**CASE REPORT**

**Gastrointestinal hemorrhage caused by rupture of a pseudoaneurysm of the hepatic artery**

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Gastrointestinal bleeding secondary to rupture of a hepatic artery pseudoaneurysm is rare. We report the case of a 61-year-old woman, who was admitted to our institution with hematemesis and melena. Upper gastrointestinal endoscopy and flexible sigmoidoscopy failed to reveal any significant abnormality. Computed tomography scan showed an aneurysm arising from the hepatic artery. A selective angiography showed a ruptured pseudoaneurysm originating from the right hepatic artery with extravasation. It was decided to embolize the pseudoaneurysm, and the neck of the pseudoaneurysm was occluded successfully with two microcoils. While these investigations and interventions were being performed, disseminated intravascular coagulation and acute hepatic failure occurred due to the massive blood transfusion and gross intrahepatic hematoma. Consequently, the patient died 10 days after admission. In this case, we observed that delayed diagnosis of hepatic artery aneurysm rupture may lead to a life-threatening situation. Thus, computed tomography and selective angiography should be obtained immediately. The percutaneous super-selective angiographic embolization of intrahepatic aneurysms is a promising form of treatment, with low risk.

Key words: Hepatic artery, pseudoaneurysm, gastrointestinal bleeding

**Hepatik arter psödoanevrizma rüptürüne bağlı gastrointestinal kanama**


Anahtar kelimeler: Hepatik arter, psödoanevrizma, gastrointestinal kanama

**INTRODUCTION**

Hepatic artery aneurysms (HAAs) represent approximately 20% of all visceral artery aneurysms, and 80% of these aneurysms are extrahepatic. The majority of these aneurysms are solitary and involve the common or right hepatic artery (1,2). Pathologic findings have shown that up to 50% of HAAs are pseudoaneurysms. This incidence of hepatic artery pseudoaneurysm (HAPA) might be caused by invasive percutaneous procedures to the liver that can cause injury to the arterial walls (3). While infection was the primary cause of hepatic aneurysms previously, infected aneurysms are less prevalent today. Pathologically, extrahepatic HAAs are usually true aneurysms with evidence of medial degeneration and secondary arteriosclerosis. Less common causes include polyarteritis

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**Manuscript received:** 21.04.2010  **Accepted:** 29.07.2010

Turk J Gastroenterol 2012; 23 (2): 160-164
doi: 10.4318/tjg.2012.0460
nodosa, as suspected in our patient, or other vasculitides, fibromuscular dysplasia, trauma, and inflammation in the context of acute pancreatitis or cholecystitis (as a result of gallstones) (1,4). True intrahepatic HAAs have similar etiologies. The presentation of HAPA varies from a silent incidental finding to an acute life-threatening hemorrhage caused by rupture. HAPA may rupture into the adjacent hepatic venous, portal or gastrointestinal system, or directly into the abdominal cavity. Patients may present with acute upper or lower gastrointestinal bleeding, signs of an acute abdomen accompanied by jaundice or fever, or a postoperative hemorrhage from an intraabdominal drain (5). In 80% of patients with HAAs, rupture of the aneurysm is the initial clinical event (6). These aneurysms are often diagnosed as incidental findings on ultrasonography or computed tomography (CT) scan. Others are detected at the time of mesenteric arteriography for gastrointestinal hemorrhage. During the last decade, the most commonly used technique for treating HAAs has been percutaneous catheter-based embolization (37%) (7). Gastrointestinal hemorrhage caused by rupture of a HAA is rare, but it has been reported (8-12). We report an unusual case of a patient with intrahepatic HAPA who presented with gastrointestinal hemorrhage.

CASE REPORT
A 61-year-old woman presented with multiple episodes of hematemesis and melena. She had a two-month history of intermittent rectal bleeding and bloody vomiting. She also complained of right lower quadrant pain. Her medical history was significant for a laparotomy due to a liver abscess 30 years earlier and an endoscopic retrograde cholangiopancreatography (ERCP) at another medical center due to choledocholithiasis one month earlier; multiple gallstones were demonstrated. There was no history of prior use of nonsteroidal anti-inflammatory drugs. On arrival, the patient was pale and had mild epigastric tenderness. Fresh blood was seen on rectal examination. Vital signs included a pulse of 80 bpm, blood pressure of 110/60 mmHg, and respiratory rate of 20 bpm. Her hemoglobin was 8.25 g/dl (normal: 12-16 g/dl), and she was resuscitated and transfused with 2 units of packed red blood cells. All her liver function tests were slightly elevated: serum aspartate aminotransferase (AST) 89 U/L (normal: 0-40 U/L), alanine aminotransferase (ALT) 57 U/L (normal: 0-41 U/L), alkaline phosphatase (ALP) 296 U/L (normal: 15-250 U/L), and total bilirubin 3.1 mg/dl (normal: 0.2-1.2 mg/dl). Serum lipase was 106 U/L (normal: 13-60 U/L) and amylase was normal. Upper gastrointestinal endoscopy and flexible sigmoidoscopy did not reveal any significant abnormality apart from bleeding from the posterior wall of the duodenal bulb; abdominal ultrasonography showed multiple gallstones in the gallbladder. Four days after the admission, abundant hematemesis and hematochezia occurred abruptly, and hypotension developed (50/20 mmHg). The patient was transferred urgently to the intensive care unit and resuscitated with 5 units of packed red blood cells and 4 units of fresh frozen plasma. A CT scan was performed because the second endoscopy and flexible sigmoidoscopy failed to reveal any significant abnormality. The abdominal CT scan showed an 18x10 cm intrahepatic hematoma and two contrast-enhancing lesions (16x9 mm and 13x8 mm) consistent with intrahepatic aneurysms arising from the right hepatic artery (Figures 1, 2). The patient was transferred to the angiography unit immediately after the CT scan, and celiac angiography was performed using a right femoral approach. After catheterization of the main hepatic artery with a 5-F shepherd-hook angiographic catheter (Shepherd Hook, Terumo, Japan), selective angiography (SA) showed a thin-necked, bilobulated, 24x12 mm ruptured pseudoaneurysm originating from the right hepatic artery with extravasation (Figure 3). After the diagnostic images had

