Case reports

Delayed Dissection of the Internal Carotid Artery Following Major Facial Trauma: A Case Report

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ABSTRACT

Dissection of the internal carotid artery is often caused by trauma to the face or neck. It usually has a delayed onset neurological presentation, a partial middle cerebral artery territory syndrome, ‘normal’ early CT scan, MRI evidence of middle cerebral artery occlusion, progressive partial or complete neurological recovery, and duplex scan evidence of a reestablished lumen in the internal carotid artery after 10 weeks.

A case is reported of a dissection of the right internal carotid artery in a patient with severe facial trauma. The patient presented with a left sided hemiplegia 8 hours after a motor vehicle accident. A cerebral CT scan performed 16 hours after the accident revealed a small wedge shaped area of cerebral infarction within the right temporo-parietal region. An MRI angiogram performed four days after the accident revealed a right carotid artery dissection with an occlusive thrombus of the dissected portion of the right internal carotid artery and right middle cerebral artery and a haemorrhagic infarct of the right parieto-occipital lobe.

The patient was anticoagulated and over the next two weeks made a slow recovery, using her left hand effectively and walking unaided. Four months after the accident a duplex scan revealed that the right carotid artery lumen was patent with normal arterial flows. Five months after the accident the patient had returned to work. (Critical Care and Resuscitation 1999; 1: 285-287)

Key words: Carotid artery dissection, internal carotid artery, facial trauma, cerebral infarction

CASE REPORT

A previously healthy 50 year old female was admitted at 0100 hours to the Emergency Department one and a half hours after a car accident. At the time of the accident the patient was wearing a loosely fitted seatbelt and the motor vehicle was travelling at approximately 80 kph. On admission, the patient was alert and orientated with a Glasgow coma score (GCS) of 15. She was moving all limbs to command and was haemodynamically stable. Examination of her face...
showed severe facial swelling with a left sided epistaxis and small amount of fresh blood appearing from the left ear. There was a large deep laceration over the bridge of the nose. The mid-face was mobile and there were bilateral periorbital haematomas.

Chest, skull, cervical spine and pelvic X-rays were within normal limits. The facial X-rays showed multiple mid-facial fractures, including the lateral wall and floor of the right orbit, the floor of the left orbit, the right zygomatic arch in two places and the nasal bone complex.

At 0500 hours the patient was admitted to the Critical Care Area for observation. At 0730 hours she developed a left hemiplegia. On examination the patient was alert and orientated with a GCS 14. Her pupils were small and reactive, her left upper limb was flaccid and hyporeflexic, with no response to pain, and her left lower limb was flaccid, hyperreflexic, and withdrew to pain. The left sided Babinski reflex was positive (upgoing). A cerebral CT scan showed extensive mid-facial fractures with no obvious intracerebral pathology. The patient developed a progressively more dense left hemiplegia, aphasia, and drowsiness with a GCS 10. At 1035 hours she was intubated, prior to helicopter transfer to a major neurosurgical centre.

A repeat CT scan of the head at 1600 hours showed a hypodense lesion with a wedge shaped appearance in the right temporo-parietal lobe, suggestive of infarction. The right middle cerebral artery appeared patent but a carotid artery dissection with embolic phenomena was considered likely (Figure 1).

It was decided not to commence anticoagulant therapy because of the facial injuries, and the patient was treated with continued ventilation, sedation and mild hypothermia.

A duplex scan of the carotid arteries on day 3 showed a dissection with near occlusive thrombosis of the dissected portion of the right internal carotid artery (ICA). A MRI scan on day 4 showed a hyperintense area (indicative of a haemorrhagic infarct) in the left parieto-occipital lobe and a right middle cerebral artery territory infarct (Figure 2).

Figure 1. A cerebral CT scan showing a slight hypodense lesion in the right temporo-parietal region.

Figure 2. MRI scan demonstrating a hyperintense area of the right parieto-occipital region.

Figure 3. A Cerebral angiogram demonstrating an occluded right middle cerebral artery.
MRI angiography revealed the right middle cerebral artery to be occluded, indicating stenosis, spasm or embolus (Figure 3). Heparin and warfarin were commenced on day 4, with the APTT kept greater than twice normal. The patient was extubated on day 5 and transferred to the ward on day 6. She made a progressive recovery and by day 17 she was using her left hand functionally, walking with longer strides and she was able to pick up a pen from the floor. It was elected not to pursue a surgical option in regard to the facial fractures and the patient has made a good functional recovery in this area.

A duplex scan 4 months later showed the carotid lumen had returned to near normal diameter with normal blood flow velocities in the lumen. The warfarin therapy has been continued, maintaining an INR 2.5 - 3.0. The patient returned to work some 5 months after the accident.

**DISCUSSION**

Traumatic carotid artery dissection is a major cause of cerebral infarction in the young. The extracranial portion of the ICA is much more frequently involved than the intracranial portion with the segment of the ICA just proximal to the petrous bone being most susceptible. When a haematoma dissects the media and adventitia of the carotid artery wall, the outer wall weakens and may dilate without narrowing the lumen. Rupture through the intima causes the formation of thrombus within the lumen and may lead to a critical occlusion of the internal carotid artery or a thrombembolic episode. ICA dissection is mainly characterised by local signs such as headache, Horner’s syndrome, pulsatile tinnitus and cranial nerve palsies. Nearly all cases have an episode of delayed deterioration after the initial injury. The time of onset varies from 4 to 36 hours.

An embolus to the brain usually causes the sudden onset of a focal neurological deficit that is maximal within a few hours. Deficits correspond to the areas of the cerebral cortex supplied by the affected artery. The middle cerebral artery in this patient was occluded in its distal portion resulting in a partial middle cerebral territory syndrome. This has been previously reported. Cortical collateral blood flow and differing arterial configurations are probably responsible for the variable symptoms and signs, which may include a combination of sensory and motor deficits, aphasia and hemianopia. In the MR angiogram one can see not only the occluded middle cerebral artery but also the increased collateral flow from branches of the posterior cerebral artery (Figure 3).

It is difficult to say with certainty the exact mechanism of injury in this patient, but it is probably a combination of a rapid severe neck rotation and hyperextension injury causing stretching of the distal internal carotid artery, and blunt trauma from the patient’s seatbelt to the right side of her neck, resulting in the dissection. Both of these insults have been linked to this type of injury previously.

The patient also presented treatment problems as severe facial injuries contraindicated the early use of anticoagulation therapy. Hypothermia and sedation were used to decrease cerebral oxygen consumption prior to the commencement of heparin on day 4. After awakening and extubation the patient made a progressive recovery.

This patient demonstrates many of the classical signs, symptoms and radiological appearances of this condition, including delayed neurological presentation following facial or neck trauma, partial middle cerebral artery territory syndrome, ‘normal’ early CT scan, MRI evidence of middle cerebral artery occlusion and collateral flow, duplex scan evidence of ICA dissection with partial thrombotic occlusion, progressive partial or complete neurological recovery, and duplex scan evidence of a reestablished lumen in the ICA after 10 weeks.

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**REFERENCES**

1. Easton JD, Hauser SL, Martin JB. Cerebrovascular Diseases. Chap 366, Harrison’s Principles of Internal Medicine, Ed 14, 1998