Hepatic and Splenic Infarction and Bowel Ischemia Following Endoscopic Ultrasound-Guided Celiac Plexus Neurolysis

Hee Yoon Jang, Sang-Woo Cha, Byung Hoo Lee, Ho Eun Jung, Jin Woo Choo, Yun-Ju Cho, Hye Young Ju and Young Deok Cho

Institute for Digestive Research, Digestive Disease Center, Department of Internal Medicine, Soonchunhyang University College of Medicine, Seoul, Korea

Endoscopic ultrasound-guided celiac plexus neurolysis (EUS-CPN) is a well-established intervention to palliate malignant pain. We report a patient who developed hepatic and splenic infarction and bowel ischemia following EUS-CPN. A 69-year-old man with known lung cancer and pancreatic metastasis was transferred for debilitating, significant epigastric pain for several months. The patient underwent EUS-CPN to palliate the pain. After the procedure, the patient complained continuously of abdominal pain, nausea, and vomiting; hematemesis and hematochezia were newly developed. Abdominal computed tomography revealed infarction of the liver and spleen and ischemia of the stomach and proximal small bowel. On esophagogastroduodenoscopy, hemorrhagic gastroduodenitis, and multiple gastric ulcers were noted without active bleeding. The patient expired on postoperative day 27 despite the best supportive care.

Key Words: Celiac plexus neurolysis; Endoscopic ultrasound-guided fine needle aspiration; Infarction and ischemia

INTRODUCTION

The neurolytic celiac plexus block is a well-established intervention to palliate pain originating from pancreatic cancer and can potentially improve the quality of life of patients. The most frequent indication for celiac plexus neurolysis (CPN) is pain secondary to an upper abdominal malignancy, specifically pancreatic cancer, although it is also effective for pain generated by retroperitoneal tumors or metastases. Recently, endoscopic ultrasound (EUS)-CPN was developed. The side effects of this procedure include orthostatic hypotension, diarrhea, alcoholic neuritis, and retroperitoneal hemorrhage. We present a patient who developed hepatic and splenic infarction and bowel ischemia following EUS-guided CPN.

CASE REPORT

The patient was a 69-year-old man with a history of small cell lung cancer with pancreatic metastasis. He received radiotherapy and chemotherapy, which were completed in September 2011. He suffered from debilitating, significant epigastric pain for several weeks before being offered CPN.

The procedure was performed under direct EUS visualization with linear array endosonography (Linear EUS Olympus GF-UCT240; Olympus, Tokyo, Japan) used to obtain sagittal views of the celiac trunk. The needle was inserted via the transgastric approach, under EUS guidance, immediately anterior-superior to the takeoff of the celiac trunk. Color Doppler EUS procedure was performed to rule out vessel penetration. After insertion of a 22-gauge needle, an aspiration test was performed in front of the celiac trunk with no blood return, and then 5 mL of 0.25% bupivacaine, 10 mL of dehydrated 98% ethanol, 1 mL of triamcinolone acetonide, and 3 mL of normal saline were injected gradually (Fig. 1).

On postoperative day (POD) 1, the patient complained of recurrent abdominal pain, abdominal distension, nausea, and vomiting. On POD 5, a 38°C high fever developed. Abdominal computed tomography revealed infarction of the left lateral segment of the liver and spleen and ischemia of the stom-
ach and proximal small bowels (Figs. 2, 3). On POD 6, bloody diarrhea and hematemesis were observed. On esophagogastro-duodenoscopy (EGD), diffuse erythematous, edematous, exudative, and hemorrhagic mucosal changes were noted on the body and multiple gastric ulcers were noted in the antrum without active bleeding (Fig. 4). Hemorrhagic duodenitis and a duodenal ulcer were also visualized. Oral feedings were withheld from the patient, followed by treatment using a pro-

![Fig. 1](image1.png)

**Fig. 1.** (A) An endoscopic ultrasonography (EUS) image from the proximal stomach demonstrates a longitudinal view of the aorta at the level of the celiac trunk. The vascular landmarks were confirmed by color Doppler image. (B) EUS image of the needle (white arrow) located at the celiac plexus. CT, celiac trunk; AO, aorta.

![Fig. 2](image2.png)

**Fig. 2.** (A) Computed tomography (CT) scan of the abdomen on postoperative day (POD) 5 demonstrating splenic infarction (white arrow). (B) CT scan of the abdomen on POD 5 demonstrating hepatic infarction (black arrow).