Figure 1. Computed tomography scan of the abdomen showing large common intrahepatic hematoma.
been reviewed, it was decided to embolize the pseudoaneurysm. To perform super-selective catheterization of the pseudoaneurysm neck, we used a microcatheter (Fasttracker, Boston Scientific, USA) and a 0.016-inch guidewire with a gold coil (Guide Wire GT, Terumo, Japan), placing the catheter tip onto the pseudoaneurysm neck. The pseudoaneurysm neck was occluded successfully with 2 microcoils (3 x 30 mm, 5 x 30 mm; Platinum Coil, Boston Scientific, USA). A follow-up angiography showed no flow into the pseudoaneurysm; nevertheless, the blood flow into the artery continued (Figure 4). One instance of disseminated intravascular coagulation (DIC) and acute hepatic failure occurred due to massive blood transfusion (17 units of red blood cells) and gross intrahepatic hematoma. All of her liver function tests continued to be excessively elevated: ALT 1631 U/L, AST 4213 U/L, and ALP 360 U/L; her lactate dehydrogenase (LDH) was 7250 U/L, and total bilirubin was 8.9 mg/dl. The high level of fibrin degradation products and thrombocytopenia were indicative of the DIC. Tracheal intubation was performed as a consequence of respiratory failure. During her time in the intensive care unit, hypotension occurred, which was resistant to vasopressor drugs. Consequently, 10 days after admission, the patient died due to acute hepatic failure and DIC.

**DISCUSSION**

We observed in this report a fatal outcome due to the delayed diagnosis of the HAPA rupture in a patient who presented with hematemesis and melena.

In our patient, we did not consider the laparotomy (due to the liver abscess) and ERCP as etiologic factors for HAPA, because the laparotomy had been performed 30 years earlier, and the ERCP had been done after the onset of hematemesis and melena.

Most HAAs remain asymptomatic. These aneurysms are often diagnosed as incidental findings on ultrasonography or CT scan. When symptomatic, the most common presentation of patients with intact aneurysms is continuous right upper quadrant or epigastric abdominal pain (1). In 80% of patients with HAAs, rupture of the aneurysm is the initial clinical event (6). The overall mortality rate associated with HAA rupture is 35% (13,14). Rupture occurs into the biliary tract, the peritoneal cavity, or gastrointestinal tract. Gastrointestinal hemorrhage caused by rupture of HAAs is rare but has been reported (8-12). In these case reports, gastrointestinal endoscopy revealed no significant abnormality, and HAA was diagnosed on ultrasonography, CT scan, or angiography, as was the case in our patient. However, ultrasonography did not show a significant abnormality in our pati-
ent. SA remains the most sensitive test for detecting HAA. Tobben and coworkers (15) reported that 100% of HAPAs were detected by SA in their series of 10 patients, compared with only 67% detected by CT and 33% detected by duplex sonography. SA may also show active bleeding and anatomic variations such as anomalous or replaced hepatic arteries (16). Thus, some authors recommend an initial CT scan if HAPA is suspected, followed by a confirmatory SA (17).

The mainstay of treatment is selective endovascular embolization of the feeding artery proximal to the HAPA (18,19). The short-term outcome of patients with repaired HAPA is directly related to its preoperative rupture status. For stable, nonruptured HAPA, occlusion is achieved by embolization in 88%-100% of cases (19,20). Good results of embolization for even ruptured HAPA have been reported (21). There have been isolated reports of hepatic necrosis after embolization, probably as the result of nonselective deployment or embolization in the presence of an occluded portal vein (22). Although both proximal ligation and embolization can result in subsequent liver necrosis, embolization limits hepatic devascularization by reducing the area of liver deprived of arterial flow. Additionally, embolization eliminates the need for an exploratory laparotomy and the complications of general anesthesia (1). Other complications of embolization include rupture, abscess formation, infection, sepsis, ascites, jaundice, gallbladder necrosis, and the formation of pseudoaneurysm or hematoma at the catheter site (1). In our patient, a selective transcatheter embolization of the aneurysm had been performed. Surgical repair of HAPA may be required when embolization fails, if there is an orthotopic liver transplant or an associated pseudocyst. Surgical interventions for intrahepatic aneurysms in the past have included segmental resection of the liver parenchyma or ligation of the involved afferent and efferent vessels. Although there are no large studies, the surgical mortality in these patients has been observed to be high (23-25).

In summary, if the cause of gastrointestinal hemorrhage remains unknown after routine diagnostic measures, and the origin of bleeding in the upper gastrointestinal tract cannot be identified, aneurysms of the abdominal arteries should be considered. This source of bleeding could not be brought under control endoscopically. Delayed diagnosis of the rupture of HAAs may lead to a life-threatening situation. Thus, CT and SA should be obtained immediately. Percutaneous super-selective angiographic embolization of intrahepatic aneurysms is a promising form of treatment, with low risk.

REFERENCES


