neurological symptoms may be immediate, or more commonly delayed by several hours or even days or weeks.^{1,3} Many cases probably remain subclinical. Blunt carotid trauma may mimic direct cerebral injury or intracranial bleeding, and is an important cause of stroke in the young.² Computer tomography (CT) scanning may be unhelpful in the early clinical course and angiography is required to make the definite diagnosis.

Case reports

CASE 1

A 44 year old left-handed woman was the driver of a car involved in an accident on 10 December 1980. There was no loss of consciousness. She sustained a fracture of the mandible, as well as a fractured right maxilla. The first and second ribs were fractured with a resultant small pneumothorax. Multiple lower limb fractures were also sustained.

On admission to hospital 2 h later she was noted to have a right hemiparesis, right upper motor neuron facial weakness, a right sensory neglect, and extensor right plantar response. Speech was

unaffected. Her conscious state remained normal. Both carotids were normally palpable as were the superficial temporal arteries.

A CT head scan performed the same day was normal. Two days later a right carotid bruit was noted. On 13 December 1980, an arch aortogram and selective left carotid arteriogram demonstrated two areas of marked irregularity on the posterior wall 3 cm and 5 cm distal to the origin of the internal carotid, with severe stenosis extending over 2-3 cm and sluggish distal flow (Fig. 1).

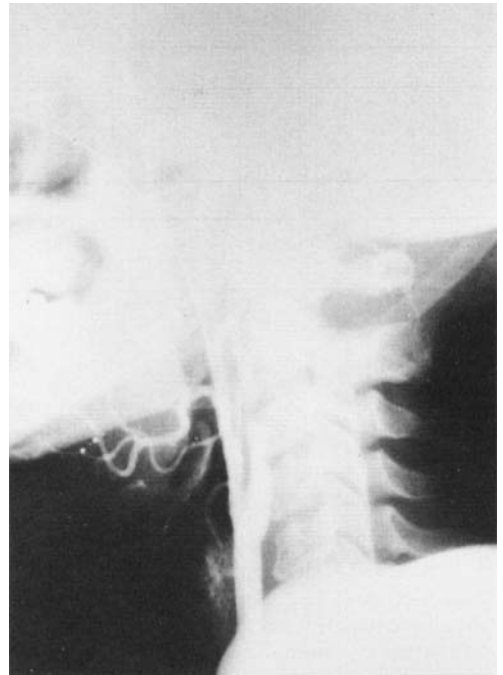


Fig. 1. Left carotid angiogram on admission (case 1).

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The patient was managed without anticoagulants. A repeat angiogram 3 months later showed only minor narrowing at the level of the second and third cervical vertebrae (Fig. 2). No further episodes of neurological deficit occurred, but the patient is left with a permanent handicap of a mild right hemiparesis at 6 years follow-up. She walks independently but has persistent reduced co-ordination of the right hand.

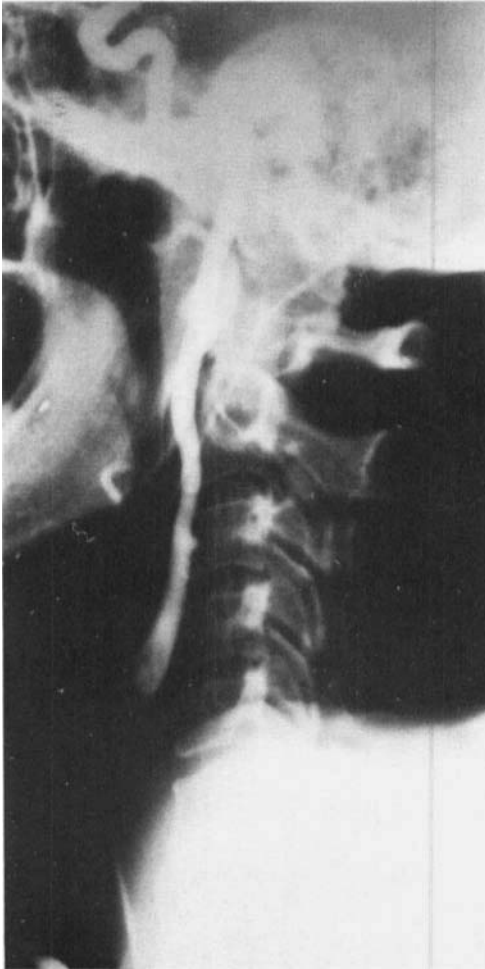


Fig. 2. Left carotid angiogram 3 months following injury (case 1).

