Posterior Spinal Artery Syndrome following Cervical Transforaminal Epidural Steroid Injection

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INTRODUCTION

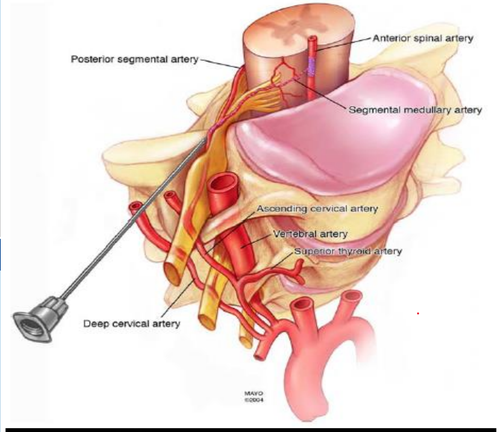
Cervical transforaminal epidural steroid injections (CTFESIs) carry a rare but catastrophic risk of spinal cord infarction, estimated at 1 in 10,000–50,000 procedures. Intra-arterial injection via a radiculomedullary feeder or vertebral artery is a recognized mechanism of embolic or vasospastic cord injury. Non-particulate dexamethasone lowers but does not eliminate this risk. Posterior spinal artery (PSA) infarcts are much less common than anterior spinal artery strokes. We report an extensive C6–T5 PSA infarction with pneumocephalus following CTFESI.

CASE PRESENTATION

A 51-year-old female with no significant past medical history presented for left C6–C7 CTFESI for intractable C7 radiculopathy non-responsive to conservative management. Under fluoroscopic guidance, contrast was injected, demonstrating neural foraminal flow without initial intravascular spread. A total of 10 mg dexamethasone (non-particulate; off-label neuraxial administration) with 1 mL 0.25% bupivacaine was injected. During final needle positioning, the patient reported a sudden left-sided "electric shock" paresthesia, immediate severe headache, and transient dizziness, indicating unintentional dural puncture.

Within 4 hours post-procedure, the patient developed dynamic lower extremity weakness, loss of vibration and proprioception below T2, marked sensory ataxia, and a positive Romberg sign, with intact pain and temperature sensation. Non-contrast head CT confirmed acute pneumocephalus within the anterior cranial fossa and basal cisterns. Cervicothoracic MRI demonstrated extensive longitudinal T2/STIR hyperintensity from C6 to T5 along the posterior columns with restricted diffusion on DWI/ADC maps, indicating acute PSA territory infarction. The patient was admitted to the Neuro-ICU for blood pressure augmentation, strict flat bedrest, and physical therapy. At 6 months post-injury, motor strength recovered to 5/5 bilaterally, with partial proprioceptive recovery allowing ambulation with a single-point cane.

IMAGING



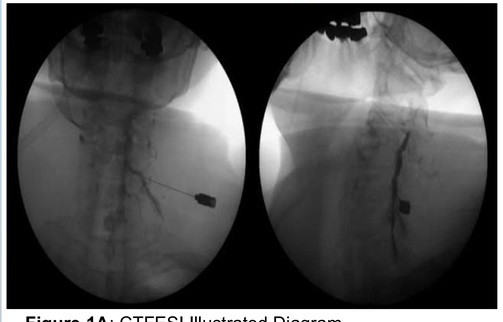


Figure 1A: CTFESI Illustrated Diagram.
Figure 1B: Sample AP/Oblique view of C6 CTFESI under Fluoroscopic Guidance (not the patient's image)



Figure 2: MRI Cervical Spine: T2 Sagittal View: Cord signal abnormality with T2 hyperintensity within the left central cord extending from the C6-C7 level through to the T5 level, likely at least partially involving gray matter.



MATERIALS AND METHODS

This case report contains no identifiable patient information, and is exempt from IRB review per Burke Rehabilitation Hospital policy. Informed consent was obtained from the patient to publish this case report.

DISCUSSION

While non-particulate steroids have widely been seen as lower risk for embolic cord injuries, they do not eliminate ischemic injury from needle-tip trauma, vasospasm, or unrecognized intra-arterial injection despite a negative contrast run. Static fluoroscopic imaging can miss transient intra-arterial placement during needle repositioning. To mitigate this risk, interventionalists should use real-time or digital subtraction angiography, target the posteroinferior foramen, inject incrementally under live imaging, avoid heavy sedation, and maintain a low threshold for urgent neuroimaging when atypical deficits arise.

CONCLUSION

This case demonstrates that posterior spinal artery infarction can complicate CTFESI despite non-particulate steroid use and a negative contrast run. Prompt recognition of atypical neurologic deficits and aggressive supportive care are essential to optimizing recovery from this rare but potentially devastating complication.

REFERENCES

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