Pearls & Oy-sters: Acute spinal cord infarction following endoscopic ultrasound-guided celiac plexus neurolysis

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PEARLS

- Celiac plexus neurolysis (CPN)/celiac plexus block (CPB) is used to treat excruciating cancer-related abdominal pain.1
- CPN is done via injecting 50%–100% alcohol into the celiac plexus, which is located under the diaphragm at the level of T12-L1 adjacent to the abdominal aorta and the take-off of the celiac trunk.2
- Endoscopic ultrasound guided neurolysis (EUS CPN) has been found to have 85% success rate with only 2 cases (0.8%) of major non-neurologic complications reported.3,4

OY-STER

- Major neurologic complications—including spinal cord infarction—are seen in fewer than 1% of patients, more commonly with a posterior approach.

CASE REPORT A 76-year-old man with hypertension and adenocarcinoma of the pancreatic head and hepatic metastasis complained of extreme abdominal pain associated with nausea and vomiting. Drug regimens including hydromorphone 2 mg every 4 hours, oxycodone 15 mg every 12 hours, and acetaminophen and hydrocodone 5/500 mg 6 tablets daily had failed to control his pain. EUS CPN was performed advancing a 22-gauge needle into the celiac ganglia and injecting 5 mL 1:5 mixture of bupivacaine 0.25% with epinephrine and alcohol. The rest of the mixture (19 mL) was injected into the area around the celiac artery. There were no immediate major complications noted. Mean blood pressure dropped from 103 to 71 mm Hg over 13 minutes but returned to 102 mm Hg in 3 minutes after a 750 mL bolus of normal saline. During the procedure, multiple aspirations were negative for blood or CSF.

On arrival at the recovery ward, his cardiovascular and respiratory condition was stable. He was unable to move his legs. Neurologic examination revealed normal tone, strength, and reflexes in the upper extremities. He had flaccid paraplegia. On sensory examination, he had reduced pinprick and temperature below L1 with reduced touch and vibration, but some preserved proprioception. There was no sacral sparing. Tendon reflexes were absent in the legs. Plantar responses could not be elicited. Bladder and anal sphincter control was absent. MRI performed the same day showed a spinal cord infarct extending from T10 to the conus.

The patient underwent lumbar drain placement for CSF diversion therapy. A total of 57 mL CSF was drained over 10 hours with no evidence of increased CSF pressure. The lumbar drain was removed after noticing no improvement. The patient was discharged to a skilled nursing facility after 5 days. At the time of discharge there was no improvement in his weakness or sensations in the legs.

DISCUSSION A considerable number of cases of acute paraplegia from anterior spinal artery infarction following celiac plexus injections have been reported, mostly in the anesthesia literature. Although this complication remains rare, it is pertinent to neurologists who are consulted emergently to evaluate patients with acute paraplegia after this procedure. Our patient developed paraplegia secondary to EUS CPN, a technique widely considered safer than a blind approach. Time of onset of paraplegia in reported cases has varied from periprocedure to 14 hours after the procedure. As expected in an acute cord lesion, patients present with motor weakness, decreased pain and temperature sensation below T7-L1, and detrusor atonia. Some patients may have intact vibration and proprioception as confirmed by somatosensory evoked potentials.5

Anterior spinal cord syndrome secondary to celiac plexus neurolysis may be related to injury of the lumbar artery leading into the artery of Adamkiewicz. This major artery originates from the aorta, varies in position from T7-L4, supplies the lower two-thirds of the anterior spinal artery, and anatomically is closely related to the celiac ganglion (figure, left...
The mechanism of acute spinal cord ischemia after vascular puncture may involve vasospasm due to high alcohol strength (50%–100%) and high-volume (20–50 mL) needle injury causing acute thrombosis, or propagation of alcohol along segmental arteries causing multiple artery spasm (figure, right panel). Animal studies have shown concentration-dependent sustained contraction of vascular smooth muscle in segmental radicular arteries and the artery of Adamkiewicz from alcohol.7 Periprocedural prolonged hypotension, commonly seen during CPN secondary to vasodilation of the splanchnic capacitance vessels, may also contribute in some patients.

In our patient the MRI of the spine, performed within 6 hours after the procedure, already showed restricted diffusion from T10 to L1 levels, indicating an acute infarct in the territory of the artery of Adamkiewicz. However, in some patients, MRI may be normal or may show delayed ischemic changes.5,8,9

Paraplegia from CPN/CPB can be either temporary or permanent.5,8 Temporary paraplegia is thought to be due to transient vasospasm induced by alcohol.5 Sensation may improve as early as within 3 days whereas recovery of strength may take 2 weeks to 3 months. High-dose steroids were not beneficial in prior cases of post CPN paraplegia.8 Lumbar drainage had no effect in our patient.

Long-term follow-up data are not available in cases of spinal cord ischemia after CPN due to the short life span in these patients with pancreatic cancer. Fortunately, most patients had complete pain relief following the procedure despite the paraplegia and lived up to 7 months.5,8,9

EUS CPN is currently a preferred technique for refractory cancer-related abdominal pain because of its lower risk for major complications, lower cost compared to CT-guided procedure, less invasiveness as compared to posterior approach or CT-guided procedure, and higher patient satisfaction.2,10 CPN improves the quality of survival in patients with medically refractory pain from pancreatic cancer. The procedure is generally safe, and highly effective, when performed by practitioners with expert training. Yet the risk of a markedly disabling spinal cord injury should be mentioned as a rare complication when consenting patients for this procedure.

AUTHOR CONTRIBUTIONS
Dr. Mittal: conceptualization of study, data gathering and analysis, drafting and revision of manuscript. Dr. Rabinstein: conceptualization of study, drafting and revision of manuscript. Dr. Wijdicks: conceptualization of study, drafting and revision of manuscript.

DISCLOSURE
Dr. Mittal reports no disclosures. Dr. Rabinstein serves as section editor for Year Book Neurology and Neurosurgery and for Neurocritical Care; and receives research support from CardioNet and Boston Scientific. Dr. Wijdicks serves as Editor-in-Chief for Neurocritical Care and receives royalties from The Comatose Patient (2008), Neurological Complications of Critical Illness (2009), The Practice of Emergency and Critical Care Neurology (2010), Brain Death (2011), and Neurocritical Care: What Do I Do Now? (2012) (all published by Oxford University Press).

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