

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

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*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 hemostazi ve transkater arteriyel embolizasyonu takiben gelişen büyük duodenal hematom: Olgu sunumu

Bugüne kadar endoskopik tedaviye dirençli duodenal ülser kanaması nedeni ile uygulanan transarteriyel kateter embolizasyonu takiben gelişen duodenal intramural hematom olgusu bildirilmemiştir. Sirozu olan bir hastada kanayan duodenal ülserin başarısız endoskopik tedavisi ertesinde yapılan, transarteriyel kateter embolizasyonu ile ilişkili olarak oluşan duodenal hematom nedeni ile obstrüktif kolanjit ve pankreatit gelişmiştir. Perkütan biliyer drenaj ile hastanın tedavisi başarılı olmuştur. Bu olgudan elde edilen bilgiler ışığında, transarteriyel kateter embolizasyonu ertesinde duodenal intramural hematom gelişeceğini ve kana ma diyatezisi olan hastalarda perkütan biliyer drenajın biliyer ve pankreatik obstruksyonlarının tedavisi için cerrahi yerine tercih edilebilecek bir yöntem olduğunu düşünmektediriz.

**Anahtar kelimeler:** Duodenum, hematom, arteriyel embolizasyon, kolanjit, pankreatit, biliyer drenaj

## 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 dyscrasia, 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 duct compression (7).

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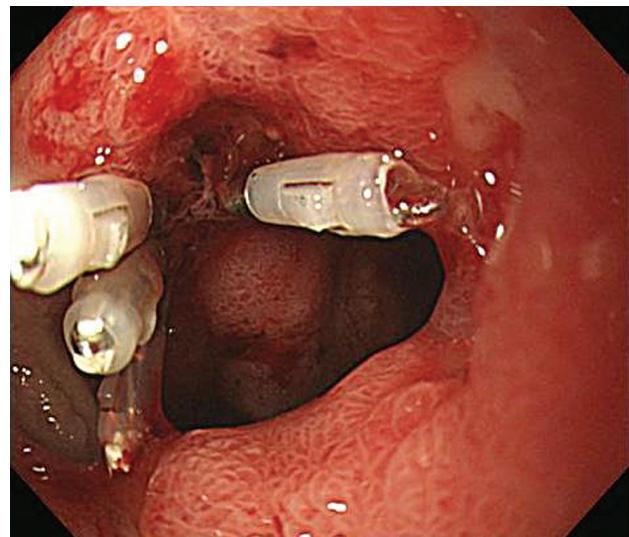
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There is no published report on duodenal intramural hematoma following transcatheter arterial embolization (TAE) for the treatment of bleeding duodenal ulcer refractory to endoscopic hemostasis. We report a case of obstructive cholangitis and pancreatitis 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 pale 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/ $\mu$ L, platelet count: 74,000/ $\mu$ L, 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) mixed with normal saline was injected submucosally around the ulcer, and the bleeding spot was clipped, which stopped the bleeding completely (Figure 1). The patient was subsequently managed conservatively with intravenous pantoprazole and packed red blood cell (RBC) transfusion, in conjunction with total parenteral nutrition (TPN). Three days post-endoscopic treatment, the patient developed tarry stool and hypotension with a systolic blood pressure of 90 mmHg. Repeat endoscopic examination revealed recurrent active bleeding at the site of ulcer treated previously at the distal duodenal bulb. Active bleeding continued despite additional endoscopic he-



**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.

mostatic procedures. Emergency selective gastroduodenal angiography demonstrated extensive contrast extravasation from a small branch of the anterior superior pancreaticoduodenal artery (Figure 2A). Subsequently, a TAE with coil was performed (Figure 2B), which successfully stopped the bleeding and the hemoglobin level stabilized. Five days post-TAE, he presented with abrupt onset upper abdominal pain and pyrexia of 38°C. Physical examination revealed upper abdominal tenderness associated with guarding. Emergency abdominal computed tomography (CT) revealed a large intramural hematoma at the second to third portion of the duodenum associated with marked dilatation of the whole biliary duct above the distal common bile duct (Figure 3). Follow-up laboratory investigation results were as follows: white blood cell count: 13,100/ $\mu$ L, platelet count: 101,000/ $\mu$ L, prothrombin time: 14.2 sec, and partial thromboplastin time: 47.5 sec. His serum hemoglobin level decreased from 9.7 g/dL to 7.2 g/dL, serum amylase was 860 IU/L, serum lipase 1280 IU/L, and total bilirubin 12.4 mg/dL, with 8.8 mg/dL of direct bilirubin. Percutaneous transhepatic cholangiography (PTC) revealed marked dilatation of the whole biliary duct and narrowing of the distal portion of the common bile duct, suggestive of a bulging, extrinsic compression (Figure 4). Emergency PTBD was performed and intravenous cefazidime and metronidazole were commenced. The



**Figure 2.** Selective gastroduodenal angiogram showed extensive contrast extravasation (arrow: pseudovessel sign) from a small branch of the anterior superior pancreaticoduodenal artery (arrowhead) (A), and successful transcatheter arterial embolization with coil was performed. After embolization, coil (arrowhead) and previously fixed clips (arrow) were noted (B).

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 he-



**Figure 3.** Abdominal computed tomography revealed a huge intramural hematoma at the markedly dilated second to the third portion of the duodenum, and dilatations of all biliary ducts above the distal common bile duct. A tiny high-density spot at the proximal portion of the hematoma was considered the location of previously fixed clips and the starting point of the duodenal intramural hematoma.



**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.

matoma. 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 operative 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).

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 ot-

her 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.

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