CLINICAL REPORT

Posterior reversible encephalopathy syndrome in a context of isolated cervical spine fracture: CT angiogram as an early detector of blunt carotid artery trauma

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Carotid dissection; Cervical fracture; Angiography; Computed tomography; Posterior reversible encephalopathy syndrome

Summary
Blunt carotid injury associated with cervical spine fractures is a rare entity but potentially lethal. An initial, clinically silent period can be misleading. Prompt diagnosis and treatment are mandatory to avoid neurological damages and death. We present the case of a 36-year-old man diagnosed with an isolated cervical spine fracture, where an associated carotid artery lesion was initially overlooked and diagnosis was made after development of a neurological deterioration secondary to a posterior reversible encephalopathy syndrome (PRES). We discuss a simple algorithm that can be used to make the diagnosis, even during the clinically asymptomatic period of this injury.

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Introduction

Cervical spine fractures commonly occur during high-velocity traumas. The screening for associated cervical spine injury and blunt cerebrovascular injury (BCVI) constitutes a part of the investigation workflow when multiple injuries are present in polytrauma patient. However, in the case of isolated cervical spine fracture, diagnosis of BCVI can be easily missed. The association between cervical spine fractures and cervical vascular injury has been well-documented especially vertebral artery lesion for which specific fracture patterns have been described [1–6]. However, the association of cervical spine trauma and blunt carotid artery injury is uncommon. We report the case of a 36-year-old man who had a car accident and was diagnosed with an isolated cervical spine fracture, where an associated blunt carotid injury was initially missed and complicated by a posterior reversible encephalopathy syndrome (PRES). We insist on the importance of early diagnosis and treatment and we suggest a protocol to avoid missing this potentially lethal lesion.
Case report

A 36-year-old man was the belted driver of a car, which collided with another car at a speed of 80 km/h. He sustained a head and neck trauma without loss of consciousness. Transported with a neck collar to the nearest hospital by an emergency medical assistance service, he was conscious with a Glasgow Coma Scale (GCS) of 15/15 and was complaining of neck pain. The complete neurological assessment was normal. Investigations revealed an isolated left superior articular facet fracture of C7 with minimal antelthesis of C6 over C7 (Fig. 1). He was referred to our regional trauma and spine center for cervical spine lesion treatment. Magnetic resonance imaging (MRI) showed a discoligamentous injury at C6-C7 level. We planned an anterior approach for arthrodesis of this level. At 48 hours of injury (a few hours before surgery), the patient complained of severe headache, vomiting and drowsiness. Physical signs included agitation, hypertension, bradycardia and bilateral Babinski sign. A seizure attack was noted in the intensive care unit, treated promptly with 1 mg of clonazepam. The patient was intubated because of agitation and for investigations. The brain CT scan showed no abnormalities and the diffusion-weighted MRI revealed bilateral multifocal brain hyperintensities (Fig. 2), compatible with a posterior reversible encephalopathy syndrome (PRES). Magnetic resonance angiography of neck vessels showed right internal carotid dissection of more than 50% over 4 cm (Fig. 3). He was treated with anticoagulants with favorable outcome. Surgery was delayed for 2 months after stabilization of the carotid lesion. At 3 months after spine surgery, the patient still had no neurologic sequelae and was taking an antiplatelet agent for 3 additional months. Magnetic resonance angiography at 8 months showed complete regression of carotid artery lesion and anticoagulation was stopped.

