Transient Paraplegia in a Patient with Bilateral Posterior Frontal Contusions and Traumatic Thoracic Aortic Dissection

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Figure 1

Magnetic resonance imaging of the brain demonstrating bilateral parasagittal contusions. (A) T2-weighted sequence, axial cut; (B) T2-weighted sequence, coronal cut.

ABSTRACT

Background: In the setting of multi-system traumas, the etiology and pathophysiology of neurologic injuries can be difficult to identify.

Methods: A unique case of a pedestrian struck by a motor vehicle that presented with acute paraplegia after an endovascular stent placement for a traumatic thoracic aorta dissection. The patient had no significant motor function in the lower extremities, but full preservation of all sensory modalities. Initial admission computed tomography (CT) imaging was negative for intracranial trauma, but noted an acute cranial fracture; no spinal trauma was identified.

Results: The patient had a lumbar drain placed to maximize spinal perfusion pressures and was immediately evaluated with magnetic resonance imaging (MRI) of the neural axis. Acute bilateral posterior frontal contusions were identified on brain imaging, which were not present on pre-procedural CT head. No spinal cord injury or ischemia was seen on spinal imaging. The patient recovered and regained use of his lower extremities following a short rehab stay.

Conclusion: In the setting of multi-system trauma, a high level of suspicion should exist for alternative etiologies of neurologic injuries. Thorough neurologic examinations and imaging assessments of the nervous system should be conducted to avoid misdiagnosis and improper management of occult injuries. This is the first reported case of acute paraplegia due to vertex trauma which may be a rare mechanism of injury and/or under-recognized.

INTRODUCTION

Trauma is the leading cause of mortality for patients less than forty years of agecheck. Further multi-system trauma has been associated with increased mortality, especially when it involves vascular injuries.⁴ Therefore, prompt diagnosis and management is crucial and increases the probability of survival and a favorable outcome.

Acute paraplegia is an associated complication of traumatic thoracic spinal cord and aortic injuries. Further, it can also result from open and endovascular repair of this injury due to decreased perfusion to the cord and ensuing ischemia and infarction.^{4,5,14} Few cases of acute lower extremity monoparesis have been reported from traumatic injury to the frontal lobe.^{2,10} We present the first case of acute paraplegia resulting from bilateral para-sagittal frontal contusions in a patient with concomitant thoracic aorta injury.

CASE REPORT

A 31 year-old male pedestrian was struck by a high velocity vehicle and thrown approximately seventy feet from the initial site of impact. The patient was intubated at the scene and transported to an outside hospital. At the time he was following commands and moving all extremities. CT of the brain revealed a bicoronal scalp laceration and underlying skull fracture with no intracranial hemorrhage or contusion; CT of the chest, abdomen and pelvis revealed an aortic isthmus tear, bilateral pneumo-hemothoraces and pulmonary contusions, multiple ribs and right clavicle fractures, liver laceration with intraperitoneal hemorrhage.

Upon transfer to our facility, the patient remained stable. He would open his eyes to voice, pupils were equal round and reactive, he would follow simple commands in all four extremities with some efforts against gravity. The patient was urgently taken to the operating room for endoscopic repair of the thoracic aortic tear. The procedure was completed without complication, with placement of a Gore TAG 26mm x 10cm endoprosthesis. The patient remained intubated and neurologic

Figure 2

Non-contrast computed tomography of the brain revealing acute bilateral high convexity posterior frontal contusion signal abnormality.

Figure 3

Three month follow-up non-contrast computed tomography of the brain showing resolution of hemorrhagic frontal contusion and residual encephalomalacia.

examination revealed his upper extremities were 5/5 strength in all tested muscle groups. In the lower extremities he had a questionable right ilio-psoas voluntary muscle twitch; he was otherwise 0/5 in all other muscle groups. Sensory exam revealed normal pinprick, light touch and proprioception. The patient had a good rectal tone but no volition.

