Acute spinal cord infarction after EUS-guided celiac plexus neurolysis

A 73-year-old woman underwent EUS-guided celiac plexus neurolysis to treat severe pancreatic cancer-associated pain. The procedure was performed after administration of intravenous midazolam followed by propofol. The depth of the patient’s sedation was titrated by continuous monitoring with a bispectral index monitor and a pulse oximetry. A 25-gauge needle was advanced anterior to the lateral aspect of the aorta at the level of the celiac trunk. Subsequently, 3 mL of 1% lidocaine and 10 mL of mixed solution consisting of pure alcohol (9 mL) and contrast agent (1 mL) were injected around each side of the celiac trunk, respectively (A). Before the puncture was performed, Doppler mode was used to confirm the absence of intervening vessels. During the puncture, aspirations were negative for blood or cerebrospinal fluid. No problem was observed during the procedure related to the patient’s movement, hiccups, or cough. Postprocedural CT scanning showed that neurolytic/contrast agents were distributed on both sides of the celiac trunk (B). After awaking from sedation, the
patient reported numbness and weakness in her legs. Neurological examination revealed acute paraplegia. Urgent magnetic resonance imaging (MRI) performed 6 hours after the procedure detected no significant acute changes; however, MRI performed the next day demonstrated diffuse intramedullary T2 hyperintensity below the T-11 level to the conus medullaris, which indicates an acute spinal cord infarction (C). Vasospasm of the radicular arteries, which supply the lower two-thirds of the anterior spinal cord, might be provoked due to direct injection or propagation of alcohol close to the arteries. She was treated with intravenous edaravone and underwent rehabilitation. The weakness in her legs gradually improved, although the follow-up MRI obtained 3 months later showed lingering intramedullary T2 hyperintensity, which suggests that the paraplegia is irreversible (D).

DISCLOSURE

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Commentary

This is the third case in the literature reporting paralysis as an adverse event of EUS-guided celiac plexus neurolysis (EUS-CPN).

Pancreatic cancer–associated pain is mediated by nociceptive fibers arising from the pancreas, which then pass through the celiac plexus and splanchnic nerves and finally reach the spinal cord. It is present in 70% of patients with newly diagnosed pancreatic cancer, and in some of them, it gradually becomes refractory to nonsteroidal anti-inflammatory drugs and opioids. When pain control is imperative to improve patients’ quality of life, new techniques appear in the therapeutic arsenal. In 1914, Kappis first described CPN as an analgesic strategy. It consists of a permanent ablation of the celiac plexus neurons by the injection of neurotoxic agents, such as phenol and alcohol. Traditionally, it has been performed under CT or US guidance, with major adverse events reported, such as paraplegia and pneumothorax. EUS allows direct real-time imaging and precise targeting of the celiac plexus, potentially decreasing the morbidity of these traditional approaches. In 1996, Wiersema performed the first EUS-CPN. This approach showed pain reduction in 80% of pancreatic cancer patients. So far, the most commonly reported adverse effects are related to blockade of sympathetic efferent activity and include worsening transient pain, hypotension, diarrhea, and inebriation. Perforation, hemorrhage, and infection are also reported, but their rates are similar to conventional EGD. Despite improved injection-site localization, extremity paraplegia can occur secondary to hypotension or propagation of alcohol causing vasospasm of radicular arteries. The majority of adverse events, including a recently described fatal case, have been reported in patients with chronic pancreatitis–related pain. This case points out that EUS-CPN is not as benign as previously thought, so it should be considered as an alternative only in patients with severe pancreatic cancer–related pain.

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Metastatic periampullary clear cell renal carcinoma

A 50-year-old man with a background history of metastatic renal cell carcinoma (RCC) presented with acute onset of fever, abdominal pain, and jaundice. A partially covered metal biliary stent had been inserted 12 months earlier when he had a mass at the head of the pancreas mass and secondary biliary obstruction. Laboratory test results demonstrated neutrophilia (18.6 × 10^9/L), elevated C-reactive protein (66 mg/L), and elevated lipase (2221 mIU/L). Total bilirubin was 34 μmol/L, alkaline phosphatase was 694 U/L, and γ-glutamyltransferase was 481 U/L. Blood cultures were positive for Klebsiella pneumoniae. An abdominal CT scan revealed dilatation of the