

# CASE REPORT

Nagoya J. Med. Sci. 77. 507 ~ 514, 2015

## Fatal case of cervical blunt vascular injury with cervical vertebral fracture: a case report

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### ABSTRACT

Blunt cerebrovascular injury (BCVI) is usually caused by neck trauma that predominantly occurs in high-impact injuries. BCVI may occur due to damage to both the vertebral and carotid arteries, and may be fatal in the absence of appropriate treatment and early diagnosis. Here, we describe a case of cerebral infarction caused by a combination of a lower cervical spinal fracture and traumatic injury to the carotid artery by a direct blunt external force in a 52-year-old man. Initially, there was no effect on consciousness, but 6 hours later loss of consciousness occurred due to traumatic dissection of the carotid artery that resulted in a cerebral infarction. Brain edema was so extensive that decompression by emergency craniectomy and internal decompression were performed by a neurosurgeon, but with no effect, and the patient died on day 7. This is a rare case of cerebral infarction caused by a combination of a lower cervical spinal fracture and traumatic injury to the carotid artery. The case suggests that cervical vascular injury should be considered in a patient with a blunt neck trauma and that additional imaging should be performed.

Key Words: blunt cerebrovascular injury, lower cervical spinal fracture, cerebral infarction, trauma, carotid artery

### INTRODUCTION

Blunt cerebrovascular injury (BCVI) is caused by neck trauma<sup>1)</sup> that predominantly occurs in high-impact injuries including motor vehicle-related accidents, car-pedestrian collisions, falls, and skiing or snowmobile accidents.<sup>2-6)</sup> BCVI may be fatal in the absence of appropriate treatment and early diagnosis.<sup>4-7)</sup> However, a lucid interval occurs in many cases,<sup>8)</sup> and this may lead to a delayed diagnosis. Reports on BCVI accompanied by cervical spinal fracture are less common.<sup>1,9)</sup> We experienced a case of cervical spinal fracture caused by a direct blunt external low-energy force. Initially, there was no disturbance of consciousness, but 6 hours later loss of consciousness occurred due to traumatic dissection of the carotid artery that resulted in cerebral infarction. Diagnosis and treatment were delayed due to the atypical manifestations. This case indicates that BCVI should be considered in a patient with blunt neck trauma, particularly when this is

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Received: September 22, 2014; accepted: March 5, 2015

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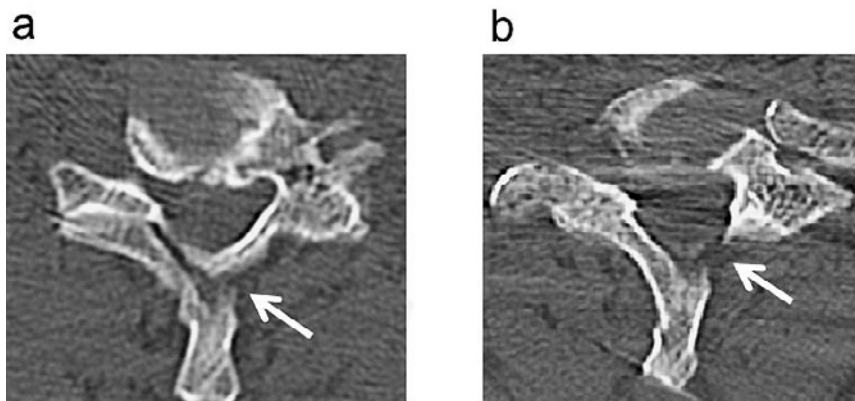
accompanied by a cervical spinal fracture. We report the case and present a literature review.

### CASE REPORT

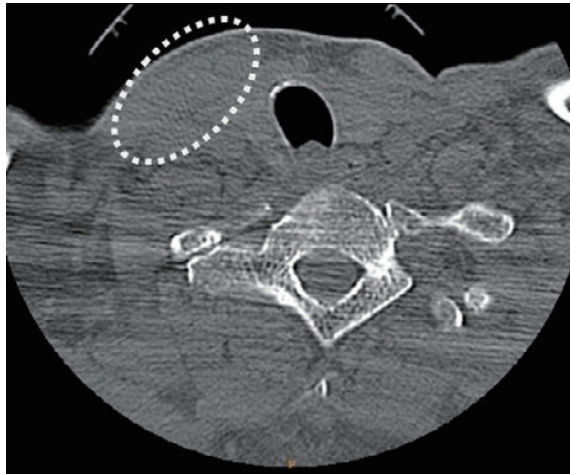
A 52-year-old man who was working at a factory put his head into a big drum-type washing machine. The door closed suddenly and crushed his neck by sandwiching him between the machine and the door. Upon transfer to our hospital, he was conscious with a Glasgow Coma Scale (GCS) of E4V5M6, and a subcutaneous hematoma was present on the right side of the neck (Fig. 1). The patient complained of neck pain and numbness in both hands, but there was no apparent limb paralysis. A spinous process fracture at the C6 and C7 levels was detected in a cervical CT (Fig. 2a,b), and there was swelling of the right neck in the CT (Fig. 3). A narrow spinal canal was apparent on a cervical spinal MRI (Fig. 4a,b). After fixation of a cervical collar, the patient was admitted to the intensive care unit.



