Bilateral Vertebral Artery Occlusion Following Cervical Spine Trauma
—Case Report—

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Abstract

A 41-year-old female presented with a rare case of bilateral vertebral artery occlusion following C5-6 cervical spine subluxation after a fall of 30 feet. Digital subtraction angiography showed occlusion of the bilateral vertebral arteries. Unlocking of the facet joint, posterior wiring with iliac crest grafting, and anterior fusion were performed. The patient died on the 3rd day after the operation. This type of injury has a grim prognosis with less than a third of the patients achieving a good outcome.

Key words: cervical subluxation, bilateral vertebral artery occlusion, vertebral artery angiography, stroke

Introduction

Cervical spinal trauma has an incidence of 5 cases per 100,000 per year and occurs mainly in people in the third decade. The etiology varies from motor vehicle accidents (40%) to falls (30%) and other causes such as yoga exercise, sports accidents, etc. The consequences vary from mild to severe, including spinal cord and vertebral artery (VA) injuries.

The VA in the neck courses through the transverse foramen, which provides some protection from penetrating and blunt trauma, but increases susceptibility to damage after cervical spine injury. Injury to the cervical VA usually occurs after blunt trauma and is associated with spine injury. Head injury is also frequently present, so cerebellar and brain stem ischemia symptoms can be missed or interpreted only as the result of the head injury. The symptoms due to VA occlusion usually manifest in the first 24 hours after trauma. Angiographic injury of the VA occurs in 46% of cases of midcervical spine fracture or subluxation, but is rarely symptomatic as less than 40 such cases have been reported.

Bilateral VA occlusion is less frequent. We describe a case of bilateral VA occlusion caused by traumatic cervical subluxation after a high fall.

Case Report

A 41-year-old female was admitted to our emergency department after a fall from a height of 30 feet. Physical examination revealed ecchymotic areas on the posterior aspect of the neck. The patient was alert and fully oriented on admission. Cranial nerve examination was normal. She was quadriplegic and had anesthesia from the C-5 dermatome down. Cervical radiography revealed a C-6 compression fracture, and C5-6 anterior subluxation with bilateral VA occlusion (Fig. 1). Computed tomography (CT) of the cervical spine demonstrated bilateral transverse foramen fractures of the C-6 vertebra (Fig. 2). Cranial CT was unremarkable. Emergent digital subtraction angiography was performed. Vertebral angiography showed bilateral VA occlusion at the C-6 level (Fig. 3). On carotid angiography, the collateral circulation from the anterior system was not enough to support the brain stem.

The patient was rapidly placed in cervical traction.
using Crutchfield tongs, adding weight in 5-pound increments every 10 to 15 minutes until reduction was achieved. Corticosteroid administration was started. She became drowsy and lost response to speech, but not to pain. The bilateral cornea reflexes were diminished. Her changing clinical status was apparently due to brain stem ischemia caused by bilateral VA occlusion, since the cranial CT was unremarkable. Other possibilities were embolization of thrombus due to manipulation, occlusion of collateral channels to the VAs distal to the occlusion, and hemodynamic compromise with subsequent decreased blood flow through the circle of Willis to the basilar system. Anticoagulant and hyperperfusion therapy was begun. To expose both VAs, to perform decompression and stabilization and so to achieve the continuity of the blood supply through the posterior circulation, the patient was taken emergently to the operating room.

The posterior approach was used to reduce the locked facets. Decompression of the transverse foramen revealed that the wall of the VA was intact, but the artery was pulseless. The right VA was torn and started bleeding from the proximal site, so was tied proximally and distally. Posterior fusion from the C3-4 vertebrae superiorly to the C-7 vertebra inferiorly was achieved using wire and iliac crest graft. Then, the anterior approach was used to perform a C-6 corpectomy and C5-6 fusion with iliac crest graft. Intraoperatively, the patient had a cardiac arrest related to brain stem ischemia from which she was successfully resuscitated.

