OCCLUSION OF THE VERTEBRAL ARTERY IN CERVICAL SPINE DISLOCATIONS

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We studied 12 consecutive patients with facet joint dislocation in the cervical spine to assess the incidence, site and clinical sequelae of occlusion of the extracranial vertebral artery. Intra-arterial digital subtraction angiography was performed after the orthopaedic management of the dislocations. This demonstrated vertebral artery occlusion (one bilateral) in five of the seven patients with bilateral dislocations and in four of the five patients with unilateral dislocations.

Two of the nine patients with vertebral artery occlusion had neurological deficits above the level of the injury, all of which resolved spontaneously within two months. In our experience, a distraction–flexion injury appears to be the most common cause of closed traumatic vertebral artery occlusion.

Vertebral artery injury, the syndrome of Babinski-Nageotte (Mehalic and Farhat 1974), may result from penetrating injuries (Golueke et al 1987), chiropractic manipulation (Mehalic and Farhat 1974), prolonged abnormal positioning of the neck (Okawara and Nibbelink 1974), birth trauma (Yates 1959) or from closed head and neck trauma (Six et al 1981). Damage to the artery as a result of closed injury is considered to be rare (Kobernick and Carmody 1984).

Extracranial occlusion of the vertebral artery may cause serious and even fatal neurological deficit due to brain-stem and cerebellar infarction (Heros 1979; Six et al 1981). This may result in stroke (Hilton-Jones and Warlow 1985) or may give rise to isolated or combined symptoms and signs of altered consciousness, speech defects, diplopia, blurred vision, nystagmus and dysphagia (Kobernick and Carmody 1984). Loss of blood supply to the posterior inferior cerebellar artery (a branch of the vertebral artery) may cause Wallenberg's syndrome with cranial nerve deficits (V, IX, X, XI), Horner's syndrome, ataxia, dysmetria and contralateral loss of pain and temperature sensation (Golueke et al 1987). A bruit and an expanding mass in the neck may be clinically detectable.

The mechanism of injury to the vertebral artery by closed trauma is stretching and tearing of the intima and media, dissections, mural thrombosis, clot propagation, aneurysmal dilation and occlusion (Carpenter 1961; Lyness and Simeone 1978). Slow progression of the thrombosis or gradual swelling of the vessel wall may allow an asymptomatic interlude of a few hours between the trauma and the onset of neurological symptoms (Marks and Freed 1973).

Unilateral occlusion of the vertebral artery seldom results in a neurological deficit if the collateral supply through the other vertebral and posterior inferior cerebellar arteries is sufficient (Golueke et al 1987). Bilateral occlusion may also be asymptomatic (Six et al 1981) but it may cause neurological deficit or even death (Marks and Freed 1973).

We could find no previous reports of the incidence, pattern of injury or neurological manifestations of vertebral artery occlusion caused by facet joint dislocations of the cervical spine and have therefore studied these issues.

PATIENTS AND METHODS

We studied prospectively 12 consecutive patients with facet joint dislocations of the cervical spine admitted to our hospital. In seven patients the facet joint dislocations were bilateral and in five unilateral. All the patients had sustained a distraction–flexion injury (Allen et al 1982) in some form of motor vehicle accident. There were 10 men and two women with an average age of 38.8 years (range 27 to 50).
The bony injury was at the C5/6 level in seven cases, C6/7 in three and at C4/5 in two. The anterior and lateral displacements of the dislocated vertebral body were measured and recorded. History-taking and neurological examination of the patients were specifically directed to the symptoms and signs of insufficiency of the vertebral and posterior inferior cerebellar arteries. After reduction and stabilisation of the dislocations, intra-arterial digital subtraction angiography (Seeger and Carmody 1985) of both vertebral arteries was performed on all the patients.

