Large duodenal hematoma associated with transcatheter arterial embolization following endoscopic hemostasis in a cirrhosis patient: Case report

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Key words: Duodenum, hematoma, arterial, embolization cholangitis, pancreatitis, biliary drainage

INTRODUCTION

Duodenal intramural hematoma is an unusual condition in adults (1,2). However, it has often been reported as a complication of blunt trauma to the abdomen (3). Non-traumatic duodenal intramural hematomas due to anticoagulant therapy, blood dyscrasias, pancreatic disease, and collagen vascular disease (4, 5) are reported less often.

Intramural duodenal hematoma following endoscopic intervention is extremely rare (3), and conservative management is almost always adopted with favorable outcome (6). However, surgical treatment is advocated when intramural duodenal hematoma is complicated by biliary or pancreatic obstruction secondary to duodenal intramural hematoma, especially in patients with bleeding diathesis.

CASE REPORT

To date, there has been no report on duodenal intramural hematoma following transcatheter arterial embolization in bleeding duodenal ulcer refractory to endoscopic hemostasis. We experienced a case of obstructive cholangitis and pancreatitis secondary to duodenal intramural hematoma associated with transcatheter arterial embolization, following endoscopic hemostatic procedures for a bleeding duodenal ulcer in a patient with cirrhosis. The patient was successfully treated with percutaneous transhepatic biliary drainage. We suggest that transcatheter arterial embolization can be a cause of duodenal intramural hematoma, and that percutaneous transhepatic biliary drainage, rather than surgical intervention, can be useful in the treatment of biliary or pancreatic obstruction secondary to duodenal intramural hematoma, especially in patients with bleeding diathesis.

Key words: Duodenum, hematoma, arterial embolization, cholangitis, pancreatitis, biliary drainage

Sirozlu hastada endoskopik hemostazı ve transkateter arteriyel embolizasyonu takiben gelişen büyük duodenal hematom: Olgu sunumu


Anahtar kelimeler: Duodenum, hematoma, arteriyel embolizasyon, kolanjit, pankreatit, biliyer drenaj

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There is no published report on duodenal intramural hematoma following transcatheter arterial embolization (TAE) for the treatment of bleeding duodenal ulcer refractory to endoscopic hemostasis. We report a case of obstructive cholangitis and pancreatitis due to intramural hematoma of the duodenum associated with TAE, following endoscopic hemostatic procedures for a bleeding duodenal ulcer, which was successfully treated by percutaneous transhepatic biliary drainage (PTBD).

CASE REPORT

A 68-year-old man was admitted to our hospital with a three-day history of epigastric pain and tarry stool. He had been a heavy drinker for over 30 years. He had no history of taking non-steroidal anti-inflammatory drugs, anti-platelet agents or anti-coagulants prior to hospital admission. Physical examination was unremarkable except for palpe conjunctiva and mild epigastric tenderness, although he was hemodynamically stable. Initial laboratory investigations revealed the following results: hemoglobin: 5.8 g/dl, white blood cell count: 12,700/μL, platelet count: 74,000/μL, serum albumin: 2.3 g/dl, total bilirubin: 0.8 mg/dl, serum aspartate transaminase: 37 IU/L, serum alanine transaminase: 20 IU/L, prothrombin time: 10.8 sec, partial thromboplastin time: 35.6 sec, and gamma-glutamyltranspeptidase: 204 U/L. Hepatitis B surface antigen, hepatitis B core antibody and hepatitis C virus (HCV) antibody were negative. The severity of his liver cirrhosis was classified as Child-Pugh grade B and was most likely due to his heavy alcohol intake. Upper gastrointestinal endoscopy revealed cardia varices and a round active ulcer measuring about 1.5 cm with a blood-oozing red spot at the distal portion of the duodenal bulb. About 15 ml of diluted epinephrine (1:10,000) was injected submucosally around the ulcer and the bleeding spot was clipped.

Figure 1. Upper gastrointestinal endoscopy revealed an approximately 1.5 cm round active ulcer with a blood-oozing red spot at the distal portion of the duodenal bulb. About 15 ml of diluted epinephrine (1:10,000) was injected submucosally around the ulcer and the bleeding spot was clipped.
patient’s serum white blood cell, amylase and lipase level normalized after six days and his abdominal pain gradually improved. Ten days post-PTBD, an upper gastrointestinal series revealed a deep ulcerative lesion at the duodenal bulb, and passage of contrast was visible through the second and third portion of the duodenum. Fourteen days post-PTBD, TPN was ceased and the patient commenced a liquid diet orally. PTBD catheter was removed four weeks after biliary drainage. A repeat abdominal CT performed in the third month revealed near complete resolution of the duodenal hematoma (Figure 5). The patient has now been followed for two years on an outpatient basis and remains well without specific symptoms.

