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Silent gastric perforation in a pancreatic cancer patient treated with neurolytic celiac plexus block

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Introduction

The neurolytic celiac plexus block (NCPB) is used to treat the pain of upper abdominal cancer when it fails to respond to standard opioid analgesia. A recent meta-analysis indicated long-lasting pain relief for 70% to 90% of patients with pancreatic and other abdominal visceral cancers [1]. The adverse effects related to this procedure are mostly mild and transient, and severe complications are uncommon [2]. The primary mechanism of its analgesic action is interruption of nociceptive impulses from the upper abdominal viscera by chemical destruction of the primary afferent fibers [3]. It is possible, however, that persistent blockade of visceral nociception could mask important diagnostic signs of newly developed adverse changes in the referred organs. In this paper, we report a case of gastric perforation caused by direct invasion of a tumor in a pancreatic cancer patient treated with NCPB, whose relatively mild initial symptoms resulted in a critical delay in treatment.

Case report

A 64-year-old man with unresectable pancreatic cancer was referred to the anesthesiology-based pain service in our division for the management of intractable abdominal pain. Mid-epigastric pain radiating through the back was his first symptom in the diagnosis of cancer in the pancreatic tail, which had been made 6 months previously. At the time of the diagnosis, an advanced stage of malignancy was highly suspected by multiple examinations including radiographic imaging and serological tumor markers. The patient did not choose to have surgical resection of the tumor, and antineoplasmic therapy consisted of gemcitabine chemotherapy, which had no remarkable effect on the tumor progression. His pain intensity progressively increased during the treatment period in spite of opioid therapy. On appearance at our division, his general condition remained relatively good without abnormal findings from either routine blood examination or tests of coagulation function, except for anemia (hemoglobin 11 $g \cdot dl^{-1}$) and hyperglycemia (blood glucose 318 $mg \cdot dl^{-1}$). The latest computed tomography revealed a relatively normal anatomy of the retrocrural space of the diaphragm at the 12th thoracic (Th12)–second lumbar (L2) vertebral levels. Although oral morphine at doses increased to 90 $mg \cdot day^{-1}$ accompanied by 150 $mg \cdot day^{-1}$ of oral diclofenac sodium at our division had failed to relieve his pain, uncontrollable nausea and dizziness prevented a further increase of the morphine dose. Continuous epidural block with 1% lidocaine at the Th5–12 dermatome levels, to which the majority of the splanchnic nerves refer, reduced his pain significantly. We informed him of NCPB as a possible treatment option for his pain. After the patient had given full consent to receive the treatment, NCPB was performed 5 h after termination of the epidural injection of local anesthetics. Using a single-needle transintervertebral disc approach [4,5] (Fig. 1), the celiac neurolysis was completed with 20 ml of 99.5% alcohol after confirming optimal pain relief with a diagnostic injection of 20 ml of 2% lidocaine. The patient obtained significant pain relief with no immediate problems. His daily dose of morphine sulfate was reduced to 20 mg with no rescue doses required upon discharge from the

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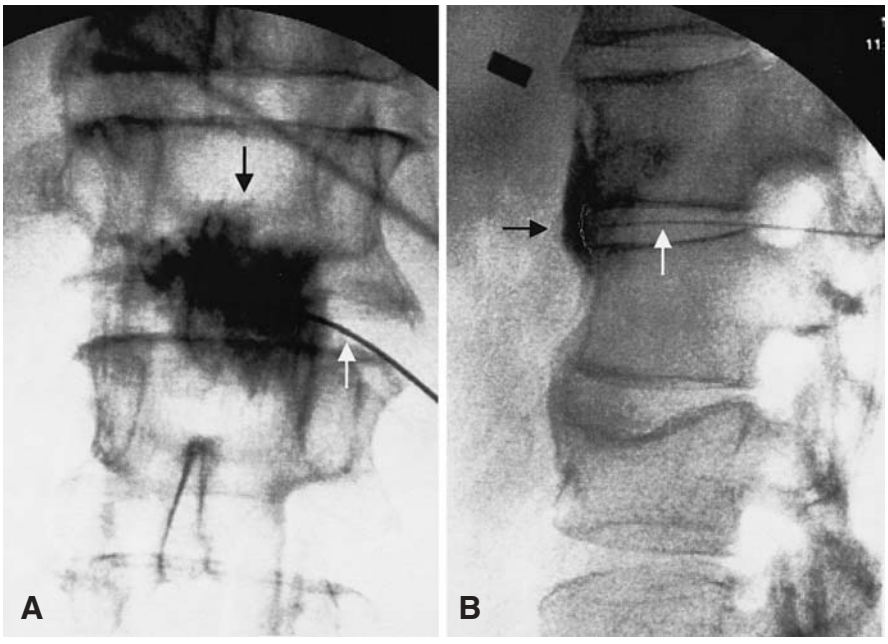

Fig. 1. Celiac plexus block with a single-needle transintervertebral disc approach: the spread of contrast medium (*black arrow*) in the anteroposterior (**A**) and lateral (**B**) views. *White arrow* indicates a 22-gauge needle inserted through the Th12–L1 intervertebral disc

hospital 2 days later. A follow-up blood examination 7 days after discharge showed no remarkable changes compared with the preprocedural values, and the patient experienced persistent pain relief.

