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Distal Embolic Brain Infarction Due to Recanalization of Asymptomatic Vertebral Artery Occlusion Resulting From Cervical Spine Injury: A Case Report

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Abstract

Objective: The purpose of this case report is to describe a patient with brain infarction due to recanalization of an occluded vertebral artery (VA) following closed reduction and open fixation of cervical spinal dislocation and to discuss the management of asymptomatic VA injuries associated with spine trauma.

Clinical Features: A 41-year-old Asian man experienced a C4-5 distractive-flexion injury manifesting with quadriplegia and anesthesia below the C3 cord level (including phrenic nerve paralysis), and bowel and bladder dysfunction. Magnetic resonance angiography and computed tomography angiography showed left extracranial VA (V2) occlusion and a patent contralateral VA.

Intervention and Outcome: The patient was observed without antiplatelet and/or anticoagulation therapy and underwent open reduction and internal fusion of C4/5 and tracheostomy 8 hours after the injury. After surgery, supraspinal symptoms such as left horizontal nystagmus and left homonymous hemianopsia led to cranial computed tomography and magnetic resonance imaging, which showed left-side cerebellar infarction in the posterior inferior cerebellar artery territory and right-side posterior cerebral artery infarction. Magnetic resonance angiography and computed tomography angiography demonstrated patent bilateral VA (but hypoplastic right VA) and occluded right posterior cerebral artery. The injured VA was treated conservatively, and there were no other ischemic complications.

Conclusion: The management of asymptomatic VA injury is controversial, with several treatment options available, including observation alone, antiplatelet therapy, anticoagulation therapy, or invasive intervention. Although there are some reports described where management with observation alone seems safe, serious attention should be given to the VA injury caused by cervical spine trauma.

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Introduction

Various nonpenetrating injuries, as well as penetrating injuries, have been associated with cerebrovascular injuries, not excepting cervical spine fracture and dislocation. Compared with carotid arterial injuries, vertebral artery (VA) injuries associated with blunt cervical spine trauma were thought to be infrequent because most of them were asymptomatic and because imaging studies were not performed routinely. Recent improvements in the imaging technology and increased use of screening protocols have led to a greater number of these injuries being identified.[1] Traumatic VA injury can have disastrous consequences of basilar territory infarction and death.[2] Although it is often assumed that reduction of a fracture without treatment of an associated asymptomatic VA injury is safe,[3] when vertebrobasilar ischemia occurs, the mortality rate is 75% to 86%.[4] Vertebral artery injuries are relatively frequent and may be associated with significant morbidity and mortality in patients with cervical spine fractures or dislocations.[2] Foremost in dealing with such an unusual but potentially devastating injury is determining whether the injury can be detected and effectively treated before complications occur.

Despite such a situation, well-defined treatment recommendations are still lacking.[1]

The frequency of vertebrobasilar ischemia in patients with cervical spine trauma is reported as low in many published papers, but there have been some case reports describing cervical spine injury associated with blunt VA injury. Many aspects of the management of VA injuries remain controversial, including the screening criteria, the diagnostic modality, and the optimal treatment for various lesions.

To contribute to further discussion of this topic, we present a case of brain infarction due to recanalization of the occluded VA following closed reduction and open fixation of cervical spinal dislocation, and discuss the management of asymptomatic VA injuries associated with spine trauma.

Case Report

A 41-year-old right-handed Asian man was attacked from behind by a cow and fell down. He received hyperflexion injury when he was struck on the top of the head. He did not lose consciousness but described immediate loss of power and sensation in both his arms and legs. He received emergency transport, was immobilized, and was transferred to the local hospital, where cervical spine trauma was suspected. There were no findings suggesting brain infarction in cranial computed tomography (CT). Unavailability of spine specialists prompted the patient's transfer to our institution, where he arrived 6 hours after injury.

Clinical assessment in the hospital found a Glasgow Coma Scale of 15 of 15 but complete neurologic deficits below the level of C5. Plain film radiographs obtained at this time showed a C4 on C5 dislocation (Fig 1). Further information regarding the severity of the injury was required, and so a cervical CT scan and magnetic resonance imaging were performed.

Computed tomographic scan showed a bifacet dislocation with more than 50% displacement of C4 on C5, and the magnetic resonance imaging revealed severely compressed dural tube at C4-C5 level and high signal intensity in the spinal cord on T2-weighted imaging (Fig 2). A sagittal CT showed bilateral dislocation of facet joints. Magnetic resonance angiography (MRA) showed complete occlusion of the right VA.

The patient was taken to the operating room 100 minutes after arrival. We performed closed reduction and posterior open surgery under general anesthesia. Pedicle screws on the left side and spinous process wiring were used for fixation, and local bone was grafted around C4-C5 facet joints bilaterally. Plain film radiographs obtained just after the surgery showed good fixation (Fig 3).

The patient demonstrated consciousness deterioration associated with repeated vomiting and left hemianopsia. These symptoms led to brain examination and studies. There were low dense lesions in the right posterior lobe and left cerebellum (Fig 4). Magnetic resonance angiography showed occlusion of right posterior cerebral artery (P2) and recanalization of occluded left VA (Fig 5). This seemed to be due to distal embolization of the clot around the occluded vessel. We did not administrate tissue plasminogen activator because the time of onset was unknown and because of the recent cervical spine surgery. Left hemianopsia had remained, although there was no change in his cervical spinal cord dysfunction.

Informed consent was obtained from the patient prior to publication of this case report and accompanying images. The document was completed by his wife because the patient could not sign it himself because of severe tetraplegia. The protocols for human procedures used in this study were approved by the ethics committee of our institution.