Internal Medicine
The Japanese Society of Internal Medicine
doi: 10.2169/internalmedicine.8721-21
Intern Med 61: 1959-1962, 2022
http://internmed.jp

【 CASE REPORT 】

Benign Duodenal Stenosis Caused by Huge Mesenteric Hematoma Conservatively Improved with Long-term Use of Double Elementary Diet Tube

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Abstract:
Mesenteric hematoma is an uncommon condition caused by focal bleeding in the mesenteric vessels. Hematomas are related to trauma, pancreatitis, arteriopathy, and the use of antithrombotic agents. Although hematomas cause intestinal stenosis by compressing the adjacent small bowel, duodenal stenosis due to hematoma is rare. Therefore, the treatment indications for cases of hematoma with stenosis have not been established. We herein report a case with a large mesenteric hematoma that caused duodenal stenosis by compressing the third portion of the duodenum. Stenosis was successfully ameliorated after long-term use of a double elementary diet tube.

Key words: mesenteric hematoma, duodenal stenosis, double elementary diet tube

(Intern Med 61: 1959-1962, 2022)
(DOI: 10.2169/internalmedicine.8721-21)

Introduction

Mesenteric hematoma is an uncommon condition caused by focal bleeding in the mesenteric vessels. Hematomas are associated with trauma, coagulopathies, arteriopathy, pancreatitis, aneurysm, and administration of antithrombotic drugs (1). There have been some cases of idiopathic disease; however, duodenal stenosis associated with abdominal bleeding is rare. Most cases are caused by retroperitoneal bleeding with an arterial aneurysm (2, 3).

We herein report a case of duodenal stenosis caused by a huge mesenteric hematoma that improved after long-term conservative treatment with a double elementary diet tube.

Case Report

A 69-year-old man who had visited our hospital with antiphospholipid syndrome and Buerger's disease complained of upper abdominal pain for 2 days. The patient was a non-drinker and not obese; his body mass index was 21.9 kg/m^2. He had been taking four antithrombotic agents and a small amount of steroids for a long time. His blood pressure was 88/55 mmHg, with facial pallor.

Laboratory findings showed a slightly elevated amylase level and an elevated level of C-reactive protein (257 IU/L and 9.55 mg/dL, respectively). His hemoglobin level was 14.8 g/dL (Table). After infusion of extracellular fluid, his vital signs stabilized.

Dynamic computed tomography (CT) revealed a well-circumscribed mixed-density mass along the mesentery with a maximum diameter of 136 mm. The cranial part of the lesion lies behind the superior mesenteric artery and reaches near the root of the artery. He also had high-density fluid in the abdomen, suggesting hemorrhagic ascites. There were no signs of extravasation (Fig. 1). Mild peripancreatic fat stranding was detected, suggesting acute pancreatitis, but enlargement of the pancreas was not observed. Cysts were observed in the pancreas without dilation of the main pancreatic duct.

We diagnosed him with a mesenteric hematoma possibly related to acute pancreatitis or antithrombotic therapy. Although the hematoma was huge, an emergent surgical operation was not necessary because his vital signs were normal

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Received: September 24, 2021; Accepted: November 3, 2021; Advance Publication by J-STAGE: December 18, 2021
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Table. **Result of Blood Tests on Admission.**

Hematology		Biochemistry			
White blood cells	20,690 /μL	Total protein	7.3 g/dL	Ca	8.4 mg/dL
Neutrophils	91.4 %	Albumin	2.8 g/dL	P	2.7 mg/dL
Lymphocytes	6.4 %	AST	20 U/L	TG	67 mg/dL
Monocytes	1.7 %	ALT	21 U/L	T-Chol	77 mg/dL
Basophils	0.4 %	ALP (JSCC)	232 U/L		
Red blood cells	523 $\times 10^4$/μL	GGT	49 U/L	Serology	
Hemoglobin	14.8 g/dL	LDH	183 U/L	CRP	9.55 mg/dL
Hematocrit	44.5 %	CK	14 U/L		
Platelets	34.8 $\times 10^4$/μL	T-AMY	257 U/L		
Prothrombin activity	14 %	P-AMY	220 U/L		
PT-INR	3.53	Total bilirubin	1.4 mg/dL		
APTT	51.3 s	Direct bilirubin	0.2 mg/dL		
Fibrinogen	423 mg/dL	BUN	9 mg/dL		
FDP	6.2 μg/dL	Creatinine	0.9 mg/dL		
AT-III	79 %	Na	137 mmol/L		
		K	3.7 mmol/L		
		Cl	100 mmol/L		