CASE 2

A 21 year old male pedestrian was struck by a car on 19 June 1982. He sustained a closed head injury, facial lacerations and bruising, and a fractured pelvis and fibula. He was conscious for 30 min and then 'irritable, confused and drowsy', but was fully conscious, alert and oriented the following day, without focal neurological signs.

Twenty-four hours after the accident he suddenly became drowsy again, irritable and disoriented. There was a left hemiparesis affecting mainly the arm. He was transferred to the Royal Melbourne Hospital where a CT scan showed generalized cerebral oedema only.

The following day it was noted that the right superficial temporal pulse was diminished. Carotid angiography was performed and showed narrowing of the right internal carotid artery opposite the first and second cervical vertebrae, with irregular narrowing extending as far as the carotid siphon (

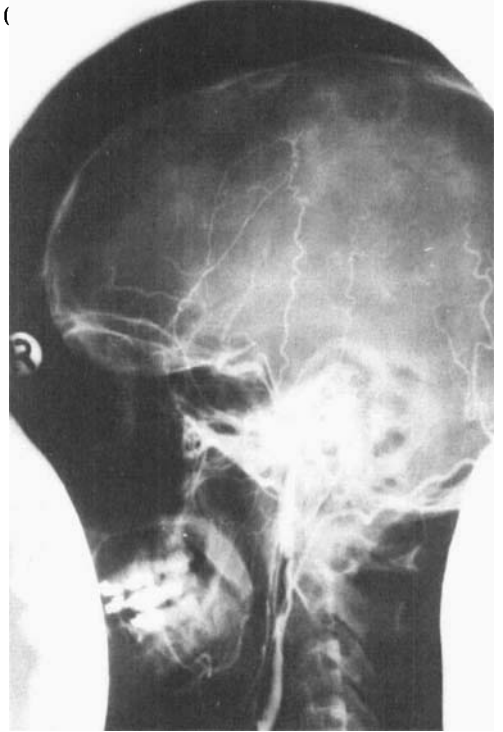


Fig. 3. Right carotid angiogram on admission (case 2).

He was managed without anticoagulants, except for aspirin which was commenced 1 month after injury and discontinued after 3 months.

Repeat angiography 3 months after the injury showed that the narrowing of the right internal carotid was much less and situated proximal to an aneurysm at the base of the skull. The distal artery appeared normal (Fig. 4). His hemiparesis improved steadily with rehabilitation and he made a good neurological recovery. At 4 years follow-up digital subtraction angiography was normal apart from minimal dilatation of the left internal carotid artery (Fig. 5). At this time he had no residual deficit.

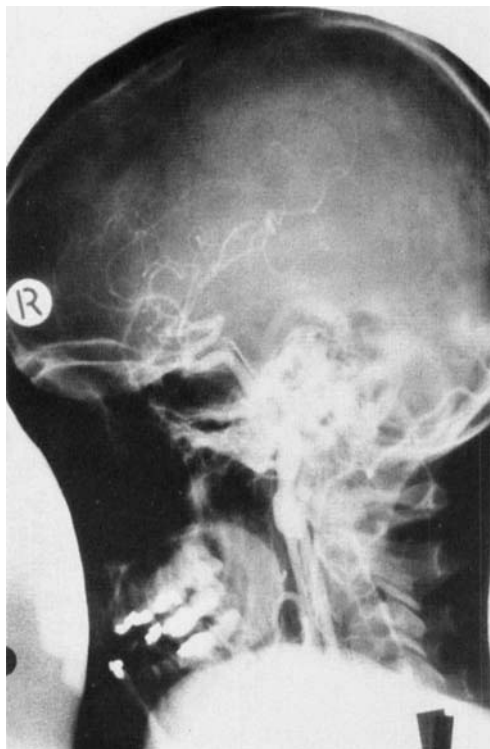


Fig. 4. Right carotid angiogram 3 months following injury (case 2).

