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CASE REPORT

Resolution of traumatic bilateral vertebral artery injury

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Abstract

Purpose Cerebrovascular ischaemia is a rare but serious complication of damage to the carotid or vertebral arteries in the neck caused by blunt injury to the neck. Screening for blunt cerebrovascular injury should be performed in patients with certain signs or symptoms and risk factors. We described a case of traumatic bilateral vertebral artery injury (VAI) including unilateral vertebral arterial occlusion that resolved 3 months post-injury with antiplatelet and direct oral anticoagulant therapy. This resolution of traumatic bilateral VAI is very rare. Vertebral artery injury should be suspected in patients with displaced fracture dislocation of the cervical spine, particularly in the elder and those with ankylosing spondylitis, and therefore imaging of these patients should include a modality to look at the patency of the vertebral arteries.

Case description A 70-year-old man who was diagnosed with ankylosing spondylitis collapsed and presented with tetraplegia. Computed tomography showed C3 fracture dislocation, and magnetic resonance imaging showed a high-signal intensity and intense compression of the spinal cord from C2 to C3. Cerebral angiogram showed left vertebral artery occlusion and right vertebral artery stenosis. Heparin was administered to prevent posterior circulation stroke and he underwent posterior fixation. Three months post-injury, a cerebral angiogram showed the resolution of the bilateral VAI.

Keywords Spinal cord injury · Blunt cerebrovascular injury · Vertebral artery injury · Vertebral artery occlusion

Introduction

Blunt cerebrovascular injury (BCVI) is rare, and the incidence of blunt traumatic vertebral artery injury (VAI) is reportedly < 1% among patients with blunt trauma [1–3]. However, VAI has been reported in around 15% of the patients with high risk for blunt VAI who were screened [4, 5]. In addition, posterior circulation stroke and a mortality rate of up to 8% have been reported in up to 24% patients with blunt VAIs [6–8]. Because VAIs cause potentially serious sequelae, screening for BCVIs should be performed in patients with certain signs or symptoms and risk factors (Table 1) [9]. The Biffl scale is commonly used for the classification of VAI when patients are diagnosed with it [10]. Although the optimal treatment regimen for VAIs remains unclear, the early administration of systemic heparin or antiplatelet therapy has been recommended to reduce the risk of stroke in patients with VAI of grades I–IV [11–16]. Endovascular therapy may represent a treatment option for certain injuries, but there has been no clear indication for its use [17]. Here we describe the case of traumatic bilateral VAI including unilateral vertebral arterial occlusion that resolved 3 months post-injury with antiplatelet and direct oral anticoagulant therapy.

Case description

A 70-year-old man who was diagnosed with ankylosing spondylitis and restricted neck extension and flexion collapsed, hit his forehead and presented with tetraplegia. Cervical spine had impact from anterior to posterior. He was conscious, and his blood pressure was 120/60 mmHg, pulse rate was 66/min, and respiratory rate was 16/min, with an O_2 saturation of 91% on room air. His neurological examination revealed tetraplegia. He presented with dyspnea, and he was intubated and artificially ventilated due to hypercapnia under anesthesia with appropriate precautions against displacing. Computed tomography (CT) showed C3 fracture dislocation, and magnetic resonance imaging (MRI) showed a high

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Table 1 Denver criteria: indications of screening for BCVI

Signs/symptoms of BCVI
Arterial hemorrhage from neck, mouth, nose, ears
Large or expanding cervical hematoma
Cervical bruit in a patient younger than 50 years
Focal or lateralizing neurologic defect: hemiparesis, transient ischemic attack, Horner's syndrome, oculosympathetic paresis, or vertebrobasilar insufficiency
Evidence of cerebral infarction on CT or MRI
Neurologic deficit that is inconsistent with CT or MRI findings
Risk factors for BCVI
An injury mechanism compatible with severe vervical hyperextension with rotation or hyperflexion
Displaced mid-face fracture (LeFort II or III)
Basilar skull fracture involving the carotid canal
Closed head injury consistent with diffuse axonal injury and GCS score < 6
Cervical vertebral body or transverse foramen fracture, subluxation, or ligamentous injury at any level, or any fracture at the level of C1–C3
Near-hanging resulting in cerebral anoxia
Seat belt or other clothesline-type injury with significant cervical pain, swelling, or altered mental status