In the morning of the 19th day after the NCPB, the patient felt general fatigue and dull pain in the epigastric area. Rescue morphine at a dose of 4 mg and suppository 50 mg diclofenac sodium temporary resolved his symptoms. In the following period, he also noticed mildly increased body temperature (below 38°C), continuous sweating, and decreased urine output. His appetite was reduced, but he continued to eat on that day. However, his discomfort had further progressed by the next morning, and he presented to the emergency room in our hospital by ambulance. Upon admission, he was slightly drowsy with hypotension at 70/45 mmHg and anuria. He complained of nausea and moderate diffuse abdominal pain and had epigastric tenderness. Abdominal muscular defense was not obvious, and lower bowel sounds were still audible at this point. Emergency hematological examination showed leukocytosis (14 000·μl^{-1}), renal failure (blood urea nitrogen 61 mg·dl^{-1}, serum creatinine 3.0 mg·ml^{-1}), and acute inflammatory condition (C reactive protein, 28.7 mg·dl^{-1}). Subsequent abdominal radiography revealed free air under the diaphragm. Peritonitis due to perforation of the gastrointestinal tract was diagnosed. During emergency laparotomy, the surgeons found pyoperitonea in the upper abdominal cavity with perforation of the posterior gastric wall that was caused by direct invasion of the primary cancer. Because of the patient's poor general condition as well as the advanced

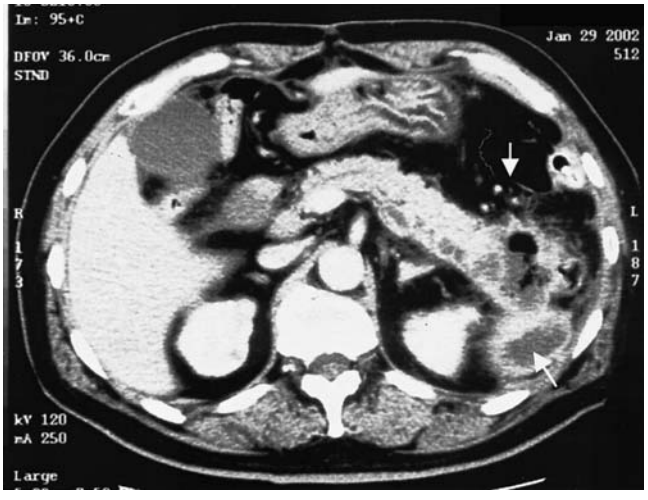

Fig. 2. Computed tomographic scan obtained 14 days after emergency laparotomy. The large tumor in the pancreatic tail (*arrow*) includes a low-density area with air, suggesting residual abscess with a gastric fistula. An abscess accompanied by air bubbles was also found in and around the spleen

regional tumor progression, the surgeons completed the omentopexy on the perforated area with continuous drainage. Temporal mechanical ventilation and inotropic support in the intensive care unit and parenteral nutrition were required for the patient's recovery from the postoperative critical period. Fortunately, he regained most of the preoperative daily activities and pain control a month later, but a small amount of pyodrainage has continued as of this writing (Fig. 2). We are now discussing the possibility of further pallia-

tive surgery or chemotherapy with the patient but have not yet concluded whether it would improve his quality of life.

Discussion

The profoundly depressed general condition as well as direct surgical inspection of the patient strongly suggested that the first onset of gastric perforation occurred one or even two days before the emergency admission. It is surprising that he felt only general fatigue and dull epigastric pain and could continue to eat during the progression of the critical intraabdominal event. We assume that the persistent blockade of visceral nociception induced by the NCPB was primarily responsible for his extraordinary insensibility to the worsening condition. In addition, the sympathetic blockade that was simultaneously accomplished by the NCPB might have suppressed the peritoneal reflexes, modifying his signs and symptoms. As a result, emergency transport to the hospital and subsequent surgical treatment were delayed until the patient's condition had been critically compromised.

The possibility that the initial symptoms in the present patient were reduced by the continuously prescribed analgesics, especially morphine, rather than by the NCPB cannot be absolutely excluded. This possibility is supported by previous reports of compromised diagnosis of abdominal emergency in cancer patients treated with chronic morphine [6,7]. However, the chronic use of analgesics alone rarely blocks rapidly increased nociception effectively; patients often complain of increased pain intensity or require frequent rescue dosing when adverse events develop. This may also be the reason that a large number of cancer patients experience breakthrough pain even under chronic opioid therapy with optimal stable doses. In contrast, complete neurolysis theoretically interrupts all nociceptive impulses nonselectively from the projected organs. Physicians therefore may be confronted with a dilemma, because the destruction of the afferent pathways effectively relieves nociceptive cancer pain but simultaneously disables important physiological alarms for possible emergency conditions.

Several previous studies have validated the significant benefit of NCPB in management of the pain of pancreatic and other upper abdominal cancers [1,8,9]. The adverse effects of this procedure are usually transient and predictable. Hazardous complications that are mostly related to accidental injury of the somatic nerves, vascular structures, or other adjacent viscera are rare [2,3]. Thus, NCPB may not be the last option in a palliative setting and could be used at any stage of the illness, with or without anticancer therapy. However, the present case emphasizes the possibility that effective NCPB paradoxically results in increasing the risk of delayed diagnosis and treatment of newly developed abdominal pathogeneses.

In conclusion, we report silent gastric perforation followed by progressed panperitonitis at diagnosis in a pancreatic cancer patient whose pain was optimally controlled by NCPB. Although neurolysis is a powerful treatment option for managing opioid-resistant cancer pain, the present case alerts us that the persistent blockade of the afferent input can undesirably mask the symptoms of emergency pathogenesis in the related area.

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