PT-INR: prothrombin time international normalized ratio, APTT: activated partial thromboplastin time, FDP: fibrin degradation product, AT-III: antithrombin III, AST: aspartate aminotransferase, ALT: alanine aminotransferase, ALP: alkaline phosphatase, GGT: gamma-glutamyl transferase, LDH: lactate dehydrogenase, CK: creatine kinase, T-AMY: total amylase, P-AMY: pancreatic amylase, BUN: blood urea nitrogen, TG: triglyceride, T-Chol: total cholesterol, CRP: C-reactive protein

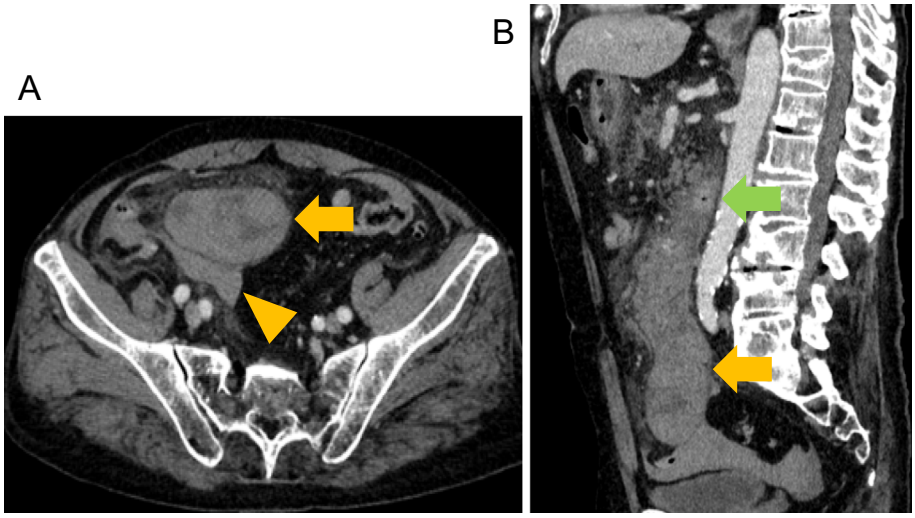

Figure 1. **A: Computed tomography images showing a heterogeneous mass in the abdomen (yellow arrow). High-density fluid is also observed (arrowhead). B: The cranial part of the mass is approaching the inferior wall of the duodenum (green arrow).**

and there were no signs of active arterial bleeding; thus, surgery was not performed.

The day after admission (day 2), his hemoglobin level had decreased to 12.0 g/dL. CT showed enlargement of the hematoma of up to 163 mm. We performed angiography of the celiac artery and superior mesenteric artery, but there were no signs of extravasation, interruption, caliber change, or aneurysm at any branch. We continued careful monitoring and found no indications of bleeding. On the 9th day of admission, since his amylase level had normalized and his abdominal pain had disappeared, he started oral intake. We also resumed antithrombotic drug treatment at the same time.

On the 15th hospitalization day, the patient complained of nausea. Follow-up CT revealed an expanded stomach and duodenum in the first and second parts. There was a radical caliber change in the third portion of the duodenum in the imaging, indicating duodenal stenosis due to compression resulting from the adjacent unchanged hematoma (Fig. 2). Upper endoscopy showed stenosis of the third portion (Fig. 3).