signal intensity and intense compression of the spinal cord from C2 to C3 (Fig. 1). He received emergency reduction of C3 fracture manually and was fitted with a halo ring and vest to support his neck under radiographic guidance. After these procedures, CT angiogram and cerebral angiogram showed left vertebral artery occlusion (Biffl grade IV) and right vertebral artery stenosis (Biffl grade II) because indications of screening for BCVI were met and VAI was highly suspected (Fig. 2). Brain MRI did not show an abnormally high-signal intensity on fluid-attenuated inversion recovery and diffusion-weighted imaging in the posterior circulation territory. Heparin was administered to prevent posterior circulation stroke immediately after the cerebral angiography; it was then replaced with clopidogrel. Surgical intervention was needed; however, it was delayed because of the need for anticoagulation. He underwent posterior fixation on day 26 post-admission. CT angiogram showed that VAIs did not improve on the post-operation day 1. He was diagnosed with deep vein thrombosis (DVT) post-operatively, and apixaban was administered to reduce the risk of recurrent DVT and pulmonary embolism. Three months post-injury, a cerebral angiogram showed the resolution of the bilateral VAI (Fig. 3). Brain MRI did not show any abnormality. He did not make a recovery, but he was transferred to another care facility for rehabilitation.

Discussion

BCVI is believed to be rare, but cervical spine fracture is a strong risk factor for BCVI. Denver screening criteria (Table 1) demonstrated that patients with cervical

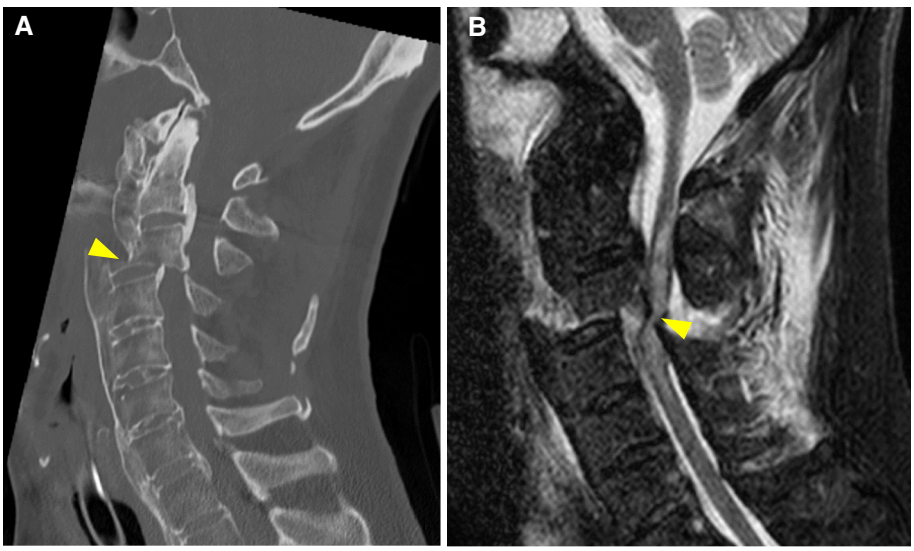

Fig. 1 A 70-year-old man who was diagnosed with ankylosing spondylitis and restricted neck extension and flexion collapsed and presented with tetraplegia. **a** CT image showing C3 fracture dislocation. **b** MRI showing a high signal intensity and intense compression of the spinal cord from C2 to C3