When she woke up, she was breathing spontaneously, but was kept intubated because of altered mental status. Her level of consciousness worsened rapidly. She started vomiting, then her gag reflex and spontaneous breathing disappeared after 7 hours. She was pronounced dead on the 3rd postoperative day.
Discussion

Extracranial injury of the VA usually occurs in three locations. The first location is where the VA enters or comes close to the C-6 transverse foramen. The mobility of the artery suddenly decreases as it enters the transverse foramen of C-6. The amount of flexion extension in the cervical spine is important at the C-6 level. Hyperextension injury will stretch the artery where it enters the foramen and mural hemorrhage or intimal injury may occur. Thrombosis may spread also distally and emboli may be released. The second location is the midcervical region. Although the bony canal formed by the transverse foramen provides same protection, the VA is tethered and exposed to the risk of injury due to cervical spine dislocation. The third location is where the VA crosses the atlanto-occipital and atlanto-axial joints. The VA is vulnerable to injury at this level because of the major change of direction of the VA, its fixation at the atlanto-occipital membrane and the great mobility of the upper cervical spine. Injury to the VA can occur with upper cervical spine dislocation, or without spine trauma. Cadaver studies have shown that VA can be occluded or narrowed at this level by head rotation.

The associated spine injury may be a dislocation or a vertebral body fracture with transverse foramen fracture. All previous patients had lateral dislocation. Four of five patients with unilateral facet dislocation had VA occlusion, and five of seven patients with bilateral subluxation had VA occlusion, suggesting a relationship between facet dislocation and VA occlusion. VA occlusion can also occur without associated spine injury.

The posterior circulation is supplied by both VAs, so unilateral VA occlusion, especially the non-dominant VA, may not cause ischemic symptoms. VA occlusion was present in nine of 12 consecutive patients with facet joint dislocation, but was symptomatic in only two patients. Facet injury occurred in 15% of cases of cervical spine trauma (96 of 640 cases) but only five of 96 patients had symptoms suggestive of vertebrobasilar ischemia. Abnormalities of the VAs occurred in nine of 37 patients with major blunt cervical trauma. The one patient with bilateral abnormalities had non-visualization of the left VA and narrowing of the right VA, and was the only patient manifest symptoms due to VA occlusion. Lateral mass fracture involving the foramen transversarium or facet dislocation between the C-2 and C-6 vertebrae was associated with angiographic VA injury in 12 of 26 patients, but no patient was symptomatic and none had bilateral VA injury.

Symptomatic VA injury usually manifests as brain stem and cerebellar ischemia symptoms in the first 24 hours after trauma. Occasionally these symptoms may be delayed further, and a lucid interval of up to 3 months has been reported. VA injuries due to hyperextension might occur after craniospinal junction trauma. Neck movement may increase symptoms in patients with a history of cervical trauma, so VA injury should be suspected in this situation. Thrombosis with cervical VA injury is common because the blood flow will become relatively slow. Spread of the thrombosis from the cervical to the intracranial VA segment will cause brain stem ischemic infarction. 

Angiography can usually identify VA injury after recognizing the ischemic neurological deficits. Angiography will certainly increase the rate of diagnosis of this type of injury. Magnetic resonance (MR) angiography is an attractive modality for the diagnosis of VA injury, since most patients with cervical spine injuries will undergo MR imaging for the spinal lesions.

The goal of the treatment for traumatic VA occlusion should be to straighten and immobilize the spine. This prophylactic approach may be sufficient for stable and asymptomatic patients. Treatment of VA dissection can begin with heparin and continue with oral anticoagulants for an interval of 3 to 6 months. Surgical treatment is rarely recommended for such cases. However, if the dominant VA is injured, reconstruction and graft or extracranial-intracranial bypass grafting after balloon occlusion should be considered. In this case, a short vein graft bypass distal and proximal to the torn VA portion might be preferred to protect the brain stem from ischemia.

Only 10 cases of bilateral VA occlusion have been reported (Table 1). Unlike unilateral VA occlusion most in which only 20% of patients were symptomatic, most patients with bilateral VA occlusion are symptomatic. Only one patient did not have any symptoms related to the bilateral VA occlusion, possibly due to adequate collateral supply from the posterior communicating arteries. Two patients did not have symptomatic spinal cord injury. None of the 11 patients had subluxation in the midcervical spine mainly at the C5–6 levels. Although the bilateral VA occlusion usually corresponded to the level of bony trauma, occasionally it did not, and one patient had no bony injury. This is probably related to the difference in mobility of the VA segments, with stretch injury occurring on extreme rotation or extension. The outcome was very grim in only three patients with a good outcome.
Table 1  Reported cases of bilateral vertebral artery (VA) occlusion

