Traumatic Bilateral Carotid Artery Dissection

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Introduction: Bilateral carotid artery dissection following blunt trauma is a rare but potentially lethal injury if not diagnosed early and treated.

Case report: We report a collision patient who suffered bilateral asymptomatic carotid artery dissections. He also had multiple fractures of the pelvis and upper and lower extremities. The patient was managed acutely with aspirin. He underwent open reduction and internal fixation of fractures without complication and was discharged home on aspirin.

Discussion: Carotid artery dissections are increasingly being recognized in blunt trauma patients. Management options include systemic anticoagulation versus antiplatelet therapy. The need to acutely address the carotid artery injury must be balanced with the need for hemostasis with associated solid organ injury and/or future operative procedures.

Conclusions: Carotid artery dissections should be screened for in patients with appropriate mechanism of injury. This case report details the successful management of a patient of bilateral carotid artery dissection and other major orthopedic injuries using aspirin.

INTRODUCTION

Carotid artery dissection following blunt injury is a rare but significant cause of morbidity and mortality. Only 0.08% to 0.67% of patients admitted to the hospital after road traffic accidents have blunt carotid injury.1,2 Bilateral carotid artery dissection is an even more infrequent injury with a high mortality. Of the less than 1% of patients with blunt carotid artery injury the incidence of bilateral carotid artery dissections is between 20%-80%. We report a motorcycle collision patient with bilateral carotid artery dissection and major orthopedic trauma who was successfully managed with oral aspirin.

CASE REPORT

A 24-year-old man was involved in a motorcycle collision with a truck. He was ejected approximately 65 feet with an estimated speed of 50 mph. Emergency medical technicians were unable to obtain a blood pressure or heart rate initially in the field. There was blood in the nares and trachea and an airway could not be obtained in the field. He was transferred to an outside hospital for emergency airway management. On initial examination the patient was in severe respiratory distress with laboured breathing, obvious multiple fractures of upper and lower extremities. Neurologic exam did not reveal any focal deficits. Patient was intubated at an outside hospital. The Glasgow Coma Score (GCS) was 6T at the time of initial assessment. He was transferred to our trauma center for definitive management of his injuries.

On arrival at Louisiana State University Health Sciences Center (LSUHSC)-Shreveport Trauma Center his vital signs were a systolic blood pressure of 110mmHg, and a heart rate of 130 beats per minute. He was intubated with a GCS of 6T. The saturation of peripheral oxygen (Spo₂) was 90% on a fraction of inspired oxygen (Fio₂) of 40%. Other injuries included an open-book pelvis, left ankle fracture, and a right forearm fracture. His injury severity score (ISS) was 43. A trauma panel of laboratory examinations was completed. The patient had severe respiratory acidosis and hemoglobin of 12.5 grams after transfusion of four units of packed red blood cells (PRBC). He also had gross hematuria. Chest radiograph revealed bilateral pulmonary contusions. Pelvic radiograph showed widening of the pubic symphysis by 5cm. Computerized tomography of the chest revealed bilateral pulmonary contusions and a right-sided hemothorax. Computed tomography of the head showed fluid in the right maxillary sinus. Computed tomographic (CT) angiography of the neck showed tortuous carotid arteries with a linear hypodense signal traversing each internal carotid artery at the level of the foramen...
magnum consistent with bilateral internal carotid artery dissections (Figures 1, 2). CT angiography of the lower extremity was negative for vascular injury.

The patient was managed in the surgical intensive care unit (SICU) for respiratory failure. Orthopedic surgical consultation was placed for the open tibial, fibular, distal radius, and open-book pelvic fractures. Hourly neurochecks were done in the SICU. Neurosurgical consultation confirmed that the patient had asymptomatic dissections and was neurologically stable, and no acute neurosurgical intervention was deemed necessary.

Conservative management of his bilateral carotid dissections was planned. Vascular surgery recommended aspirin 325 mg once daily by mouth. Systemic anticoagulation with heparin was not possible due to significant orthopedic fractures and the need for operative management.

The patient underwent surgical repair of his numerous fractures. He was weaned off the ventilator and recovered well. Neurologically he remained completely asymptomatic as regards to the carotid dissections, and repeat CT angiography showed stable dissections with widely patent internal and external carotid arteries. He was discharged home on 325 mg of aspirin daily.

He is being followed up as an outpatient and has had no neurological events.

