



Intramural Gastric Hematoma after Acute Necrotizing Pancreatitis: A Case Report and Review of Imaging Findings

급성 괴사성 췌장염 이후 발생한 위벽 내 혈종:
증례 보고 및 영상 소견 고찰

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Intramural hematoma of the gastrointestinal tract is a rare disease entity. Pancreatitis-induced intramural gastric hematoma (IGH) is far more seldom reported. Here, we report a rare case of a giant IGH occurring as a delayed complication of pancreatitis in a 51-year-old man. The diagnosis was made using computed tomography (CT) and endoscopic ultrasonography. The patient was conservatively managed, and follow-up abdominal CT showed marked decreases in the size of the IGH.

Index terms Stomach; Hematoma; Pancreatitis; Computed Tomography, X-ray;
Endosonography

INTRODUCTION

Intramural hematoma of the gastrointestinal tract is a rare disease entity. Most of such cases are due to blunt abdominal trauma, including endoscopic procedure complications, ulcer disease, and coagulation disorders (1, 2). Intramural hematoma of the gastrointestinal tract related to pancreatitis is rarely reported, and it is most frequently reported in the duodenum (3). Pancreatitis-induced intramural gastric hematoma (IGH) is far more seldom reported. Two cases of pancreatitis-related IGH are currently report-

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Fig. 1. A 51-year-old man with an IGH associated with pancreatitis.

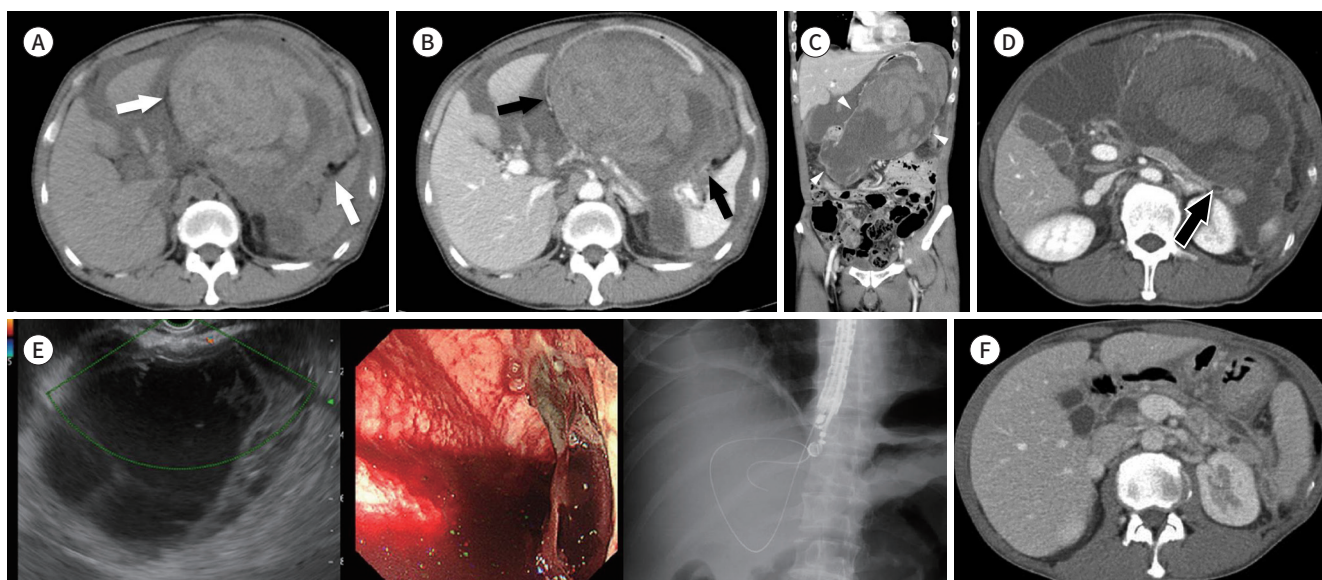
A–C. Axial CT scans showing a mixture of fluid-dense and hyperdense lesions (50–70 Hounsfield units) **(A)** without enhancement (white arrows) and **(B)** along the greater curvature of the stomach (black arrows). **(C)** Coronal CT scan showing a huge hematoma (white arrowheads) along the gastric wall and distension of the gastric wall. There is a small amount of fluid collection with mild peritoneal thickening in the omentum and perisplenic space, suggesting remnant or recurrent walled-off necrosis of pancreatitis.