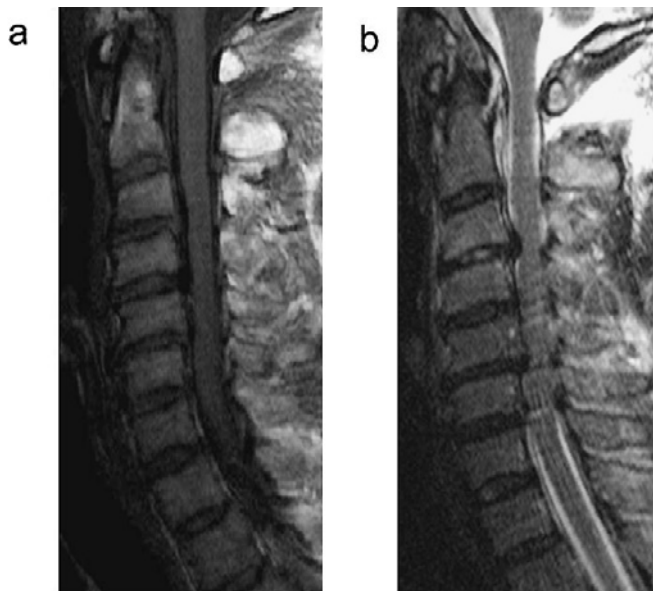
**Fig. 1** Subcutaneous hematoma (arrow) in the neck



**Fig. 2** Cervical CT showed a spinous process fracture (arrow) at the C6 (a) and C7 (b) levels.



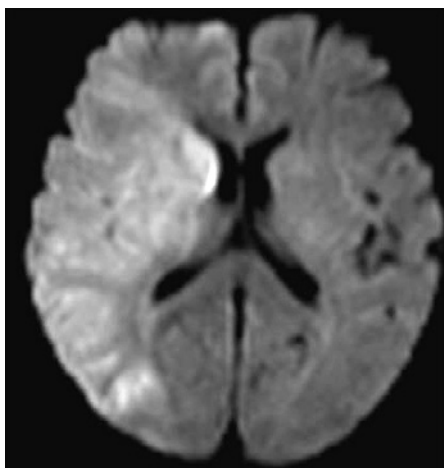
**Fig. 3** Swelling was present around the right neck in cervical CT (circles)



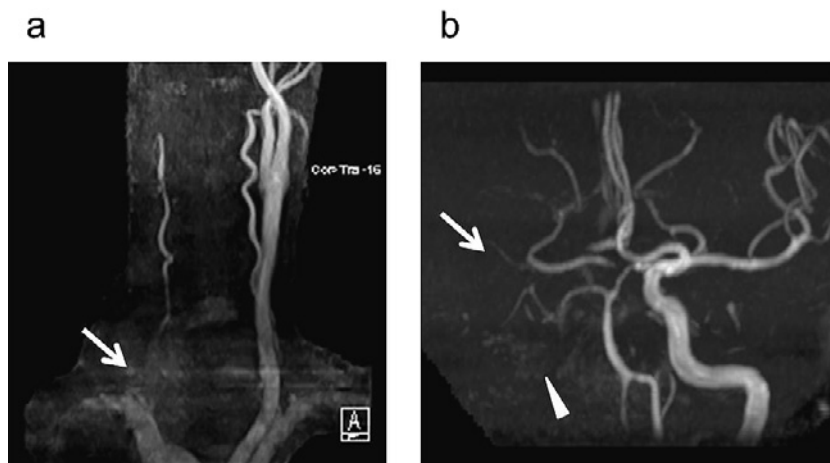
**Fig. 4** Cervical spondylotic changes observed in cervical spine MRI T1 (a) and T2 (b). A hematoma was present around the spinous process.