**DISCUSSION**

Duodenal intramural hematoma is a rare complication of diagnostic and therapeutic endoscopy (3,5). Local injection and forceps biopsy can cause mucosal shearing from the fixed duodenal submucosa that results in intramural hematoma (4,8) because of its fixed retroperitoneal location and rich submucosal blood supply.

To date, there exists no published report on a case associated with TAE, and indeed, this report is the first on the formation of an intramural duodenal hematoma following TAE in the treatment of a bleeding duodenal ulcer refractory to endoscopic hemostasis.

We hypothesize that TAE with coil and the endoscopic hemostatic procedures of submucosal epinephrine injection and clipping against the background of bleeding diathesis may have contributed to the development of a duodenal intramural hematoma.
angiographic embolization is the best alternative option for poor surgical candidates with gastrointestinal bleeding (9,10) refractory to endoscopic intervention.

We postulate that intramural ischemia of the duodenum after TAE was potentially an important etiology of the intramural duodenal hematoma, and contributed to the development of secondary obstructive cholangitis and pancreatitis in this patient. Occlusion of terminal vessels by TAE eliminates collateral flow via the precapillary arterioles (11). Extensive tissue necrosis, local intravascular thrombosis and pancreatitis have been reported as complications of embolization (12,13). In addition, submucosal epinephrine injection performed before TAE in this case is considered another important factor for tissue damage, probably leading to the development of duodenal intramural hematoma. The submucosal injection may have resulted in submucosal dissection in the duodenal wall mucosa by hydrostatic pressure, similar to that produced by endoscopic submucosal dissection. Disruption of submucosal base vessels can cause intramural ischemic necrosis (14-16).

Mechanical hemostasis by means of hemoclips arrests bleeding and has the advantage of minimizing tissue trauma (17). Although hemoclipping can prevent intraluminal bleeding from the submucosal vessels, it may contribute to intra-submucosal bleeding from the injured submucosal vessels beneath the clipping site. It supports our hypothesis in part that previously fixed clips were located at the proximal end of the hematoma on CT in this case.

There are two established treatment strategies for intraluminal hematoma: conservative treatment with nasogastric suction and intravenous fluids, or evacuation of the hematoma surgically (4,7). Other treatment options such as ultrasound-guided aspiration and endoscopic balloon dilatation (18), as well as endoscopic incision and drainage of the hematoma have also been reported (19). Conservative management is considered the treatment of choice if the patient remains stable (7). Laparotomy may become necessary if there is unsatisfactory improvement with non-surgical treatment (4,7).

Most cases of duodenal intramural hematoma with near-total obstruction of the ampulla of Vater complicated by obstructive cholangitis and pancreatitis were treated with laparotomy (3,19). We avoided surgical intervention because our patient had bleeding diathesis and was at high risk of massive bleeding, which may have resulted in poor or operable visual field. The reported mortality rates in patients with cirrhosis undergoing various surgical procedures range from 8.3% to 25%, in comparison to 1.1% in non-cirrhotic patients (20).

**Figure 4.** Cholangiogram revealed marked dilatation of both intrahepatic ducts and proximal common bile duct with narrowing of the distal portion of the common bile duct, suggestive of a bulging extrinsic compression. It is considered a striking feature of a large-diameter duodenal hematoma (arrowhead) of the second portion of the duodenum.

**Figure 5.** Abdominal computed tomography performed three months after transcatheter arterial embolization with coil revealed complete resolution of the duodenal hematoma.
We had decided not to pursue endoscopic excision and drainage in this case because excision with a metal needle knife with aspiration can cause significant re-bleeding in a thrombocytopenic patient. The cholangiogram performed showed marked dilatation of both intrahepatic ducts and the proximal common bile duct associated with narrowing of the distal portion of the common bile duct by a large, round, bulging extrinsic compression. This is a striking feature of a large duodenal hematoma of the second portion of the duodenum and is distinct from the cholangiogram in the other diseases such as cholangiocarcinoma, extrahepatic biliary stone and juxtapapillary duodenal diverticulum.

In conclusion, TAE as well as endoscopic hemostatic procedures such as submucosal epinephrine injection and clipping may have been important causes of duodenal intramural hematoma in this case. We suggest that PTBD might be useful in the treatment of duodenal intramural hematoma complicated by obstructive jaundice, cholangitis and pancreatitis, especially in patients with liver cirrhosis.

REFERENCES