CASE REPORT

Carotid Artery Injury with Cerebral Infarction Following Head and Neck Blunt Trauma: Report of a Case

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Blunt injury to the carotid artery is rare but may produce a devastating outcome with long-term morbidity. Initial recognition by clinicians is often difficult because of the diverse clinical manifestations, the delay in presentation of symptoms, and the associated multi-organ system injuries that accompany carotid injury. Early diagnosis and successful management of traumatic carotid artery injury require a high index of clinical suspicion. We report herein a 20-year-old male victim of internal carotid artery injury induced by a motorcycle accident who initially presented with a clear consciousness and had normal computed tomogram (CT) of brain. Two days after injury, the patient suffered from left hemiplegia and coma. The follow-up brain CT showed acute infarction of right cerebrum and severe cerebral edema. Emergency craniotomy for brain decompression and anticoagulation therapy was carried out. After a three-month treatment, he was discharged and underwent regular follow-up in the outpatient department. Six months later, the patient had intact awareness but remained in a left-sided hemiparetic state.

INTRODUCTION

Blunt carotid injuries are uncommon, often occult, and potentially devastating [1-3]. Early series had a combined mortality rate of 5 to 43 percent and combined neurological sequelae rate of 37 to 70 percent [4, 5]. Blunt carotid injuries have been diagnosed in up to 1 percent of blunt trauma patients using angiographic screening programs [1, 3-5].

CASE REPORT

A 20-year-old male motorcyclist wearing a helmet was injured in a road accident. The emergency medical technicians on the scene of the accident reported that he was awake, with no respiratory distress, and was hemodynamically stable. Although no details regarding the situation at the scene were known, there was immediate loss of consciousness for about five minutes after the accident. On arrival at the emergency room, he was conscious, lucid, and complaining of pain in his chin. The physical examination at the ER revealed an ill-looking appearance and full consciousness (Glasgow Coma Score = 15). Vital signs included a blood pressure of 140/86 mmHg, pulse rate of 92 beats
per minute, respiratory rate of 18 breaths per minute, and body temperature of 36°C. There was no evidence of jugular venous engorgement, tracheal deviation, palpable mass of neck, or carotid bruits. The patient had a three centimeters facial laceration on the left chin and had abrasions on right neck, left elbow, and both hands. His left upper limb was weak (4/5 on the Medical Research Council [MRC]† scale). No sensory deficit of left upper limb was noted. The other extremities were at full motor strength (5/5 on the MRC scale) and the sensory examination was intact. Deep tendon reflexes of all four extremities were normal and symmetrical. The pulses were strong and symmetric in the upper and lower extremities bilaterally.

Chest radiography showed a normal cardiac silhouette, clear lung fields, and no fracture. Radiography of cervical spine and pelvis were normal. Skull radiography showed mandibular fracture, which occurred in the right angle and left parasymphysis of the mandible (Figure 1). The brain CT scan was normal (Figure 2A). Cervical cord injury or cervical radiculopathy could not be ruled out, and magnetic resonance image (MRI) of the cervical spine was requested immediately. The cervical spine MRI was interpreted as normal.

After one hour, the left upper limb weakness subsided, and his muscle strength was fully recovered. The left upper limb weakness was attributed to the

Figure 1. The skull radiography showed left-sided mandibular fracture (arrow).

Figures 2A and 2B. 2A (left): The initial brain CT showed no abnormal findings. The brain CT two days later showed a large area of acute tissue edema of the right cerebrum, a compressed right lateral ventricle, and shifting toward the left side. 2B (right): Right middle cerebral artery infarction was impressed (2B).
pain of shoulder trauma. He was admitted for further observation. The next day, the left mandibular parasymphysis fracture was immobilized with an 8-hole plate through the intraoral approach, and arch bars fixation was performed. The surgical procedure took four hours. Twenty-four hours after the operation, the patient became deeply comatose with paralysis of the left extremities. A brain CT was performed again and showed a large area of acute edema of the right cerebrum, a compressed right lateral ventricle, and shifting toward the left side (Figure 2B). A diagnosis of right middle cerebral artery infarction was made. An emergency CT of neck was requested after the brain CT was completed. The neck CT showed total occlusion of the right internal carotid artery above the level of the third cervical spine (Figure 3), which confirmed the diagnosis of right internal carotid artery occlusion with brain infarction.

Emergency craniotomy for brain decompression and post-operative anticoagulation therapy for thrombus prevention were instituted. The anticoagulation therapy administered was a low molecular weight heparin to maintain an APTT of one-and-a-half to two times control. The patient's neurologic state improved gradually, and physical and occupational therapies were instituted. After a three-month treatment, he was discharged and underwent regular follow-up in the outpatient department. Six months later, the patient maintained intact awareness but remained in a left-sided hemiparetic state.