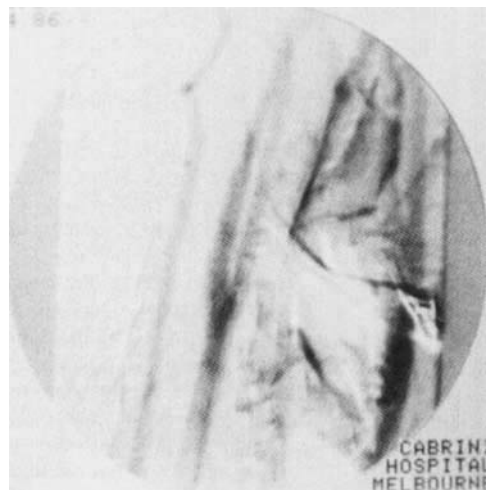


Fig. 5. Right carotid angiogram 4 years following injury (case 2).

CASE 3

A 42 year old woman was involved, as the driver, in a motor vehicle accident on 19 July 1982. She was trapped in the vehicle for 1.5 h. There was no significant head injury, with only brief loss of consciousness and a short period of retrograde and post-traumatic amnesia. She was fully alert and oriented on arrival at hospital. There were facial lacerations, fractured pelvis, shaft and neck of the right femur and also a fractured radius and patella. A bruise was noted over the right side of the neck. There were no focal neurological signs.

Three days after the accident there was a sudden deterioration with the development of bilateral neurological signs consisting of dysphasia, left visual neglect and right sided weakness. Cervical and cranial pulses were strong and symmetrical but a bruit was clearly audible over the right carotid.

An emergency CT head scan showed subtle hypodense areas in the right frontoparietal area. Aortic arch and carotid angiography, performed the following day, showed a grossly irregular and narrowed left internal carotid from a point 3 cm distal to its origin extending up to the base of the skull (Fig. 6). Many branches of the ipsilateral middle

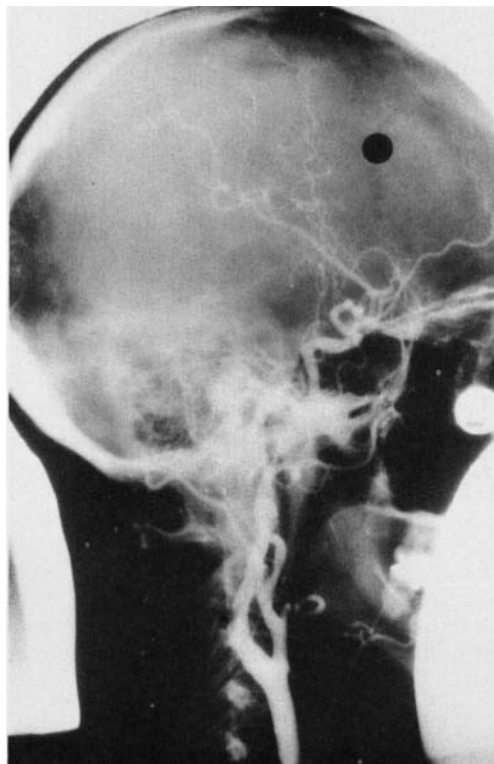


Fig. 6. Left carotid angiogram on admission (case 3).

cerebral artery were occluded with collateral supply from the left anterior cerebral artery. The right internal carotid was irregularly narrowed from a point 4 cm beyond its origin as far as the base of the skull (Fig. 7).



Fig. 7. Right carotid angiogram on admission (case 3).

She was managed with intravenous Rheomacrodex over 4 days and then anticoagulated with warfarin for a period of 4 months. She made slow progress from her multiple injuries but with rehabilitation subsequently made a good recovery. Her most significant functional deficit was micrographia, which, interestingly, progressed over a period of years.

At review in July 1986 she exhibited some residual hesitancy of speech and there was still an increase in tone affecting the right arm and leg. She had returned to tertiary studies. Follow-up digital subtraction angiography in July 1986 (Fig. 8) revealed residual slight irregularity at each carotid bifurcation. No narrowing of either vessel was noted.

Discussion

Several possible mechanisms of injury exist in patients with non-penetrating injuries to the internal



Fig. 8. Digital subtraction angiogram 4 years following injury (case 3).

carotid arteries. Direct trauma, which may occur with a blow to the neck, has been well documented as a cause of this type of injury.^{3,4} Trauma to the mandible with or without fracture may crush the internal carotid against the upper cervical spine, and this may have been a factor in Case 1.

