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GI/GU/Thoracic/Nonvascular Interventions *Case Report*

Severe hypoglycemia after celiac plexus neurolysis: An underreported adverse event?

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ABSTRACT

Celiac plexus neurolysis (CPN) is a technique used to provide palliative analgesia to patients with intractable abdominal pain. We present the case of a male patient who was treated with CT-guided percutaneous CPN to manage pain from metastatic pancreatic cancer. Immediately following the procedure, the patient experienced severe hypoglycemia, which has not previously been reported as a complication of CPN. This complication may be underdiagnosed due to its similarities in presentation to alcohol intoxication, which is considered a common complication of ethanol-based CPN. This case demonstrates the need to monitor blood glucose levels following CPN, particularly in patients exhibiting symptoms of hypoglycemia.

Keywords: Adverse event, Celiac plexus neurolysis, Interventional radiology, Palliative care, Pancreatic cancer

INTRODUCTION

The celiac plexus transmits nociceptive impulses from the upper abdominal viscera to the brain.[1] Percutaneous blockade of the celiac plexus, as a method of providing analgesia for severe abdominal pain, has been practiced for over a century.[1] The procedure was originally performed "blind," but is now performed with the help of image guidance; either percutaneously using computed tomography (CT) imaging or through gastroscope using endoscopic ultrasound (EUS) if technically feasible.[1] While the term celiac plexus block refers to temporary analgesia through injection of corticosteroids or long-acting local anesthetics, celiac plexus neurolysis (CPN) involves the irreversible chemical ablation of the celiac plexus, generally using either ethanol or phenol.[1,2] CPN is often indicated in the case of intractable intra-abdominal pain resulting from malignancies of the pancreas, stomach, esophagus, and biliary tree, with pancreatic cancer being the most common indication.[1,3] Percutaneous CT-guided CPN carries a relatively low risk to patients, with only 2% experiencing major complications.[1] In this report, we describe a rare complication of severe hypoglycemia following CPN.

CASE REPORT

A male patient was referred for the treatment of abdominal pain related to metastatic pancreatic ductal adenocarcinoma. The patient was diagnosed incidentally through imaging studies performed to investigate an unrelated complaint of hematuria. Surgical resection was attempted, but the tumor was deemed unresectable due to extensive disease, and he was subsequently treated with a gemcitabine-based chemotherapy regimen.

His disease progressed over the next 4–5 years; ultimately, his pain could no longer be managed with high doses of opioid analgesics. New imaging studies were ordered, which showed a 12.0 × 3.6 cm mass centered around the pancreas. The mass encased the proximal splenic artery and extended into the lesser sac. Numerous ill-defined lesions were noted in both lobes of the liver, with the largest measuring 5.2 × 4.3 cm. Palliative radiation therapy was considered for local pain control, but it was determined that a significant portion of bowel would be radiated, which could worsen his nausea and poor appetite. After multidisciplinary consultation, it was decided that the patient should be treated with CPN.

Written informed consent was obtained. The patient was positioned in supine position, and intravenous conscious sedation was given. A planning CT of the upper abdomen was then performed. Under CT guidance, the peri-SMA/celiac artery region was targeted with two 21-gauge Chiba needles (Cook Medical, Indiana, US) inserted from an anterior approach. Nine cc of 0.5% bupivacaine (Pfizer, New York, US) mixed with 1 cc of Visipaque (GE Healthcare, Chicago, US) was injected. Repeat CT scan was performed, which demonstrated satisfactory positioning of the needles and distribution of the bupivacaine and contrast mixture [Figure 1].

Twenty cc of 100% ethanol was then injected through each needle. A final CT scan was performed, which demonstrated no evidence of extravasation of contrast into vessel or viscous. Both needles were removed without complication, and the patient was transferred to the recovery room.

