A Case of Paraplegia Following a Transforaminal Epidural Steroid Injection

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Case Diagnosis

82 year old female with T12 ASIA A following a Transforaminal Epidural Steroid Injection.

Introduction

The spinal cord blood supply is mainly from the anterior spinal artery and posterior spinal arteries. These arteries are fed by 8-10 segmental spinal arteries. The segmental arteries split into anterior and posterior branches; the posterior branches divide further into the radiculomedullary artery, the muscular branch, and the dorsal somatic branch. The largest of anterior radiculomedullary artery is the Artery of Adamkiewicz which is the major supplier of the anterior spinal artery. The Artery of Adamkiewicz comes off the left side of the descending aorta between T8 to L2 then makes a hairpin turn and joins with the anterior spinal artery. Lack of flow in the Artery of Adamkiewicz can cause ischemia of the spinal cord.

Since the 1950s epidural steroid injections have been used for pain relief and are indicated for radicular pain, including but not limited to, radicular pain caused by compression fractures of spinal vertebra. Injection of steroids is theorized to interrupt the inflammatory cascade thereby reducing pain. Particulate steroids were once favored because they were believed to be longer lasting. However, recent research has suggested that particulate steroids can aggregate larger than a red blood cell which can cause neurological injury. This is less likely with non-particulate steroids.

References

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Figures

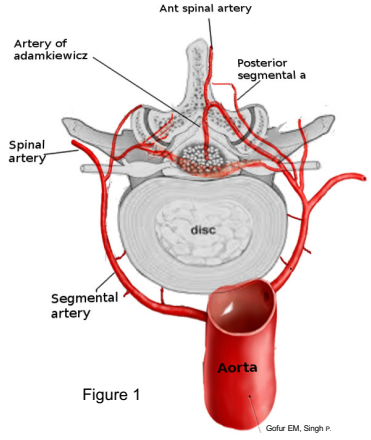

Figure 1

Figure 2

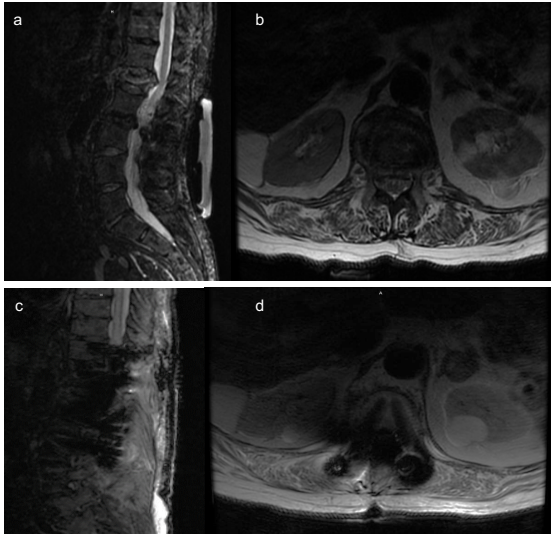

Figure 1: Anatomy of vertebral canal vasculature demonstrating the hairpin turn of the Artery of Adamkiewicz as it joins the anterior spinal artery. Image used with permission from the article entitled "Anatomy, Back, Vertebral Canal Blood Supply" from Gofur et al.
Figure 2: *a:* Sagittal T2 STIR MRI Immediately Post –TESI with High grade L1 compression fracture with retropulsion (similar in appearance to CT completed at time of fall several months prior) and multilevel spinal canal stenosis, with moderate severe canal stenosis noted at L1. Visualized spinal cord and conus medullaris is normal in size and signal without evidence for ischemic changes
b: Axial T2 frFSE MRI of L1 demonstrating compression fracture with retropulsion and severe canal stenosis
c: Sagittal T2 STIR MRI Post Emergent T11-L3 laminectomy limited study due to artifact. conus is not well-visualized. Grossly unremarkable appearance of the visualized cauda equina nerve roots.
d: Axial T2 frFSE MRI Post Emergent T11-L3 laminectomy and fusion demonstrating poorly visualized spinal cord

Case Report

Patient is an 82 year old female with a past medical history of arthritis, coronary artery disease, COVID-19, GERD, hyperlipidemia, and hypertension who underwent an L1-L2 transforaminal fluoroscopy guided epidural steroid injection for persistent low back pain after an L1 burst fracture 5 months prior. Immediately post procedure patient noted loss of sensation and inability to move her lower extremities and was incontinent of bowel and bladder. Initial MRI demonstrated high-grade L1 compression fracture with retropulsion with moderate to severe stenosis similar to prior CT visualized spinal cord and conus medularis were normal without evidence for ischemic changes. Patient underwent emergent T11-L3 laminectomy. On repeat MRI upper lumbar spine was poorly visualized due to significant artifact.

On admission to acute rehabilitation, patient had no muscle tone in the lower extremities and lack of sensation starting at the L1 dermatome. On ASIA exam patient was found to be ASIA A with a T12 neurologic level of injury, indicating complete impairment with no motor or sensory function preserved in the sacral segments of S4-S5. Rehab therapies were focused on truncal control. Patient made improvements with core strength and trunk control but remained without sensation, muscle tone in the lower extremities, and bowel or bladder control. Patient ultimately transferred out to subacute rehab facility for further therapy .

Discussion

Paraplegia post-TESI is very rare. While epidural bleed and hematoma have been described, they do not produce immediate paraplegia. Epidural abscess is also not an acute complication. Possible causes of immediate acute complete spinal cord injury may be explained on the basis of interruption of arterial supply to the spinal cord. Lumbar spinal cord ischemia or infarction is a rare, serious complication of transforaminal epidural steroid injection. Possible mechanisms include inadvertent injury of the artery of Adamkiewicz, or accidental injection and aggregation of steroid into the vasculature blocking blood flow. Some prognosis predictors after spinal cord infarction include severity of impairment at time of incident and age, both of which indicate a poor prognosis in this presented case.

Conclusion

This is a case of a very rare but life-changing and devastating complication of transformational epidural steroid injection resulting in spinal cord infarction.