**DISCUSSION**

Blunt carotid artery injury leading to thrombosis and neurologic impairment historically has been considered an uncommon injury [1-3]. With an increased index of suspicion and lower threshold for performing diagnostic arteriograms after blunt neck trauma, recent studies had indicated that carotid artery thrombosis had an incidence up to 1 percent [1, 3-5]. Symptoms associated with blunt carotid injury include headache, visual changes, vertigo, dysphasia, seizures, amnesia, syncope, hemiparesis, obtundation, and Horner's syndrome [6]. Blunt carotid injury is rarely diagnosed at the time of initial presentation as symptoms classically do not appear until 12 to 24 hours after injury [4, 7, 8]. Neurologic symptoms may develop immediately or they may be delayed for several weeks. Neurologic deficits had been noted to develop as many as 15 years after injury [8].

Blunt carotid artery injuries are typically caused by rapid deceleration with hyperextension and rotation of the neck. Five mechanisms of injury have been described: hyperextension with compression of the artery against the transverse process of the cervical spine; hyperflexion with compression of the artery between the mandible and the spine; direct blows; intraoral trauma; and basal skull fracture causing tearing of the intracranial portion of the carotid artery [9]. This stretches the internal carotid artery over the transverse process of the upper cervical vertebrae,
resulting in intimal laceration and subsequent thrombosis [5]. Complete carotid artery transection has been reported [5, 7]. Two different lesions may occur following trauma. A pseudoaneurysm or vessel wall dissection might cause secondary thrombosis resulting in distal emboli or occlusion [10, 11]. The carotid bifurcation and the first two centimeters of the extracranial internal carotid artery were involved in 85 percent of cases [8].

Clinical findings associated with closed carotid injury include neck hematomas, bruits, pulse deficits, ipsilateral Horner's syndrome, transient ischemic attacks, and contralateral motor or sensory deficits. A number of factors have been found to be associated with the presence of blunt carotid artery injury, including motor vehicle or motorcycle accidents, falls, and assault [12]. Blunt carotid artery injury is associated with reported mortality rates of 5 to 43 percent and permanent neurological deficits in 37 to 70 percent of patients [4, 5]. To reduce the associated morbidity and mortality, carotid artery thrombosis must be considered in any trauma patient with acute mental status deterioration or any focal neurologic signs.

Fabian et al. [5] suggested four circumstances that should prompt the immediate investigation of possible blunt carotid artery injury: physical signs of soft tissue injury to the neck; a neurologic examination that is incompatible with the CT findings; development of a neurologic deficit after hospital admission; and presence of Horner's syndrome. We think that in our case, the mechanism of injury may be due to a direct blow to the left mandible, which forcefully hyperextended and rotated his head to the right or to direct blow of the right neck. A high index of suspicion is necessary to avoid delay in initiating appropriate treatment.

Moreover, clinical manifestations of this condition are thought to be influenced by the extent of injury and the available collateral circulation. Temporary occlusion or minor injury to the carotid artery may lead to transient brain ischemia, which is the most common result of vascular injury. Consequently, the lucid interval probably occurred before the progression of intimal dissection was established or before distal embolization took place.

This patient had a left chin laceration, mandibular fracture, right neck abrasion, and left upper limb transient weakness, but no palpable mass of neck and carotid bruit. The initial brain CT and MRI of the cervical spine were normal. The symptoms might have been due to transient cerebral ischemia. The image studies failed to show abnormalities that could explain the evolving neurological deficits on the basis of direct trauma. Although there were no carotid bruits or neck mass, other evidence implicated that the patient might have internal carotid injury.

Although duplex ultrasound scanning, CT angiography, and magnetic resonance angiography had been suggested as having a role in the diagnostic evaluation of this injury, conventional angiography remains the gold standard for diagnosis of blunt carotid artery injury [2-7, 12-14]. Evaluation was usually prompted by mental status changes or development of focal neurologic defects that do not correlate with brain CT findings. CT of the brain has been found to be normal in 39 to 85 percent of patients with blunt carotid artery injury [4, 5, 7, 11, 14].

The first step in diagnosis was brain CT to eliminate closed-head injury as the cause of symptoms. This should be followed by angiographic evaluation of the carotid arteries. Once the diagnosis of carotid injury was confirmed, the first line of treatment is anticoagulation. Medical management has been shown to provide good results in patients with minimal neurologic deficits. Patients with severe neurologic deficits on initial examination may benefit from surgical intervention.

The recommended treatment for carotid artery thrombosis has evolved over the past decade. Whereas operative inter-
vention was originally advocated, recent large-scale studies have suggested that heparin anticoagulation was independently associated with improved survival and neurological outcome and an acceptable incidence of complications [4, 5, 7, 12, 15, 16]. The rationale for post-operative systemic anticoagulation is to minimize clot formation at the site of intimal injury, decrease further propagation of the clot that has formed, and prevent embolization of the clot from any pseudoaneurysm sac [5].

High suspicion of injury, coupled with early diagnosis and treatment of carotid injuries, improves outcome. Injuries are often missed because there was a delay in the onset of signs and symptoms and when these signs do develop, they were often attributed to an associated head injury rather than a vascular injury. Failure to make precise diagnosis of carotid artery dissection and to institute appropriate therapy may result in long-term neurologic sequelae or death, although spontaneous resolution of the condition does sometimes occur.

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