Traumatic Subclavian Pseudoaneurysm Resulting in an Internal Jugular Vein–Vertebral Artery Fistula with Vascular Steal Phenomenon

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Introduction

Vertebral arteriovenous (AV) fistulas have been described in the literature secondary to penetrating trauma, or more commonly, iatrogenic injury.[1] However, an arteriovenous fistula between the vertebral artery and the internal jugular vein as a sequela of subclavian artery pseudoaneurysm has not yet been reported. In this case report, we describe the development of a vertebrojugular fistula secondary to traumatic subclavian artery pseudoaneurysm in a gunshot wound victim that led to vascular steal phenomenon with bilateral posterior cerebral circulation infarcts. The patient developed symptomatic autonomic dysfunction due to posterior fossa ischemia.

Case Presentation

A 15-year-old male presented to an outside hospital in pulseless electrical activity (PEA) after sustaining gunshot wounds to the right anterior neck, left flank, and left upper back. He was resuscitated and taken to the operating room (OR) for an emergency exploratory laparotomy with a splenectomy and graham patch placement. He was subsequently transferred to the level I trauma center, where he arrived intubated and with an open abdomen. At presentation, he had spontaneous movement of the bilateral upper extremities but not the bilateral lower extremities and a Glasgow Coma Score (GCS) of 6 . CT angiogram (CTA) demonstrated a complex pseudoaneurysm originating at the junction of the right proximal subclavian artery and right vertebral artery (VA) with extension to the distal right internal jugular vein (IJV) as a traumatic arteriovenous (AV) fistula. **(Figure 1)**

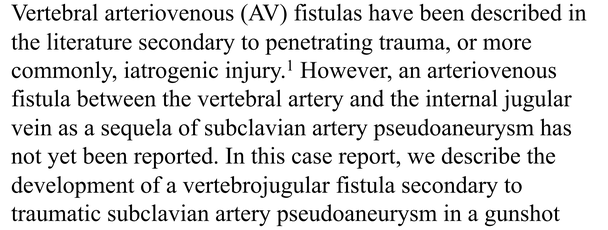

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Figure 1:
Computed tomography angiogram (CTA) of the head and neck showing a complex pseudoaneurysm originating at the proximal right subclavian artery Coronal (a). Sagittal (b) and axial (c) images showing extension into the distal right internal jugular vein (IJV).

He was emergently taken to the OR with Vascular for a right subclavian artery stent and he was admitted to the intensive care unit (ICU). CTA the following day showed a patent subclavian stent covering the origin of the right VA with filling of the traumatic AV fistula between retrograde flow from the right VA into the right IJV **(Figure 2)**. Vascular Surgery made plans for repair of the AV fistula within the coming weeks as the fistula was felt to be low flow.

Concomitant traumatic injuries included fractures of the right T1 vertebra through the inferior articular facet, lamina, and spinous process with displaced fragments into the right aspect of the upper thoracic spinal canal, fracture of the right T2 superior articular facet, rib fractures, grade 1 hepatic laceration, and traumatic injury to the stomach. The patient continued to move his bilateral upper extremities but had absent movement and sensation of the bilateral lower extremities, consistent with his initial presentation. Orthopedic Spine Surgery was consulted to evaluate the upper thoracic vertebral fractures, who felt that the lower extremity neurological deficits were likely secondary to thermal injury from the gunshot wound rather than spinal cord disruption from his fractures and no acute intervention was warranted.

On day five of admission, he had an acute change in neurological status with loss of motor in his bilateral upper extremities. He also had an episode of bradycardia with heart rate in the 30s during which he became unresponsive; heart rate and responsiveness resolved with atropine. He was sent for an emergent CT of the head and neck, which showed the development of bilateral cerebellar infarcts with posterior fossa mass effect along with new obstructive hydrocephalus **(Figure 3).**

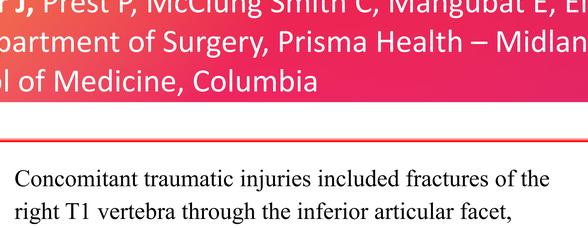

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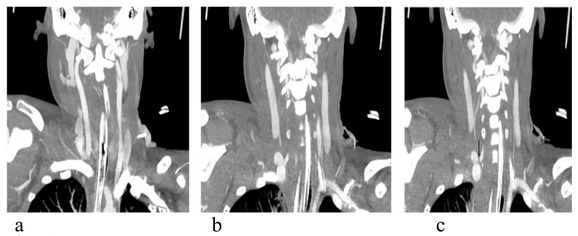
Figure 3:
Computed tomography (CT) head showing significantly increased size of lateral and third ventricles (a and b). Cerebellar hypodensities (b) with posterior fossa mass effect (c) and complete effacement of the distal fourth ventricle (d).



a b c

Figure 2:
Computed tomography angiogram (CTA) showing a stent in the right subclavian artery covering the previously damaged area at the vertebral artery (VA) takeoff (a). Persistent filling of pseudoaneurysm in the right supraclavicular region is seen (b) and an arteriovenous (AV) fistula between the right VA and right internal jugular vein (IJV) is visualized (c).