Six hours after injury, paralysis suddenly developed in the left upper and lower extremities, and a decline in consciousness appeared with a GCS of E1V2M5. Emergency head MRI was performed after consultation with a neurosurgeon, and acute infarction of the right middle cerebral artery perfusion area was found (Fig. 5). MRI angiography showed blockage of the right common carotid artery and internal carotid artery, and hypoperfusion of the middle cerebral artery (Fig. 6a,b). This led to a diagnosis of right cerebral infarction due to acute vascular occlusion associated with right carotid artery dissection in the neck trauma. Edaravone and glycerol were

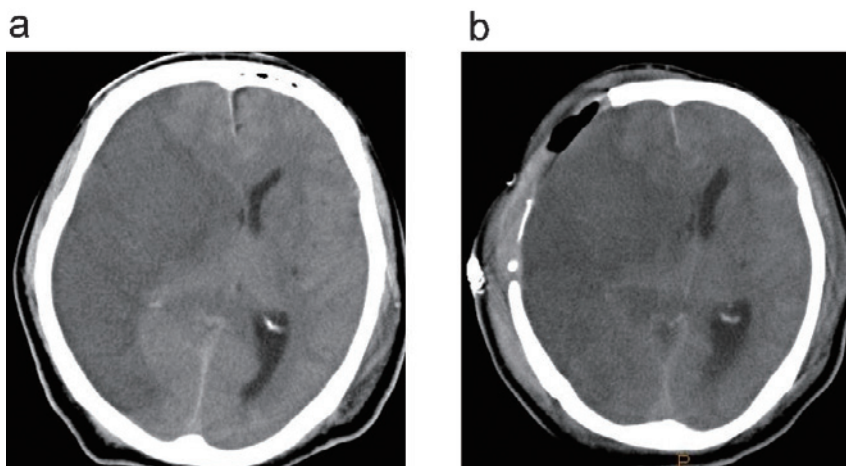
administered to improve brain edema, and mechanical ventilation after tracheal intubation was performed on the same day. On the next day, extensive brain edema was observed in a head CT, and an emergency craniectomy and internal decompression were performed by a neurosurgeon (Fig. 7a,b). However, improvement in the brain edema was insufficient, and the patient died on day 7. The patient's wife and son gave informed consent for submission of this case study for publication.



**Fig. 5** Acute infarction of the right middle cerebral artery based on perfusion of this area in head diffusion-weighted MRI at six hours after injury.



**Fig. 6** (a) Disruption of the common carotid artery at six hours after injury (arrow). (b) Due to occlusion of the right internal carotid artery (triangles) and poor collateral circulation through the posterior communicating (PCOM) artery and anterior communicating (ACOM) artery, the right middle cerebral artery was disrupted (arrow) at six hours after injury.



**Fig. 7** (a) Head CT on the day after injury showed a low-density area in the MCA region and compression of the anterior horn of the lateral ventricle due to swelling, and the midline shift was more than 10 mm. (b) Two days after injury, swelling was worse, and the low-density area had expanded on head CT after craniectomy and internal decompression.

## DISCUSSION

BCVI may occur due to damage to the vertebral and carotid arteries, mainly by blunt neck trauma.<sup>1)</sup> The frequency is between 0.50% and 1.55% of all cases of blunt trauma. Among patients at high risk based on criteria, including an extensive skull base fracture, cervical fracture, cervical soft tissue hematoma, and facial fracture, the incidence of BCVI is much higher, in the range of 24–44%.<sup>4,7,10)</sup> In 185 cases diagnosed with BCVI, Bruns *et al.* found that a motor vehicle collision (52%) was the primary cause of injury, followed by a fall (20%) and collisions involving a motor cycle (11%), a pedestrian (8%), and a cyclist (3%).<sup>11)</sup> Dienstknecht *et al.* also suggested that BCVI could occur after a minor impact, such as cervical traction and heading a soccer ball,<sup>12)</sup> but there are few reports of this condition caused by blunt external force, as described in the present case.

In a study of 17,007 admissions for blunt injury, Cothren *et al.* identified 258 patients with BCVI, including 117 with significant cervical spinal fractures.<sup>9)</sup> Most fracture patterns were subluxations (56 patients), upper C1 to C3 body fractures (42 patients), and transverse foramen fractures (19 patients), whereas spinous process fractures of the lower cervical spine were rare (2 patients).