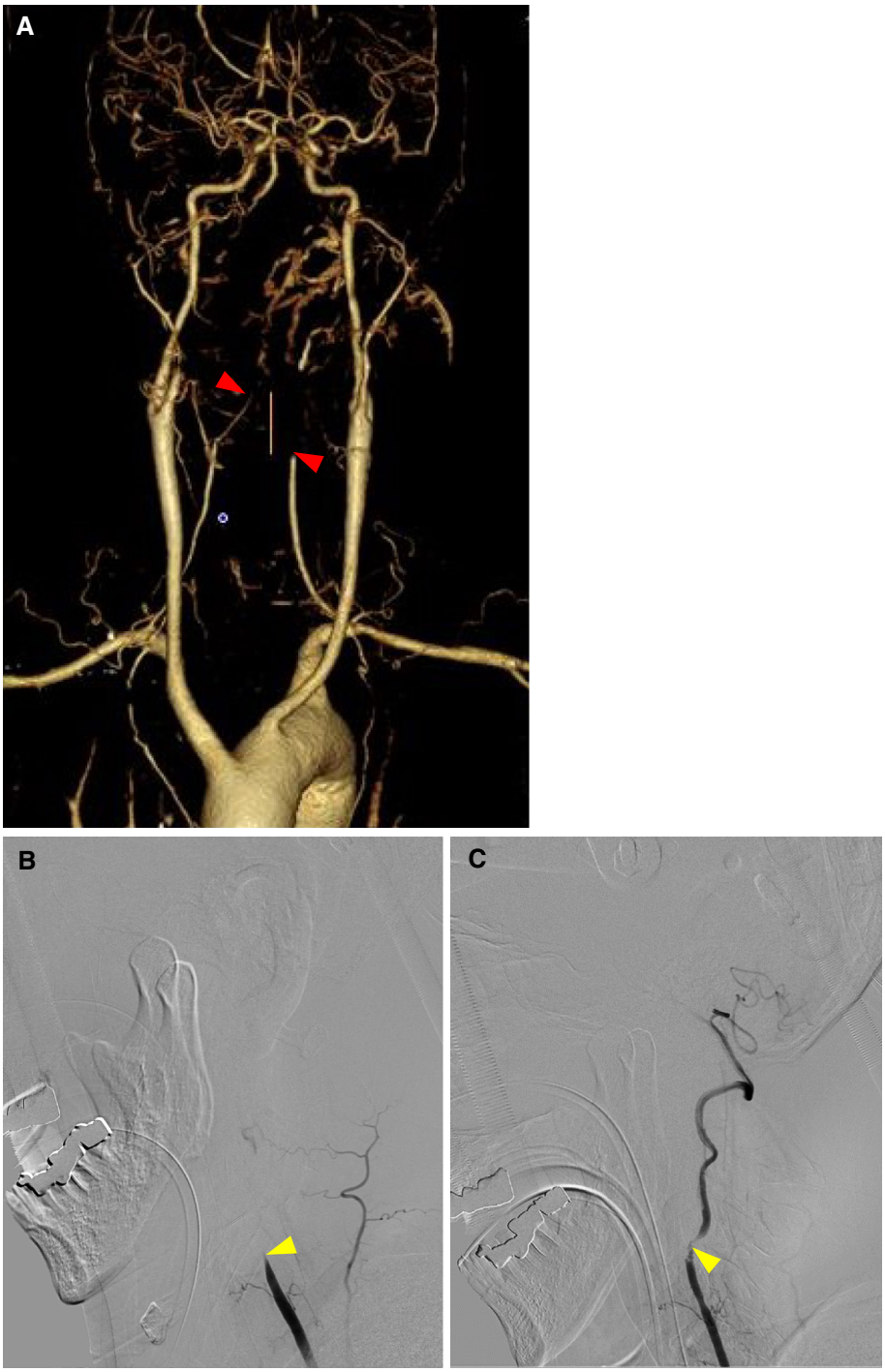

Fig. 2 CT angiogram (**a**) and cerebral angiogram showing left vertebral artery occlusion (**b**) and right vertebral artery stenosis with more than 25% (**c**)

fractures need evaluation of BCVI [18]. Magnetic resonance angiography has been considered as the best screening modality to diagnose BCVI, however, CT angiography or angiography is necessary where the presence or absence of arterial flow void cannot be determined on MRI. BCVI is classified using the Biffl scale (Table 2). VAI of grades III and IV are considered to cause stroke and are less likely to resolve spontaneously compared with grade I and II injuries [19]. In particular, ischemic stroke has been reported in up to 24% patients with vertebral artery occlusion (Biffl grade IV) [20]. Total occlusion of both vertebral arteries may cause brain stem infarction and preventing a posterior circulation stroke is extremely important (Fig. 3).