Discussion

Blunt carotid injury (BCI) is an uncommon injury with a potentially devastating outcome [7]. This entity is rarely isolated and associated injuries of head, face, skull base and cervical spine are frequent [8–11]. While vertebral arteries are directly injured in the foramen transverse at the fracture level, the mechanism of carotid injury is essentially indirect, with hyperextension and rotation playing a major role in most motor vehicle accidents [7]. The fixed position of the
carotid arteries in the carotid canal at the skull base, make them prone to external compression and stretching by the upper cervical spine lateral masses during hyperextension and rotation. Therefore, one should search for carotid injury at the C1-C2 level and not at the level of the cervical fracture, which is sometimes located in the lower cervical spine (as for our case). Although isolated cervical spine fractures are common and can be managed in a community hospital, one should remember that in some cases, an associated carotid injury may be present without any clinical signs. In fact, the diagnosis of BCVI is frequently delayed and over 40% of patients demonstrate signs and symptoms some time after an initial normal neurological examination [12]. Average time from injury to diagnosis is 53 hours [13]. In other words, a normal neurological examination at presentation does not rule out this injury. Our case is demonstrative. Although our patient did not have major neurological complications and had no sequelae, the overall mortality rates for BCVI fall in the range of 15–40% and permanent neurologic deficit approximates 25 to 40% of survivors [13–15]. Therefore, an early diagnosis is crucial before the onset of stroke. Patients diagnosed early and treated with antithrombotics would avoid neurological events [2,16]. Denver grading scale [3] (Table 1) is helpful for classification and treatment guidelines. The aim of treatment is to prevent development of a neurologic lesion, or progression of an existing one. Treatment options include observation, anticoagulation, thrombolysis, stenting and surgery [7]. Optimal management remains controversial in the absence of prospective trials but several studies demonstrate the crucial role of heparin in reducing mortality [9,13,15]. Grade I may be treated with antiplatelet agents. Grades II, III and IV should be initially treated with anticoagulants. Stenting may be indicated for pseudoaneurysms while surgery is generally considered after failure of medical therapy. Transection is usually fatal and not accessible to treatment. Conventional angiographic screening for all patients presenting with a cervical spine fracture is an aggressive attitude that is not cost-effective. Cothren et al. [17] identified three fracture patterns mandating screening to rule out BCVI: subluxation, transverse foramen and upper cervical spine involvement. According to these recommendations, our patient who had subluxation should have had initial screening. We agree with the authors that recognition of specific fracture patterns reduces imaging requirements but we believe that conventional angiography is not the screening test of choice. The 16-channel multislice computed tomographic angiography (widely available in trauma centers) has an overall sensitivity and specificity of 98 and 100% respectively [18], making this exam the ideal test for screening. Furthermore, the use of recent 64-channel multislice CT angiography would increase the sensitivity even more. CT angiography has the advantages of being readily available and easily incorporated into the routine work-up of trauma patients. We actually integrate this exam to the standard total body CT scan for all our patients who are victims of high velocity traumas regardless of cervical spine injury. In cases of isolated cervical spine fractures, we are following the fracture patterns of Cothren et al. [17] to rule out BCVI with a CT angiography (Fig. 4).

To our knowledge, this is the first reported case of posterior reversible encephalopathy syndrome (PRES) secondary to a traumatic carotid injury. PRES is characterized by transient vasogenic edema mainly involving parieto-occipital brain regions and was first described in 1996 [19]. Common causes include acute hypertension, multiple organ failure, eclampsia, auto-immune diseases, immunosuppressants and acute thrombotic thrombocytopenic purpura [20–22]. Brain edema is due to endothelial injury secondary to hypertension, leading to increased blood-brain barrier permeability [23]. An immune-mediated endothelial dysfunction was recently described as a possible mechanism [22]. Signs and symptoms include headache, visual disturbance, hypertension, drowsiness, vomiting and seizures. MRI findings consist of bilateral multifocal hyper-intense lesions on T2, mainly occipital. Lesions are usually reversible with antihypertensive treatment. Mellon and Rizvi [24] reported, in 2005, the case of a 44-year-old woman with a history of multiple sclerosis, who had spontaneous bilateral carotid artery dissection and PRES. They conclude that endothelial dysfunction associated to hypertension created an environment conducive to the development of PRES. Recently, a case of spontaneous internal carotid artery dissection

Table 1 Denver Grading Scale for traumatic carotid injury [3].

<table>
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<tr>
<th>Grades</th>
<th>Description</th>
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<tr>
<td>I</td>
<td>Luminal irregularity on angiography, or dissection with &lt; 25% stenosis</td>
</tr>
<tr>
<td>II</td>
<td>Dissection with &gt; 25% luminal narrowing, or a raised intimal flap</td>
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<tr>
<td>III</td>
<td>Pseudoaneurysm</td>
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<tr>
<td>IV</td>
<td>Complete occlusion</td>
</tr>
<tr>
<td>V</td>
<td>Transection of the carotid artery</td>
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</tbody>
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Figure 3 MRI angiography revealing dissection of the right internal carotid artery.
associated to a PRES was reported [21], in a 59-year-old female with a history of tongue squamous cell carcinoma. Authors postulated that carotid spontaneous dissection led to baroreceptor complex failure with resulting hypertension and PRES. Baroreceptor reflex failure can induce hypertensive encephalopathy after carotid endarterectomy [25] and after extracranial carotid dissections [24]. We described here the first case of blunt carotid dissection with typical signs, symptoms and imaging features of PRES. The baroreceptor complex failure cited above is a plausible mechanism and may explain the hypertensive encephalopathy with resulting PRES.

Conclusion
Blunt carotid injury associated with cervical spine fractures is rare but potentially lethal. Early diagnosis and treatment are the keys to avoid neurologic events. CT angiography integrated in the total body CT scan protocol is an excellent screening method for high velocity trauma patients. Subluxation, transverse foramen and upper cervical spine involvement are specific patterns mandating screening in cases of isolated cervical spine fractures. PRES is a rare syndrome and was described for the first time in association with a blunt carotid injury.

Disclosure of interest
The authors declare that they have no conflicts of interest concerning this article.

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