We placed a nasal double elementary diet tube for simul-

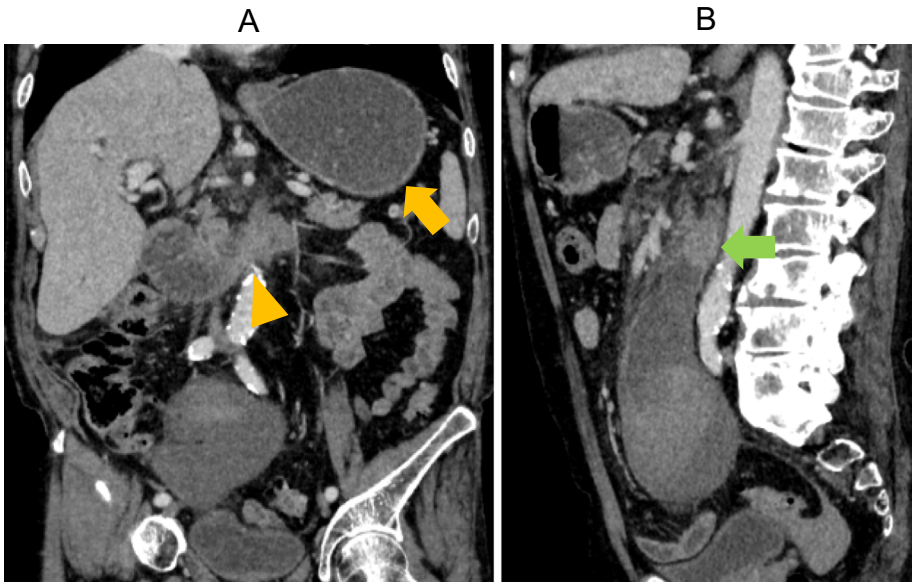

Figure 2. **Contrast-enhanced computed tomography images on day 15. The yellow arrow shows a partially visualized stomach expanding with fluid. The dilated second part of duodenum is also visualized, and a radical caliber change at the third part can be seen (yellow arrowhead). The hematoma was bigger than it was on the admission day and unchanged from day 2. It compressed the horizontal part of the duodenum (green allow).**

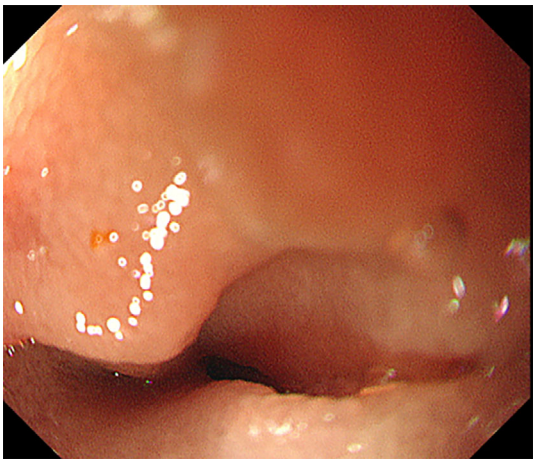

Figure 3. **Upper endoscopy on day 15 demonstrating stenosis at the horizontal part of the duodenum.**

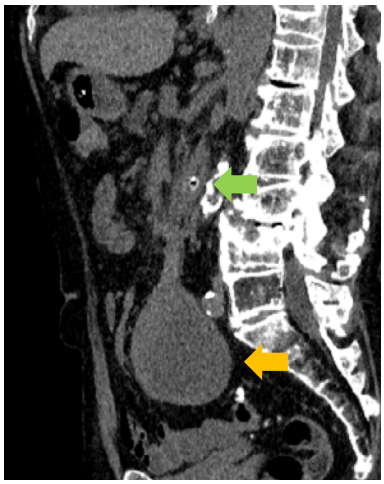

Figure 4. **Plain computed tomography image on day 48 demonstrating the duodenum with the tube (green arrow) and reduced hematoma (yellow arrow).**

taneous enteral feeding through the stenosis and intragastric decompression. Approximately 1,000 mL of output from the tube's drainage lumen was continuously observed until day 40, at which point the output decreased gradually. CT on day 48 demonstrated a reduction in the hematoma to 81.9 mm (Fig. 4).