An external ventricular drain (EVD) was placed emergently at the bedside. Follow-up imaging with a vascular ultrasound of the carotid and vertebral arteries revealed redemonstration of the VA-IJV AV fistula as well as retrograde flow in the right VA.

Suspected etiology for the posterior fossa infarcts and neurological change was hypoperfusion related to the previously described AV fistula. The patient went to Interventional radiology for embolization of the VA-IJV AV fistula **(Figure 4 & 5).**

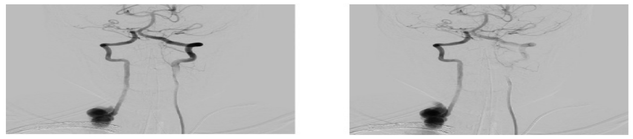
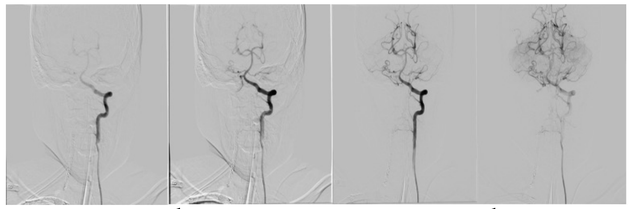


Figure 4:
Initial Fluoroscopy demonstrating right vertebral artery and internal jugular vein traumatic fistula.



a b c d

Figure 5:
Fluoroscopy with coils seen in the right vertebral artery (VA) with no retrograde contrast seen throughout the study (a-d). A patent posterior circulation on the left is redemonstrated with normal perfusion throughout the left VA and filling of the left posterior inferior cerebellar artery (PICA) and bilateral posterior cerebral arteries (PCA) (a). Normal perfusion of the remainder of the posterior circulation is seen as contrast continues superiorly to fill the bilateral anterior inferior cerebellar arteries (AICA) and bilateral superior cerebellar arteries (SCA) (b-d).

Despite the improvements in the posterior circulation blood flow and complete decompression of the ventricles confirmed on repeat imaging, cerebellar hypodensities persisted along with the motor deficit which was concerning for permanent neurological injury. The most likely etiology for the neurological change was felt to be an infarction of the cervical spinal cord and possible infarctions within the brainstem secondary to the steal phenomenon. CPAP trials were initiated however the patient couldn't be weaned off the ventilator. Eventually the bradycardic episodes resolved with atropine and stabilized with theophylline. A diaphragmatic pacer was attempted on day thirty-nine of admission, which was unsuccessful because the diaphragm muscle had atrophied to the point that there was nothing to propagate the signal from the pacer.

He remained hospitalized and his course was complicated with pneumonia, UTIs, and pressure wounds associated with his quadriplegia.

Discussion

Traumatic AV fistulas are common in thermal injuries secondary to bullets. Pseudoaneurysms are also a common injury from penetrating trauma such as high-velocity gunshot wounds. However, it is unusual for these to occur simultaneously following a traumatic injury. It is beneficial to treat these injuries soon after they occur, as the combination of AV fistula and pseudoaneurysm can result in high-output heart failure, embolism, rupture of the pseudoaneurysm, and thrombosis.[2] In this case, the patient was emergently taken to the OR by Vascular Surgery for subclavian artery stent placement. Further endovascular repair of the AV fistula was not performed at this time due to the extent of the patient's concomitant injuries and the significant blood loss that he had endured to that point.

This case is unique in that it is, to our knowledge, the first incidence of a traumatic subclavian pseudoaneurysm leading to an IJV-VA fistula. This case was also exceptional in that it resulted in vascular steal syndrome due to the low-pressure circuit caused by the AV fistula, culminating in posterior circulation infarcts and autonomic dysfunction with frequent episodes of bradycardia and unresponsiveness.

Conclusion

Early management of symptomatic fistulas has the potential to prevent neurological compromise

In cases where the patient is unstable or unable to communicate symptoms, timing of treatment becomes more complicated.

Early treatment may have benefited this patient by maintaining normal cerebral circulation.

References

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