<table>
<thead>
<tr>
<th>Author (Year)</th>
<th>Age/ Sex</th>
<th>Trauma</th>
<th>Spinal cord injury</th>
<th>CrN deficit</th>
<th>Altered MS</th>
<th>Interval</th>
<th>Spine injury</th>
<th>VA occlusion</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simeone and Goldberg (1968)38</td>
<td>40/M</td>
<td>MVA</td>
<td>—</td>
<td>—+</td>
<td>1 day</td>
<td>C3-4 and C5-6 dislocation</td>
<td>rt at C2-3, lt at 2.5 cm from the origin of VA</td>
<td>thrombectomy</td>
<td>D</td>
<td></td>
</tr>
<tr>
<td>Schneider et al. (1970)35</td>
<td>14/M</td>
<td>football</td>
<td>quadruplegia C-6 SL</td>
<td>++</td>
<td>1 day</td>
<td>none</td>
<td>bil at foramen magnum</td>
<td>rt at C-5, lt at origin of VA</td>
<td>G</td>
<td></td>
</tr>
<tr>
<td>Marks and Freed (1973)32</td>
<td>16/M</td>
<td>football</td>
<td>quadruplegia C-5 SL</td>
<td>+ —</td>
<td>36 hrs</td>
<td>C5-6 subluxation</td>
<td>bil at C-3</td>
<td>anti-coagulation</td>
<td>G</td>
<td></td>
</tr>
<tr>
<td>Six et al. (1981)39</td>
<td>25/F</td>
<td>MVA</td>
<td>—</td>
<td>+ —</td>
<td>4-5 hrs</td>
<td>C2-3 subluxation</td>
<td>bil at C-1</td>
<td>anti-coagulation</td>
<td>SD</td>
<td></td>
</tr>
<tr>
<td>Bose et al. (1985)34</td>
<td>20/M</td>
<td>rugby</td>
<td>quadruplegia C-5 SL</td>
<td>++</td>
<td>3 mos</td>
<td>C4-5 subluxation</td>
<td>bil at C-4-5</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>George and Laurian (1989)12</td>
<td>67/M</td>
<td>quadruplegia</td>
<td>—</td>
<td>—</td>
<td>C4-5 subluxation</td>
<td>bil at C-4-5</td>
<td>D</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Louw et al. (1990)2</td>
<td>34/M</td>
<td>MVA</td>
<td>quadruparesis C-5 SL</td>
<td>++</td>
<td>30 min</td>
<td>C5-6 subluxation</td>
<td>bil at C5-6</td>
<td>D</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parent et al. (1990)23</td>
<td>28/M</td>
<td>MVA</td>
<td>quadruparesis C-6 SL</td>
<td>— —</td>
<td>C-5 fracture</td>
<td>bil at C5-6</td>
<td>D</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Miyachi et al. (1994)27</td>
<td>49/M</td>
<td>crush</td>
<td>quadruplegia C-5 SL</td>
<td>+ +</td>
<td>3 days</td>
<td>C4-5 subluxation</td>
<td>rt at 2.5 cm from the origin of VA, lt at C-5</td>
<td>anti-coagulation</td>
<td>D</td>
<td></td>
</tr>
<tr>
<td>Present case</td>
<td>41/F</td>
<td>fall</td>
<td>quadruplegia C-5 SL</td>
<td>—+</td>
<td>6 hrs</td>
<td>C5-6 subluxation</td>
<td>bil at C5-6</td>
<td>ligature (rt)</td>
<td>D</td>
<td></td>
</tr>
</tbody>
</table>

CrN: cranial nerves, D: dead, G: good, MD: moderate disabled, MS: mental status, MVA: motor vehicle accident, SD: severely disabled, SL: sensory level.

Treatment of bilateral VA occlusion is still unclear varying from anticoagulation4,27,30 to ligation and thrombectomy.38 Reduction of the subluxation was performed in most cases. Prompt prophylactic cervical traction is recommended in cases of cervical spine injury.38,39 The most frequently used specific treatment was anticoagulation.4,27,30 However, it is difficult to make any valid conclusion from the small number of reported cases.

Bilateral VA injury after cervical trauma occurs infrequently, but symptomatic in most cases. Angiography is important for the diagnosis of this injury. Bilateral VA injury is frequently associated with midcervical subluxation (mostly C5-6) but can occur without bony injury. Treatment is mainly reduction of the associated subluxation. The prognosis is grim with less than a third of the patients achieving good outcome.

References

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