After removal of the double elementary diet tube, he was able to eat and did not complain of nausea. He was discharged on day 62 of admission. Follow-up CT performed 6 months after discharge revealed a significant reduction in the hematoma to 25 mm. The hematoma was not visible on CT performed one year after discharge.

Discussion

We reported a case of mesenteric hematoma with duodenal stenosis that was successfully treated with long-term conservative management using a double elementary diet tube. Mesenteric hematoma is a rare disease that is typically caused by trauma. Pancreatitis, coagulopathies, arteriopathy, artery aneurysm, and the administration of antithrombotic drugs are risk factors of hematoma (1). The present case had several of these risk factors, including pancreatitis, arteriopathy, and the use of four antithrombotic agents.

Buerger's disease is an arteriopathy that commonly affects

small- to medium-sized arteries of the extremities. It also damages the veins and occasionally visceral vessels. Although there have been reports of an association between Buerger's disease and ischemia of the intestine (4), there have been no reports of intraperitoneal bleeding with the disease. Pancreatitis is a cause of mesenteric hematoma, but most cases are related to arterial aneurysms. Acute pancreatitis was suspected in our case because the patient showed an elevated amylase level and peripancreatic fat stranding, but there was no evidence of extravasation or arterial aneurysm. Although our case's direct cause is unclear, due to a lack of evidence of arterial bleeding and his relatively slow progress course, pancreatitis may have caused bleeding from the veins affected by Buerger's disease, with the bleeding worsened by the use of antithrombotics.

The common symptoms of mesenteric hematoma are nonspecific, including abdominal pain and nausea (5). Nausea and constipation occur when a hematoma compresses the adjacent small intestine (1). However, duodenal stenosis caused by compression of the mesenteric hematoma is rare. Although some cases of duodenal intramural hematomas or retroperitoneal hematomas have been reported (2, 3), only one report of stenosis caused by compression with mesenteric hematoma is available in English (6). The horizontal and ascending parts of the duodenum are attached to the back of the abdominal wall. The horizontal part passes through a gap between the aorta and the superior mesenteric artery. The gap is the site of duodenal obstruction in superior mesenteric artery syndrome, which is caused by narrowing of the gap due to regular emaciation. In the present case, it was suspected that the huge hematoma had developed along with the mesentery compressing the duodenum around the gap and evoked functional stenosis, since the hematoma reached nearly to the root of the superior mesenteric artery and inferior wall of the third portion.

The patient started oral intake on day 9 but complained of nausea on day 15. His oral intake started with an elemental diet and then shifted from a liquid diet to a regular diet. Halfway through his recovery, though, he started to complain of nausea. We speculate that a small amount of the liquid diet was able to pass through the stenosis, but the solid diet occluded it. The duodenal wall then became edematous and exacerbated the stenosis.

Surgical treatment is considered if a patient shows signs of severe blood loss or secondary complications, such as intestinal obstruction or ischemia (7). In stable patients, whether or not huge hematomas shrink as a result of conservative therapy remains unclear. Successful management of small bowel obstruction due to hematoma with short-term conservative nasogastric tube treatment has been reported (8). There was also a case in which shrinkage was not achieved after 60 days of observation; therefore, surgery was performed (7). In patients with small intestinal obstruction, parenteral nutrition is the only choice for long-term nutritional control. In contrast, in cases of duodenal stenosis, long-term observation using a double elementary diet tube can be an option. A double elementary diet tube is a nasal tube with holes at the tip and 40 cm from the tip. When the tip is placed in the duodenum or intestine after passing through a stenosis of the duodenum or distal part of the stomach, the tube enables simultaneous feeding and decompression with two separate lumens. Therefore, conservative therapy can be the first strategy for hemodynamically stable patients with duodenal stenosis due to mesenteric hematoma. In our case, a significant reduction in the hematoma was observed, and the patient showed improvement in obstructive symptoms 30 days after placing the double elementary diet tube.

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

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