The Denver Screening criteria<sup>13)</sup> provide a screening protocol for BCVI diagnosis based on injury mechanisms, injury patterns, and symptoms<sup>14)</sup> (Table 1). This protocol can identify trauma patients at risk for BCVI with a sensitivity of 97% and a specificity of 42%.<sup>15)</sup> In our case, “expanding cervical hematoma” and “cervical spinal fracture” were consistent with the Denver Screening criteria, but BCVI was not initially suspected and the patient was not diagnosed with BCVI until after loss of consciousness. Ultrasonography, CT angiography, and magnetic resonance angiography (MRA) are effective noninvasive diagnostic modalities for screening and evaluation of soft tissue in the initial treatment of blunt trauma.<sup>16,17)</sup> Ultrasonography is particularly useful for diagnosis of macrovascular damage and airway narrowing, and is desirable as a secondary examination. Ultrasonography, MRI, and MRA are all relatively easy to use as screening tools.<sup>1)</sup>

**Table 1** Denver Screening Criteria for BCVI.

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Signs/symptoms of BCVI
Arterial hemorrhage
Cervical bruit
Expanding cervical hematoma
Focal neurologic deficit
Neurologic examination incongruous with head CT scan findings
Stroke on secondary CT scan
Risk factors for BCVI
High-energy impact with:
LeForte II or III fracture
Cervical-spine fracture patterns: subluxation, fractures extending into
the transverse foramen, and fractures of C1-C3
Basilar skull fracture with carotid canal involvement
Petrous bone fracture
Diffuse axonal injury with GCS score <6
Near hanging with anoxic brain injury

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The pathogenesis of cerebral infarction after vascular injury involves acute occlusion of the major artery by dissection, and embolization by the thrombus around the dissection.<sup>18)</sup> In our patient, cerebral infarction could be identified as an infarct in the MCA area by MRI. Since there were no infarctions in the PCA and ACA areas, embolization would have occurred at the base of the MCA with the common carotid artery dissection. The main symptom of traumatic carotid artery occlusion is loss of consciousness that occurs after a lucid interval.<sup>8)</sup> Neurological deficits, such as aphasia and hemiplegia, and transient ischemic attack may also occur.<sup>9,19)</sup> Morgan *et al.* found that a lucid interval occurred in 74% of the cases and that the incidence within 24 hours after injury was 77.4%.<sup>20)</sup> In our case, obtundation appeared 6 hours after trauma. Initially, consciousness was normal and we did not perform a head MRI or MRA. In previous reports, onset of cerebral infarction occurred within 24 hours of injury in 80% of the cases. The prognosis is very poor, with a mortality of traumatic carotid artery occlusion of 38.5%, and serious sequelae occur in 50% of the cases.<sup>19,21)</sup>

In this case, we experienced a patient with a cerebral infarction caused by a combination of a lower cervical spinal fracture and traumatic injury to the carotid artery. Since there was no loss of consciousness in the acute phase, we missed the BCVI diagnosis that led to the cerebral infarction in the middle cerebral artery lesion resulting in loss of consciousness after 6 hours. An emergency craniectomy and internal decompression were performed by a neurosurgeon, but unfortunately this did not provide a sufficient decompression effect and was unsuccessful. There were several problems with this case. (1) In spite of the cervical hematoma, we did not perform a Doppler Us or an early CT angiography as a screening test. (2) Although the initial level of consciousness corresponded to a lucid interval, we should have paid closer attention to the possibility of a loss of consciousness. (3) Although there was a major blunt trauma to the neck, a hematoma, and a cervical fracture, we did not suspect BVCV initially. We should have



considered the injury mechanism, injury pattern, and symptoms of CVI in the Denver screening criteria as described above<sup>13)</sup> (Table 1). BCVI is a risk factor for cerebral infarction. BCVI leads to a high incidence of cerebral infarction, with 41% and 16% observed respectively in injuries to the carotid and vertebral arteries.<sup>9)</sup> This case demonstrates that cervical vascular injury should be considered in a patient with blunt neck trauma and that additional imaging (Doppler Us, CT angiography, MRA if possibly) should be performed. In addition, if BCVI were identified early, then antithrombotic treatment –Heparin (PTT 40–50 sec) or antiplatelet therapy would be desirable. If the injury is surgically accessible depending on the degree of damage, bypass surgery and endovascular treatment with a stent should be performed.<sup>1,9)</sup> We predict that if immediate action and aggressive measures were taken, it might have prevented a fatal outcome.

### CONFLICT OF INTEREST

None of the authors have a conflict of interest regarding the contents of the manuscript.

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