![Fig. 3](image3.png)

**Fig. 3.** (A) Computed tomography (CT) scan of the abdomen on postoperative day (POD) 5 with edematous wall thickening of stomach and decreased enhancement degree of gastric mucosa of the upper body (white arrow). (B) CT scan of the abdomen on POD 5 demonstrating diffuse edematous wall thickening of the duodenum (black arrow).
Clin Endosc 2013;46:306-309

End Organ Infarction Following Celiac Plexus Neurolysis

There was no further hematemesis or hematochezia. The patient continued to suffer from abdominal pain, which improved only partially; it could not be controlled with a continuous intravenous infusion of morphine and use of a fentanyl patch. On POD 12, EGD still showed hemorrhagic gastritis with necrotic debris on the posterior wall of the body and revealed healing stage gastric ulcers on the antrum (Fig. 5). The patient continued to suffer from abdominal pain, nausea, vomiting, and bloody diarrhea. He died 27 days following CPN due to multiorgan failure and sepsis despite best supportive care.

**DISCUSSION**

This report presents a patient who was suffering from abdominal pain secondary to pancreatic metastasis of small cell lung cancer. He underwent palliative CPN for abdominal pain and subsequently developed hemorrhagic gastritis and duodenitis as well as infarction of the liver and spleen.

Similar cases have been previously reported. In one case report, the patient developed splenic infarction and gastric ischemia following EUS-guided CPN. In another case report, the patient developed hemorrhagic gastritis and duodenitis after EUG-guided CPN.

It is difficult to offer a precise physiologic explanation for this complication. We speculate that inhibition of sympathetic tone resulting from CPN potentially opened the “floodgates” of unopposed parasympathetic activity, which causes increased acid secretion and blood flow to the gastrointestinal (GI) system, ultimately unmasking indolent hemorrhagic gastritis and duodenitis. Therefore, it may be beneficial for patients with a history of gastritis, duodenitis, or GI bleeding to undergo a careful upper GI evaluation before CPN. Upper GI bleeding can result in the passage of bright red blood from the rectum if the enteral transit time is rapid, which likely oc-
curred in this case. The infarction of the liver, spleen, stomach, and proximal small bowel suggests that an ischemic injury occurred during the procedure. In this case we performed color Doppler EUS and an aspiration test prior to CPN. And only after confirming that there was no penetration of blood vessels was ethanol injected in front of the celiac trunk. We postulate that the diffusion of ethanol into the celiac artery and subsequent vasospasm resulted in the injury pattern. At nontoxic concentrations, ethanol can produce vasoconstriction severe enough to cause vascular and smooth muscle cell death.\(^4,6\)

One meta-analysis found that the most common adverse effects of CPN are local pain (96%), transient diarrhea (44%), and hypotension (38%).\(^7\) Severe adverse effects are uncommon (2%) and include paralysis, paresthesia, prolonged diarrhea, renal puncture, and pneumothorax. Another meta-analysis of EUS-guided CPN for pain due to chronic pancreatitis or pancreatic cancer pain demonstrated that the rate of complications was very low with CPN using EUS as a tool compared with radiologic guidance.\(^8\) No neurological complication has been reported in EUS-guided CPN studies. Other studies have found that EUS-guided CPB and CPN are reasonably safe procedures with tolerable side-effect profiles and low overall complication rates.\(^3,8\)

Conflicts of Interest

The authors have no financial conflicts of interest.

REFERENCES

1. Pello S, Miller A, Ku T, Wang D. Hemorrhagic gastritis and duodenitis following celiac plexus neurolysis. Pain Physician 2009;12:1001-1003.
2. Mercadante S, Nicosia F. Celiac plexus block: a reappraisal. Reg Anesth Pain Med 1998;23:37-48.
3. O’Toole TM, Schmalewitz N. Complication rates of EUS-guided celiac plexus blockade and neurolysis: results of a large case series. Endoscopy 2009;41:593-597.
4. Ahmed HM, Friedman SE, Henriques HE, Berk BS. End-organ ischemia as an unforeseen complication of endoscopic ultrasound-guided celiac plexus neurolysis. Endoscopy 2009;41 Suppl 2:E218-E219.
5. Buckley NM, Brazeau P, Fraser ID, Gootman PM. Circulatory effects of splanchnic nerve stimulation in developing swine. Am J Physiol 1985;248(1 Pt 2):H69-H74.
6. Yang Z, Wang J, Zheng T, Altura FT, Altura BM. Importance of extracellular Ca\(^{2+}\) and intracellular Ca\(^{2+}\) release in ethanol-induced contraction of cerebral arterial smooth muscle. Alcohol 2001;24:145-153.
7. Eisenberg E, Carr DB, Chalmers TC. Neurolytic celiac plexus block for treatment of cancer pain: a meta-analysis. Anesth Analg 1995;80:290-299.
8. Puli SR, Reddy JB, Rechtold ML, Antillon MR, Brugge WR. EUS-guided celiac plexus neurolysis for pain due to chronic pancreatitis or pancreatic cancer pain: a meta-analysis and systematic review. Dig Dis Sci 2009;54:2330-2337.