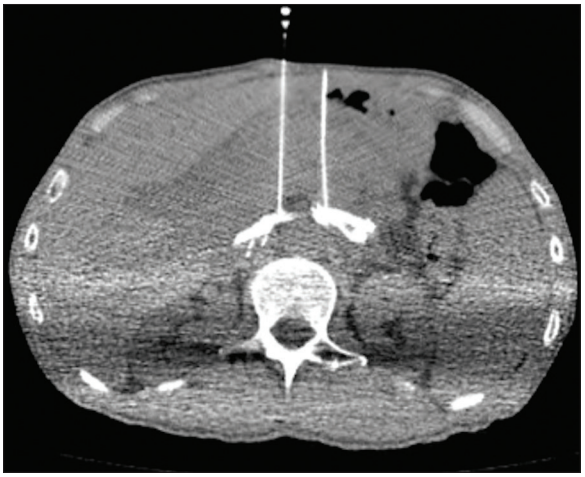

Figure 1: Male patient with severe intractable abdominal pain due to metastatic pancreatic ductal adenocarcinoma being treated with celiac plexus neurolysis. Axial CT image demonstrates position of Chiba needles in the region of the celiac plexus with satisfactory distribution of contrast.

In the recovery room, approximately 45 min following the procedure, the patient experienced sudden decrease in level of consciousness, diaphoresis, hypertension, and bilateral decerebrate posture with upgoing toes. An urgent CT scan of the head was performed to rule out an acute cerebrovascular event. When no evidence of intracranial hemorrhage or infarction was found, finger blood glucose testing was performed. The patient's blood glucose was measured at 0.7 mmol/L (normal fasting glucose: 4.4–6.1 mmol/L), which confirmed a diagnosis of acute hypoglycemia. The patient immediately received 1.5 ampules of D50 and recovered to his baseline blood sugar level within several minutes. The patient was discharged home following several more hours of observation.

Following the procedure, the patient reported that his pain was greatly diminished and was able to reduce his fentanyl patch dosage from 75 mcg/h to 25 mcg/h. However, he experienced repeated episodes of hypoglycemia, with blood glucose levels decreasing to 1.7–2.2 mmol/L intermittently, with symptoms of dizziness, diaphoresis, and tremor. He was treated with high-sugar drinks during these episodes, and it was recommended that he eat every 2–3 h to maintain his blood glucose at appropriate levels. These episodes continued for several months following the procedure, until the patient ultimately died due to complications of his cancer.

DISCUSSION

The prognosis for patients with pancreatic cancer is poor, with a 5-year survival rate of only 6%.[4] Most patients have inoperable disease at the time of diagnosis and are referred for palliative care.[2] In addition to the poor survival statistics, patients are often faced with severe, unrelenting abdominal pain. The primary mechanisms for this pain are pancreatic duct obstruction and neuropathy.[2] Approximately 80% of patients with pancreatic cancer experience pain, and half require strong opioids for pain control.[2] When analgesics are insufficient, the next step in palliative pain management is generally CPN.

CPN is a highly effective method of pain management for patients with pancreatic cancer. About 70–90% of patients experience complete or partial pain relief following CPN that lasts until their time of death.[3] It is generally considered a safe procedure, with serious complications being rare.[1,5,6] Common complications include transient back pain, diarrhea, and orthostatic hypotension.[1] Back pain is thought to result from damage to sensory fibers within the celiac plexus and may occur in as many as 96% of patients.[1,6] Transient diarrhea and orthostatic hypotension, which may present in up to 60% and 53% of patients, respectively, occur as a result of decreased sympathetic tone after the destruction of

sympathetic fibers within the celiac ganglia, leading to unopposed parasympathetic activity.[1,2,6]

The mechanism behind the hypoglycemia experienced by the patient in this case is uncertain. EUS-guided fine-needle aspiration and surgical biopsies confirmed that the patient's cancer was a typical ductal adenocarcinoma, rather than a neuroendocrine tumor capable of inducing hypoglycemia through the secretion of insulin. While hypoglycemic episodes have been observed in patients with non-neuroendocrine tumors (either due to medications, injury to organs involved in glucose regulation, or more rarely, tumors secreting insulin-like growth factor 2 or its precursors),[7] the fact that the patient's first episode took place immediately after CPN indicates that the procedure likely played a causative role.