RESULTS

Unilateral occlusion of the vertebral artery was demonstrated in four of the five patients (80%) with unilateral facet joint dislocations. One of these patients, in addition to the unilateral occlusion, also had significant stenosis of the contralateral artery (Figs 1 to 3). Five of the seven patients (71.4%) with bilateral dislocations had vertebral artery occlusion, unilateral in four and bilateral in one. The average anterior displacement of the dislocated vertebral body was 8.8 mm (range 4 to 21) in patients with vertebral artery occlusion and 14 mm (range 7 to 22) in patients with normal angiograms. The level of occlusion was that of the dislocated facet joint in only three of the 10 occluded vertebral arteries. Five arteries were occluded within 2 cm of their origins from the subclavian artery and the other two at two spinal levels below the dislocated joints.

Clot propagation was thus very common in the injured vertebral arteries, seven of the 10 cases showing propagation for more than two vertebral segments below the injured level.

None of the three patients with normal vertebral artery angiograms had any neurological deficit above the level of potential spinal cord injury, but two of the nine patients (22%) with vertebral artery occlusion had more cranial neurological deficits. Both these patients had bilateral facet joint dislocations at the C5/6 level. One, with bilateral occlusion, had blurring of vision which cleared up within 12 hours and dysphagia which persisted for one month. The other, who had unilateral occlusion had inability to speak which cleared up within 30 minutes and blurring of vision which persisted for two months.

One other patient with unilateral vertebral artery occlusion gave a history of loss of consciousness for 45 minutes following his injury, but he had also sustained a head injury and had a left-sided frontoparietal abrasion. His transient loss of consciousness was attributed to the head injury and not to vertebral artery occlusion. Neither an enlarging neck mass nor a bruit was detected in any of the patients.

DISCUSSION

The incidence of vertebral artery occlusion in our series of facet joint dislocations reached alarming proportions at 75% of the patients. However, our results support the findings of Golueke et al (1987) that unilateral vertebral artery occlusion seldom results in permanent neurological
deficit. However, transient neurological symptoms are not uncommon; they occurred in two of our nine patients with vertebral artery occlusion.

This finding gives cause for concern and indicates that a meticulous search should be made for the neurological sequelae of vertebral artery occlusion in every patient with a cervical facet joint dislocation. The potential for serious neurological deficit or a fatal outcome after even a unilateral occlusion must be recognised (French and Haines 1950; Heros 1979). Approximately 15% of patients have hypoplasia of one vertebral artery; this emphasises the fact that there may not be sufficient collateral arterial supply in a patient with unilateral occlusion.

Hyperextension injuries, with or without lateral flexion and rotation, have previously been accepted as the most common mechanism of closed injury to the vertebral artery (Simeone and Goldberg 1968; Six et al 1981). However, distraction–flexion injury is much more common, accounting for 76% of cervical spine injuries admitted to our hospital, while hyperextension accounted for only 7%. Distraction–flexion injury with facet joint dislocation, is therefore, in our experience, the most common cause of non-penetrating injury to the extra-cranial vertebral artery.

Pelker and Dorfman (1986) attributed the low incidence of vertebral artery injury in closed trauma to the fact that the vertebral artery is surrounded by bone and lies deep in a protecting layer of soft tissue. Our results indicate that the fibro-osseous tunnel does not offer adequate protection, but that the artery is at risk, especially in the mid-cervical region. All seven of our patients with facet joint dislocations at the C5/6 level (where the vertebral artery is contained within the fibro-osseous tunnel), had occlusions, which were bilateral in one case. Furthermore, both patients with transient neurological symptoms had facet joint dislocations at this level.

At other levels, one of two patients with injury at C4/5 and one of three at C6/7 had vertebral artery occlusion. The vertebral artery does not enter the foramen transversarium of C7 and is therefore more mobile at the C6/7 level; this may explain the lower incidence of injury at this level.

Conclusion. The high incidence of vertebral artery occlusion in cervical spine facet joint dislocations, with transient neurological symptoms in some patients, means that a meticulous search should be made for any neurological changes in these patients. Vertebral artery angiography is indicated in all patients with neurological deficit above the level of potential spinal cord injury. Distraction–flexion injury was the most common cause of closed traumatic vertebral artery occlusion.

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REFERENCES