D. A small tubular structure (arrow) between the pancreatic tail and IGH, suggesting the possible presence of a pancreaticogastric fistula.

E. Endoscopic ultrasonography shows a large cystic lesion with heterogeneous echogenicity and internal septation in the greater curvature of the gastric wall. There was no vascularity within the mass. About 200 mL of fluid was aspirated. As the fluid appeared dark reddish in color, the mass was confirmed as a hematoma.

F. Follow-up abdominal CT scan obtained after about 2 months showing a marked decrease in the size of IGH, and walled-off necrosis around the pancreas tail and retroperitoneum.

IGH = intramural gastric hematoma



ed in the literature (3, 4). Here, we report a rare case of a giant IGH occurring as a delayed complication of pancreatitis, which was diagnosed with computed tomography (CT) and endoscopic ultrasonography (EUS).

CASE REPORT

A 51-year-old man was referred to our hospital for the evaluation of abdominal pain and distention that had started 5 days prior. He had a history of hospital admission because of acute necrotizing pancreatitis 4 months previously. No other relevant medical history was reported. His vital signs were hemodynamically stable, and physical examination showed tenderness in the whole abdomen without definite rebound tenderness. Initial laboratory examination revealed the presence of anemia (9.1 g/dL) and elevated levels of lipase (145 U/L) and amylase (648 U/L). Otherwise, his liver and renal function tests were normal. Abdominal CT was performed at another institution on the day before the referral, which included a nonenhanced-phase scan and a contrast-enhanced portal-phase scan. A huge (maximal diameter, 26 cm), well-defined, heterogeneous, mass-like lesion was found along the greater curvature of the stomach (Fig. 1A). The mass showed a mixture of fluid-dense and hyperdense lesions (50–70 Hounsfield units) on nonenhanced CT, without enhancement on portal

phase, which suggested a hematoma. There was a small amount of fluid collection with rim enhancement close to the hematoma in the perisplenic area, omentum, and retroperitoneum (Fig. 1B, C). A tiny tubular structure between the pancreatic tail and the IGH was also noted, suggesting the possible presence of a pancreaticogastric fistula (Fig. 1D). Our initial diagnosis was IGH occurring as a delayed complication of necrotizing pancreatitis with remaining small foci of walled-off necrosis of the pancreas.

EUS was performed to confirm the diagnosis. On endoscopic examination, fresh blood and old blood clots were found in the lumen of stomach, which made detailed evaluation of the gastric mucosa difficult. Sonography revealed a large cystic lesion with heterogeneous echogenicity and internal septation in the greater curvature of the gastric wall. There was no vascularity within the mass. About 200 mL of dark reddish fluid was aspirated, and the color confirmed that the mass was a hematoma (Fig. 1E).

The patient was conservatively managed with nasogastric decompression, intravenous antibiotics (cefotaxime), intravenous fluid resuscitation, and total parenteral nutrition to allow the bowel to rest. His symptoms improved daily, and his pancreatic enzyme levels became normal on the 6th day after admission. Follow-up EUS 14 days after admission showed a decrease in the size of the cystic mass and a lessened extent of internal septation, suggesting liquefaction of the hematoma. The hematoma was seen to have continuity with the proper muscle layer of the stomach.

The patient was discharged and later followed up at an outpatient department. After about 2 months, abdominal CT showed marked decreases in the IGH size. Rim-enhancing focal fluid collections with decreased size remained at the pancreas tail and retroperitoneum below the pancreas head, and were thought to be remnant walled-off necrosis (Fig. 1F).