We propose three mechanisms by which CPN may have caused this hypoglycemic episode. First, CPN may have induced a temporary hyperinsulinemia. If the patient's pancreas came into contact with ethanol during the procedure, the resulting cellular damage may have degranulated islet β-cells and caused a sudden release of insulin. In addition, a rapid rise in serum ethanol concentration may cause vasodilation in the pancreatic islets, increasing blood flow, and stimulating insulin release.[8]

The second likely contributing factor is the sympathetic blockade observed post-CPN. Epinephrine plays an important counterregulatory role in hypoglycemia, stimulating glycogenolysis, and gluconeogenesis while inhibiting insulin release.[9] Successful CPN requires the destruction of the celiac and superior mesenteric ganglia, which contain the cell bodies of post-ganglionic sympathetic neurons that innervate the abdominal viscera, including the adrenal glands.[1,6] The resulting decreased sympathoadrenal signaling could inhibit epinephrine release, potentiating the effects of high insulin and causing more severe hypoglycemia.

Finally, ethanol metabolism is known to inhibit gluconeogenesis by expending NAD^+, which is necessary to synthesize substrates for the pathway. This effect is well characterized, but generally not dangerous unless an individual has fasted for several days and depleted their glycogen stores.[10] However, if glycogenolysis was inhibited by high insulin and impaired epinephrine release, this effect could further exacerbate acute hypoglycemia and delay recovery.

The severe hypoglycemia experienced by the patient in this case has not been previously reported as a complication CPN. While it is likely that this complication is rare, it is also possible that it has thus far been underreported. Alcohol intoxication, which is considered a common complication of ethanol-based CPN, presents with many of the same neurologic symptoms as acute hypoglycemia, including dizziness, confusion, behavioral changes, and blurred vision.[5] Furthermore, other symptoms of hypoglycemia (diaphoresis, tremor, tachycardia, and anxiety) are mediated by sympathoadrenal signaling, which, as discussed, is strongly inhibited following CPN. As a result, hypoglycemia following CPN may be difficult to detect clinically and may be mistaken for alcohol intoxication.

CONCLUSION

Severe hypoglycemia is a rare but serious complication of CPN that may be underreported. While the cause of this complication is unknown, it is likely multifactorial. Physiological changes following CPN could cause hypoglycemia to present atypically, so it should be suspected if any signs or symptoms are present. Physicians should consider routine monitoring of blood glucose in all patients recovering from CPN.

Declaration of patient consent

Patient's consent not required as patients identity is not disclosed or compromised.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Kambadakone A, Thabet A, Gervais DA, Mueller PR, Arellano RS. CT-guided Celiac plexus neurolysis: A review of anatomy, indications, technique, and tips for successful treatment. Radiographics 2011;31:1599-621.
2. Koulouris AI, Banim P, Hart AR. Pain in patients with pancreatic cancer: Prevalence, mechanisms, management and future developments. Dig Dis Sci 2017;62:861-70.
3. Eisenberg E, Carr DB, Chalmers TC. Neurolytic celiac plexus block for treatment of cancer pain: A meta- analysis. Anesth Analg 1995;80:290-5.
4. Ilic M, Ilic I. Epidemiology of pancreatic cancer. World J Gastroenterol 2016;22:9694-705.
5. Yasuda I, Wang HP. Endoscopic ultrasound-guided celiac plexus block and neurolysis. Dig Endosc 2017;29:455-62.
6. Cornman-Homonoff J, Holzwanger DJ, Lee KS, Madoff DC, Li D. Celiac plexus block and neurolysis in the management of chronic upper abdominal pain. Semin Intervent Radiol 2017;34:376-86.
7. Iglesias P, Díez JJ. Management of endocrine disease: A clinical update on tumor-induced hypoglycemia. Eur J Endocrinol

2014;170:147-57.
8. Huang Z, Sjöholm Å. Ethanol acutely stimulates islet blood flow, amplifies insulin secretion, and induces hypoglycemia via nitric oxide and vagally mediated mechanisms. Endocrinology 2008;149:232-6.
9. Sprague JE, Arbeláez AM. Glucose counterregulatory responses to hypoglycemia. Pediatr Endocrinol Rev 2011;9:463-73.
10. Steiner JL, Crowell KT, Lang CH. Impact of alcohol on glycemic control and insulin action. Biomolecules 2015;5:2223-46.

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