The majority of cases reported in the literature occur in victims of motor vehicle accidents without signs of direct neck trauma or fractures of the mandible. Many display forehead and upper facial soft tissue injuries. Rapid deceleration with flexion of the neck is followed by hyperextension. This stretches the carotid over the anterior surface of the cervical spine, and with relative tethering between the base of the skull and the aortic arch, tearing of the intima, the least elastic layer of the artery, may occur.

Rotation of the head combined with extension has been suggested as a causative factor,⁵ but the presence of bilateral carotid trauma, as in Case 3, and several other reported cases would argue against this being a major factor.^{6,7}

Depression of the shoulder has been proposed as an aetiological factor and the carrying of heavy weights on the shoulder has been cited in one case.² Intra-oral trauma⁸ and fractures through the petrous temporal bone may traumatize the internal carotid, the latter usually being found at post-mortem.

The pathogenesis of these injuries has been well demonstrated in autopsy studies and in cases submitted to surgery.⁴ Intimal tears are often found, and this exposes the underlying media, which are strongly thrombogenic. Intimal tearing may also lead to intramural dissection. Thrombosis and dissection lead to narrowing and in some cases distal

thrombo-embolism. Gross stretching of the artery may disrupt the media and lead to false aneurysm formation, and rarely rupture into the neck or thorax as an immediate or delayed event.¹

The diagnosis of blunt carotid trauma should be suspected in the presence of certain clinical features shared by the majority of these patients. Most are victims of high speed vehicle accidents. There is a characteristic time lag from initial injury to the onset of the neurological deficit, most occurring after 1–24 h,³ and the severity of the deficit is out of proportion to the degree of depression of the conscious state, with many patients remaining relatively alert. This is in contradistinction to those with intracranial clots and lateralizing signs whose conscious states are usually severely depressed. The majority have no signs of direct neck trauma, but a neck bruise may help to suggest the diagnosis.³ A bruit over the neck, diminished cranial and cervical pulses, and an ipsilateral Horner's syndrome may also be found.

Despite these characteristics and suggestive features, the diagnosis is often missed, and a high index of suspicion is required. The definitive diagnosis is made by arteriography. CT scans are usually obtained in these patients as a matter of urgency to exclude operable intracranial collections, but in general CT scan is of little help in the early stages, usually being normal or demonstrating the effects of the primary brain injury. Sequential CT scans will show the development of an infarct and these changes may take 12 h to develop. Such a sequence may suggest the diagnosis.⁹

Many patients with minor degrees of narrowing or good collateral circulations and no emboli may go completely unnoticed, and it is unknown what proportion of these injuries remains clinically silent. A long delay in presentation with neurological deficit occurs in a small number of patients,^{3,4} and usually as a result of thrombo-embolism from a stenosed artery or a false aneurysm.

In large series of cases collected from the literature, the mortality rate of these injuries has been high (38% in Yamada's series and 30% in Krajewski's).^{3,10} A high rate of permanent neurological deficit has been reported: 82% of survivors in Yamada's 51 cases and 42% of survivors in Krajewski's series had permanent neurological deficit.

Given the high morbidity rates, the choice of therapeutic option may be important. A number of conflicting recommendations, however, has been made. Direct surgical intervention with combinations of thrombectomy,¹¹ vein graft, aneurysmectomy, extracranial intracranial bypass and other vascular procedures have been recommended.⁴ Some of these are technically difficult, especially in the region of the high internal carotid, but good results have been claimed in small series.¹² Carotid

ligation, as a one-step or a staged procedure, has also been used.⁵ Intravenous streptokinase as a thrombolytic agent has been reported.¹³ Various longer term anticoagulants such as aspirin, dipyridamole and warfarin have been suggested. Finally, no treatment at all may be offered.

The prevailing difficulty in this entity has been to gather sufficient clinical experience on which to make rational recommendations regarding treatment. It is unlikely that a large enough series could be collected and studied prospectively, besides which, the variation between cases is large. It, therefore, seems prudent to judge every case individually with respect to established neurological deficit, continuing deficits, transient ischaemic episodes, the general status and prognosis of the patient, and feasibility of surgical repair.

All cases in the present series were managed conservatively and showed resolution of arterial defects on long-term follow-up with angiography. This included the virtual disappearance of a high internal carotid artery aneurysm (Case 2), which in other centres may have been managed by more aggressive means.

This feature of the natural history of the condition is indicative of the apparent safety of conservative management.

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