Acute Pancreatitis with an Intramural Duodenal Hematoma

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Abstract

The patient was a 43-year-old man admitted to the hospital with intermittent epigastric pain and vomiting, without any evidence of trauma. Blood tests showed elevated lipase/amylase levels. Abdominal computed tomography (CT) revealed pancreatitis complicated by an intramural duodenal hematoma (IDH). He was conservatively treated, and one month after admission, follow-up panendoscopy showed normal duodenal mucosa without luminal narrowing. Non-traumatic IDH is typically associated with coagulation abnormalities. Abdominal CT is an excellent tool for diagnosis in cases of acute abdomen. However, the pathogenesis of and relationship between IDH and pancreatitis remain unknown.

Key words: intramural duodenal hematoma (IDH), acute pancreatitis

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Introduction

Although intramural duodenal hematoma (IDH) is uncommonly noted, it is not a rare condition. IDH was first reported by McLauchlan in 1838 (1) and is caused by blunt abdominal trauma in >70% of cases (2). Non-traumatic IDH is associated with coagulation abnormalities (3) and bleeding into the duodenal wall after endoscopic biopsies (4) or therapeutic procedures. However, IDH is rarely associated with pancreatic disease, and the relationship between these disorders has not been clarified.

Case Report

A 43-year-old man was admitted to the hospital with a one-day history of intermittent epigastric pain and vomiting without any evidence of trauma. These symptoms had resulted in food and water intolerance. The patient had no history of warfarin treatment, and his medical and family history was unremarkable. However, he was a heavy drinker and reported consuming >300 mL of sorghum liquor daily.

A physical examination revealed tenderness and abdominal muscle guarding over the epigastric region. However, the patient’s vital signs were normal. Blood tests showed a hemoglobin level of 9.9 g/dL (normal range: 14-18), white blood cell count of 9,800/μL (4,500-11,000), C-reactive protein level of 11.64 mg/dL (0-0.5), amylase level of 493 U/L (25-125), lipase level of 1,404 U/L (8-78), GGT level of 1,148 U/L (8-60), direct bilirubin level of 1.73 mg/dL (0-0.2) and total bilirubin level of 3.26 mg/dL (0.2-1.6). Contrast-enhanced abdominal computed tomography (CT) (Fig. 1) revealed swelling of the pancreas with surrounding fat stranding and fluid accumulation that resulted in wall thickening of the first to third portion of the duodenum with high-density changes, consistent with a diagnosis of pancreatitis complicated by an IDH.

The patient was therefore diagnosed with IDH complicated by acute pancreatitis and hyperbilirubinemia. During hospitalization, he was conservatively treated with nil per os, pain control and fluid replacement, which resulted in a gradual improvement in the patient’s symptoms and blood test data. Three days after admission, the total bilirubin level decreased to 1.66 mg/dL and the amylase level decreased to 294 U/L. Panendoscopy indicated swelling of the duodenal mucosa and luminal narrowing (Fig. 2). The patient consumed a low-fat diet without discomfort four days after admission and was subsequently discharged 10 days after admission. One month after admission, follow-up panendoscopy showed a normal duodenal mucosa without luminal
Non-traumatic IDH is typically associated with coagulation abnormalities due to the effects of warfarin at the therapeutic or supratherapeutic level (5). Duodenal hematomas are more commonly noted in the second and third segments of the duodenum, as these segments have a relatively fixed position and rich submucosal vascular supply. Moreover, duodenal obstruction is commonly observed with a distended stomach, and surgical intervention may be recommended in such cases (6). As indicated by the findings of previous studies, approximately 14.3-33.3% of patients diagnosed with spontaneous intestinal intramural hemorrhage undergo surgical exploration (7-9). Hematomas are known to rupture into the peritoneum or retroperitoneum, although rarely into the duodenal lumen, due to the presence of the intact submucosal layer. In certain cases, small bowel hematomas extend into the colon; however, cases of intramural colonic hematomas without small bowel involvement are rare (6, 10).

In the present case, the patient presented to the emergency department with abdominal pain, nausea and vomiting. In cases of spontaneous intestinal intramural hemorrhage, the rates of abdominal pain (100%) and nausea/vomit-
ing (64.7-85.7%) (7, 11) are high but nonspecific. Moreover, leukocytosis (white blood cell count: 11,870-15,668 cell/ mm³) (7, 8, 11) and anemia (hemoglobin: 10.3-11.4 g/ dL) (7, 11) are often reported on laboratory examinations in such cases, although they are also nonspecific. In the current case, the diagnosis of pancreatitis was made based on the findings of characteristic abdominal pain, elevated blood amylase and lipase levels three times higher than normal and characteristic features on CT scans, including a mildly enlarged pancreas and increased density of peri-pancreatic dirty fat tissue and fluid collection. The patient also had peritonitis, which is observed in approximately 7.7 to 64.7% of cases of spontaneous intramural intestinal hematoma (7, 8). Peritonitis is typically an important sign for emergency physicians when performing surgical intervention.

Abdominal CT is an excellent tool for diagnosis in cases of acute abdomen. This modality helps emergency physicians to exclude the possibility of intestinal obstruction, hollow organ perforation and mesenteric artery occlusion. In cases in which surgical intervention is not required, as confirmed on abdominal CT, conservative nonsurgical therapy may be considered as the optimal treatment for IDH (6, 12). The same approach is applied in cases of spontaneous intramural intestinal hematoma (7, 10).

Causes of the development of acute pancreatitis in patients with IDH include duodenal papilla obstruction and pancreatic duct compression (13). Obstructive acute pancreatitis is another complication resulting from impaired pancreatic enzyme secretion due to obstruction of the duodenal papilla. Another possible reason for this association is vascular disruption caused by the release of pancreatic enzymes during the process of acute or chronic pancreatitis. Moreover, duodenal or pancreatic artery pseudoaneurysms are usually noted in such cases, although our patient did not have this symptom. Finally, it is difficult to distinguish whether pancreatitis leads to IDH or IDH leads to pancreatitis based only on imaging studies.

In the present report, we described a case of non-traumatic IDH with acute pancreatitis and hyperbilirubinemia. In this case, we initially considered conservative treatment, which proved to be successful. However, in cases involving early complications of severe obstructive pancreatitis or patients who fail to respond to medical treatment, surgical intervention should be considered (6). Although the association between IDH and pancreatitis remains unknown, the details of this case provide useful information regarding this association, which may facilitate further studies in this field. Furthermore, the pathogenesis of and relationship between these two conditions require a considerable amount of additional study.

The authors state that they have no Conflict of Interest (COI).

References


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