Due to a concern for post-endovascular repair cord ischemia and infarction, a lumbar drain was inserted to maintain low CSF pressure and maximize spinal perfusion pressures. The patient was evaluated with an MRI of the brain and entire spine. It revealed bilateral high convexity posterior frontal parasagittal contusions underlying the area of scalp laceration (Figures 1 and 2), and a normal cervical, thoracic and lumbar spinal cord and column.

The patient had no further surgical intervention and was discharged to an acute rehab after a couple of weeks of recovery. At his 3 months follow-up, the patient had an almost complete recovery of lower extremity motor strength and was ambulating independently. Follow-up CT head revealed evolution of the contusion with complete resorption of blood products and persistent of edema and encephalomalacia (Figure 3).

DISCUSSION

Paraplegia can result from a disruption at any level of the neural motor pathway starting from the frontal parasagittal cortical neurons to the thoraco-lumbar spinal cord upper motor neurons, the peripheral lumbar spinal cord roots and peripheral nerves. Post-traumatic acute paraplegia most commonly results from

a thoracic spinal cord column fracture and spinal cord injury or vascular injury to the thoracic aorta. Aortic trauma accounts for less than 0.5% of all trauma admissions, but it is the second leading cause of trauma-related death, second to head trauma.⁴ Up to 90% of patients suffering blunt aortic injury (BAI) expire before reaching a hospital; and of those that do, another half dies within 24 hours.^{13,8,11,9} BAI also carries a significant morbidity, including spinal cord ischemia and stroke reported as high as 20% and 8% respectively.⁵ Spinal cord ischemia can be the direct result of aortic injury or can be the complication of open and endovascular repairs of the aortic injury. This is thought to be the result of decreased or insufficient blood flow to a susceptible area of the thoracic spinal cord. Endovascular repairs have been shown to have a significantly lower incidence of paraplegia, by avoiding aortic cross-clamping (results in increased CSF pressure and decreased distal aortic perfusion).¹⁴

This patient presented with an isthmic aortic tear and underwent an emergent endovascular repair. Post-operatively, his paraplegia was initially postulated to result from a spinal cord ischemia due to loss of spinal cord perfusion. A lumbar drain was placed to maintain a low intra-spinal CSF pressure and improve cord perfusion. The patient did not have any improvement from the lumbar drainage. Further assessment with an MRI failed to show any evidence of spinal cord ischemia or stroke, but did note the bilateral cerebral contusions in the primary motor cortex (Figures 1 and 2). It is postulated that the direct trauma to the patient's skull and brain parenchyma

manifested as a skull fracture was the etiology of the neurologic deterioration. As part of the endovascular procedure, the patient was anticoagulated with heparin. Therefore we suspect a hemorrhagic conversion or evolution of previously small cerebral contusions resulted in his lower extremity weakness.^{1,7} Venous infarction from superior sagittal sinus thrombosis was excluded by a negative brain MRI.

Paraplegia from cerebral contusion is extremely rare, and to the best of our knowledge has not been previously reported. This rarity of literature might reflect the fact that only ~4% of all cortical contusions occur in the superior fronto-parietal cortex^{3,6}; accordingly fewer would happen in the specific para-sagittal cortex responsible for lower extremity movement. Atac et al.² reported a case of left foot drop secondary to a cerebral contusion from a gunshot wound. Lega et al.¹⁰ also reported two cases of lower extremity monoparesis due to traumatic intraparenchymal contusions. They made the point that conventional axial CT imaging does not always provide adequate imaging of the vertex, primarily because of volume averaging. In this case one may speculate that our patient's initial CT failed to detect small vertex contusions. Our recommendation is for a high level of suspicion in patients with external signs of cranial traumas and improved screening with high resolution axial CT and coronal reconstruction.

CONCLUSION

In the setting of multi-system trauma, a high level of suspicion should exist for multivariable injuries. Acute paraplegia due to vertex trauma is a very rare injury but should be included in

the differential for a patient with bilateral lower extremity weakness and negative MRI of the spinal axis.

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