DISCUSSION

Intramural hematoma of the gastrointestinal tract is not a common disease entity. Hematoma can occur in the submucosal layer and the proper muscle layer of the gastrointestinal wall after a trauma (5). As it more commonly arises in the duodenum and esophagus, an isolated intramural hematoma in the gastric wall is rare. A review article in 2009 by Dhawan et al. (2) reported that there were 26 cases of IGH in the literature. In this review, most (14 cases) of the cases were associated with coagulopathy. Other possible causes include aneurysm, peptic ulcer disease, and spontaneous occurrence. To the best of our knowledge, 2 IGH cases related to pancreatitis have been reported (3, 4). Compared with these 2 studies, the IGH in our case was relatively huge (maximal diameter, 26 cm). In addition, in the 2 published cases, the patients presented with concurrent pancreatitis, whereas our patient was previously treated for necrotizing pancreatitis and the etiology was thought to be relevant to remnant walled-off necrosis. Therefore, we consider our case as an IGH occurring as a delayed complication of pancreatitis.

As previously mentioned, a tiny tubular structure was seen between the pancreas and the IGH. This imaging finding suggests the possible presence of a pancreaticogastric fistula. External and internal fistulae, including pancreaticogastric and pancreaticopleural fistulae, occurring after pancreatitis have been reported in the literature (6). Pancreaticogastric fistulae

can also be associated with the development of IGH. In addition, considering the anatomic structure, IGH can develop inflammation of the pancreatic tail with walled-off necrosis tracking along the gastrosplenic ligament, extending through the gastric wall and causing leakage of pancreatic juice into the gastric blood vessels.

In the case of intramural duodenal hematoma developing from pancreatitis, which is considered to be better understood than IGH, 2 hypotheses have been proposed concerning the etiologic mechanisms (7). The first one is that simultaneous inflammation of ectopic pancreatic tissue within the duodenal wall contributes to intramural hematoma formation. The other is that pancreatitis and the subsequent leakage of pancreatic juices inflict injury to the duodenal blood vessel, causing hematoma development. In our case, there was no evidence of an ectopic pancreas in the gastric wall. Considering the presence of the tubular structure between the pancreas and the IGH, the second mechanism is a possible explanation in our case.

A careful interpretation of contrast-enhanced CT findings allowed us to make the correct diagnosis. The patient's clinical history of previous pancreatitis, the presence of a high-density region, and the morphologic characteristics of the lesion, including its location along the gastric wall, were clues in our case. The initial diagnosis was confirmed through endoscopic aspiration and by the spontaneous improvement of the hematoma in the patient's clinical course.

The recommended management for pancreatitis-induced IGH is conservative treatment including continuous nasogastric decompression, adequate intravenous fluid resuscitation, and total parenteral nutrition to allow the bowel to rest (8). If there is visible contrast extravasation on CT, or any clinical evidence of hypovolemic shock or ongoing massive bleeding, surgery and transcatheter arterial embolization are alternative treatment options (1).

In conclusion, despite its infrequent incidence, radiologists and clinicians should recognize IGH as a complication of pancreatitis to avoid misdiagnosis, unnecessary additional examination, and invasive treatment.

Conflicts of Interest

The authors have no potential conflicts of interest to disclose.

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급성 괴사성 췌장염 이후 발생한 위벽 내 혈종: 증례 보고 및 영상 소견 고찰

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위벽 내 혈종은 드문 질환으로 특히 췌장염과 관련되어 발생하는 증례는 많이 보고되어 있지 않다. 저자들은 전산화 단층 촬영술과 내시경 초음파를 이용하여 진단한 급성 괴사성 췌장염 이후 발생한 위벽 내 혈종을 보였던 51세 남자의 증례를 보고하고자 한다. 이 환자는 보존적으로 치료하였고, 이후 복부 전산화 단층 촬영에서 성공적인 혈종의 감소를 보였다.

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