End-organ ischemia as an unforeseen complication of endoscopic-ultrasound-guided celiac plexus neurolysis

Celiac plexus neurolysis (CPN) has been used to treat pancreatic cancer pain for decades. Endoscopic ultrasound (EUS) has improved the safety of CPN, and has allowed its increased use for chronic pancreatitis pain relief [1]. We present a patient who developed end-organ ischemia following EUS-guided CPN, and believe this is the first report of this type of side effect stemming from this procedure.

The patient is a 44-year-old man with a history of chronic alcohol-related pancreatitis. He suffered from debilitating pain for several years before being offered CPN. The procedure was performed under direct EUS visualization with linear array endosonography used to obtain sagittal views of the celiac trunk. Aspiration test was performed with no blood return, and then 20 mL 0.25% bupivacaine and 20 mL dehydrated 98% ethanol were injected. Postoperatively, the patient awoke with severe abdominal pain. Imaging revealed mild pancreatic inflammation and splenic infarction (Fig. 1). Abdominal CT on postoperative day 4 revealed pancreatic necrosis (Fig. 2). Esophagogastroduodenoscopy on postoperative day 6 showed extensive gastric necrosis (Fig. 3). The patient continued to suffer from gastric

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ischemia (Fig. 4), and eventually developed pyloric stenosis and gastric outlet obstruction (Fig. 5). He subsequently underwent subtotal gastrectomy with a Roux-en-Y gastrojejunostomy, and was finally discharged 94 days following CPN. To our knowledge, this is the first report of end-organ infarction in the distribution of the celiac artery following CPN. One meta-analysis found that the most common adverse effects of CPN are local pain, transient diarrhea, and hypotension [2]. Severe adverse effects are uncommon (2%) and include paralysis, parasthesias, prolonged diarrhea, renal puncture, and pneumothorax [2]. In this case, infarction of the spleen, pancreas, and gastric antrum suggest that an ischemic injury occurred during the procedure. We postulate that diffusion of ethanol into the celiac artery and subsequent arterial vasospasm resulted in the injury pattern. Ethanol at nontoxic concentrations has been shown to result in vasoconstriction severe enough to cause vascular and smooth muscle cell death [3]. Paraplegia, a rare but well-established adverse side effect of CPN, is thought to be secondary to diffusion of the neuroablative alcohol into the arteries supplying the spinal cord [3–5]. We postulate that a similar mechanism of injury occurred in this case and resulted in this heretofore unreported adverse event.

Endoscopy_UCTN_Code_CPL_1AL_2AC

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Endoscopy 2009; 41: E218–E219
© Georg Thieme Verlag KG Stuttgart · New York · ISSN 0013-726X

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