In the present case, CT showed C3 fracture dislocation and cerebral angiogram revealed left vertebral artery occlusion (Biffl grade IV) and right vertebral artery stenosis (Biffl grade II) on the admission day, with

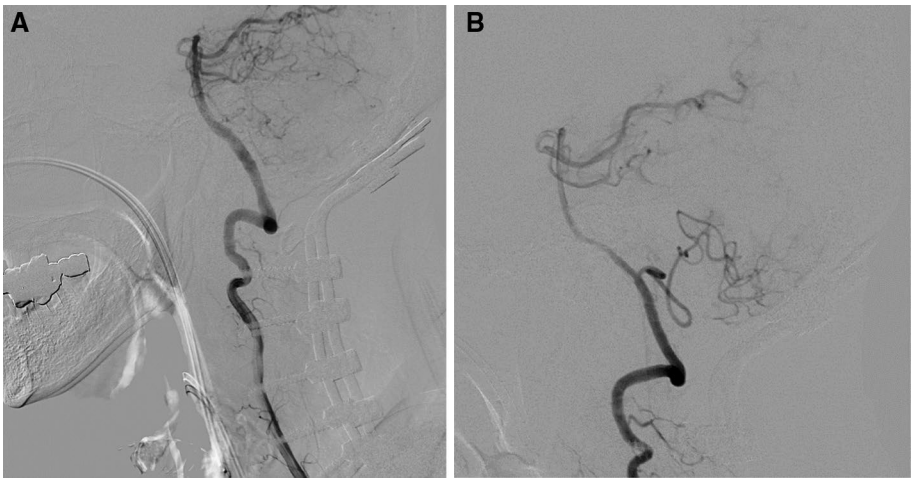

Fig. 3 Cerebral angiogram showing resolution of left (**a**) and right (**b**) vertebral artery 3 months after injury

Table 2 Biffl grading scale for blunt cerebrovascular injuries

Grade	Description
I	Irregularity of the vessel wall, dissection or intramural hematoma with < 25% luminal stenosis
II	Intraluminal thrombus or raised intimal flap, dissection or intramural hematoma with > 25% luminal stenosis
III	Pseudoaneurysm
IV	Vessel occlusion
V	Vessel transection

resolution of bilateral VAI. To our knowledge, this is the first description of the resolution of traumatic bilateral VAI.

It is known that vertebral artery occlusion often spontaneously improves but that it is less likely to resolve. Scott et al. reported that repeated imaging of grade IV VAI demonstrated 65% of their patients to be stable (persistent occlusion), 30% to be improved, and 5% to have the injury resolved at the final follow-up, despite therapeutic systemic anticoagulation [19].

Antiplatelet therapy is recommended for patients with grade IV VAI to prevent posterior circulation stroke. Endovascular stenting is also used for BCVI, however, intravascular stents are reserved for the rare patient with symptomatology or a markedly enlarging pseudoaneurysm because routine stenting entails increased costs and potential risk for stroke [21]. In the present case, anticoagulation therapy was administered in addition to antiplatelet therapy because of DVT.

Single anti-platelet therapy did not lead to re-canalisation of the vertebral artery, whereas direct oral anticoagulant (DOAC) did. However, the collateral circulation via the circle of Willis was responsible for preventing a brainstem stroke rather than the late re-canalisation of the vertebral arteries associated with starting DOAC.

Conclusions

We described a rare case of traumatic bilateral VAI including unilateral vertebral arterial occlusion that resolved 3 months post-injury. Vertebral artery injury should be suspected in patients with displaced fracture dislocations of the cervical spine, particularly in the elderly and those with ankylosing spondylitis, and therefore, imaging of these patients should include a modality to look at the patency of the vertebral arteries.

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Compliance with ethical standards

Conflict of interest There no conflicts of interests.

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