DISCUSSION

We present here a case report of a young patient with bilateral carotid artery dissection following blunt trauma that was managed with conservative therapy with aspirin with a positive outcome. Patients like ours with concomitant severe orthopedic trauma are generally unable to undergo therapeutic anticoagulation with heparin in the acute phase. There is no conclusive data that systemic anticoagulation is superior to aspirin in patients with carotid artery dissections. Aspirin may be effective in such patients for its antiplatelet effects.

The classic mechanism of extension and rotation of the neck during blunt trauma must always be recognized as a potential risk factor for blunt carotid injuries. Cases of distracting injuries such as orthopedic trauma patients with appropriate mechanisms should be screened for blunt carotid injury. Management of carotid artery injuries in this patient population is a challenge. There are few reported cases of asymptomatic bilateral carotid artery dissections in patients with contraindications to systemic anticoagulation.

Our patient had a traumatic dissection of both internal carotid arteries (ICA) which is an uncommon but well-recognized entity. In fact, it is hypothesized that Alexander the Great had a traumatic carotid artery dissection. Carotid artery injuries comprise 5% of arterial trauma, and while most of them occur with penetrating trauma, carotid injuries are increasingly being recognized as a complication of blunt trauma.

Carotid artery injuries have been described in varied injury mechanisms. Large case series have described cases occurring after high velocity motor vehicle injuries like
accidents involving all terrain vehicles (ATV) and motorcycle collisions. Car accidents including backlash cases from rear ending have resulted in bilateral carotid artery dissections. They also have been described in “exotic” cases, such as bungee jumping, scuba diving, snow and water skiing, and in accidents involving the hyperextension-hyper flexion mechanism of injury. Cases where carotid artery dissections have occurred after trivial trauma, like undergoing dental work, coughing, and chiropractor manipulations also have been described.

Biffi et al studied the association between various injury mechanisms and patterns and blunt cerebral-vascular injuries. Risk factors for bilateral carotid artery dissections in blunt injury include high energy transfer mechanism, basilar skull fracture, diffuse axonal injury with a GCS of six or lower, and cervical spine fracture or near hanging. Our patient had two of these risk factors which led to aggressive screening and early diagnosis.

Our patient remained neurologically intact. The presentation of cerebral ischemia is variable in bilateral internal carotid artery dissections (ICAD) although previous studies suggest that generally only one of the dissected arteries produces ischemic symptoms. Ipsilateral facial pain and a partial Horner’s Syndrome (ptosis with miosis) are the most frequent clinical manifestations. Other patients may present with a transient or persistent ischemic event. The pathophysiology of strokes is embolization from the dissected artery to the intracranial arteries most often the middle cerebral artery. Thus, the emergency physician must consider such dissections in younger patients with sudden neurologic deficits and absent or minimal risk factors. There may be a delay in presentation but the mean time for symptom presentation is generally within the first 24-72 hours.

The diagnosis is made via CT angiography and magnetic resonance angiography. If neither modality is available carotid duplex scanning may be useful in diagnosing a carotid artery dissection. Angiography remains the gold standard for screening patients at risk but is an invasive procedure which was associated with a puncture hematoma rate of 0.3% and a 0.1% stroke rate in a large study.

The management of carotid artery dissection is controversial. The treatment of extracranial carotid artery dissection with anticoagulation has been proven to be effective, with an approximate 50%–70% arterial recanalization rate and a 10% risk of late neurologic deficits. In the study by Biffi et al, the rate of ischemic events in the anticoagulation group was zero versus 46% in the untreated group. Recently, endovascular stent placement is gaining favor due to a low risk of complications and excellent recovery rates. However, the long term effects of this modality are still unknown.

There have been no randomized therapeutic trials comparing systemic anticoagulation with antiplatelet agents, but two meta-analysis and one non-randomized study have shown no difference in outcomes among patients treated with the two modalities. In our patient, the apparent benefit of early heparinization had to be balanced against the risks of significant bleeding from his multiple open fractures. Thus a conservative approach was deemed best, especially since the patient had no neurological signs of the dissection.

According to Broomberg et al, the optimum modality for the treatment of BCVI is as yet undetermined. Prospective studies will be necessary to compare invasive intervention versus anticoagulation. Furthermore the optimal anticoagulation regimen is as yet unknown in terms of agent (anti-platelet agents, heparinoid, or warfarin) as well as the duration and endpoint of therapy. Clearly there is room for further study in this regard. In light of the relative rarity of the disease entity, systematic, multi-institutional studies will be required to answer this question.

REFERENCES


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