Hemobilia after 49-days after Accidental Hepatic Trauma and Twice Perihepatic Packing

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ABSTRACT

Hemobilia occurs in only 1.2–5% of patients with accidental liver trauma. Clinical presentation of hemobilia include one symptom and two signs known as the classic Quinke triad, upper abdominal pain, upper gastrointestinal bleeding and jaundice. This report describes a case of patient with hemobilia that had been diagnosed 49 days after accidental hepatic trauma and twice perihepatic packing.

A 29-year old man with hematemesis melena came to the hospital with the history of hepatic accidental trauma and had already received twice perihepatic packing treatment. At first, the esophagogastroduodenoscopy could only find gastritis and duodenal ulcer. Subsesquently, the ultrasonography revealed liver hematoma. Diagnosis of hemobilia was made when bleeding exiting from the ampulla of Vater which then was confirmed by the second esophagogastroduodenoscopy. Surgery had been planned but the patient and his family had refused the procedure.

Hemobilia should be considered in patients presenting with gastrointestinal blood loss after liver injury. The diagnosis is confirmed by esophagogastroduodenoscopy and angiography. Modalities used to stop bleeding include angiography with embolization, surgical intervention, observation, and electrocoagulation or photocoagulation.

***Keywords**: hematemesis, melena, liver injury, hemobilia, esophagogastroduodenoscopy*

INTRODUCTION

Hematemesis and/or melena are clinical manifestations of upper gastrointestinal (GI) bleeding. Simadibrata reports that the cause of upper GI tract bleeding in Cipto Mangunkusumo hospital between 1996-1998 were variceal bleeding 27.2%, erosive gastritis 19%, portal hypertension gastropathy 11.7%, duodenal ulcer 5.7%, gastric ulcer 5.5%, combination cause 22.1%, others 8.8%; however, there has been no data about hemobilia.[1]

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The annual incidence of hospital admissions for upper GI bleeding in the US and Europe is 0.1%. The common cause of bleeding as recorded from 2000 to 2002 were ulcers in 31-59% cases, varices in 7-20%, Mallory-Weiss tears 4-8%, gastroduodenal erosion in 2-7%, erosive esophagitis in 1-13% cases.[2]

Hemobilia is a rare clinical condition that has to be considered in differential diagnosis of upper GI bleeding.[3] After accidental liver trauma, the incidence of hemobilia is rare, occurring in only 1.2–5% of patients.[4] The following report describes a case of patient with hemobilia that had been diagnosed in 49-days after accidental hepatic trauma and twice perihepatic packing.

CASE ILLUSTRATION

A 29-year old male patient with previous motor vehicle accident 35 days prior to his admission came to Cipto Mangunkusumo hospital. Right after the accident, ultrasonography was performed in a private hospital and showing fluid in Morrison pouch and recto buli as well as minimal right pleural effusion. The patient was diagnosed with hepatic rupture and underwent an exploration laparotomy immediately. During surgery they found hepatic ruptured in at the VI-VII zone. Perihepatic packing was given to the patient. After the procedure, patient got better, hemodinamically stabil, no hematemesis or melena occurred. The patient was hospitalized for 15 days.

Five days after being discharged from hospital, the patient had hematemesis for 4 times, ± 700 cc; then he was admitted again to the hospital. He went through second exploration laparotomy and perihepatic packing. After the procedure, hematemesis and melena still occurred. He was subsesquently referred to another private hospital to had esophagogastro-duodenoscopy (EGD) procedure, which revealed erosive gastritis and duodenal ulcer. No abnormality at papilla of Vater was found. He decided to discontinue the hospitalization after the procedure. Third day after the second hospitalization, he experienced melena for 5 days with the amount of 100 cc of bleeding each day and 3 hours prior to his admission to Cipto Mangunkusumo hospital, he had hematemesis along with ten minutes colic abdominal pain.

The physical examination revealed pale, no jaundice, there was no rebound tenderness or Murphy's sign, no abdominal masses was found and the bowel sounds were normal. There was pain in epigastric and right upper quadrant. A digital rectal examination showed melena. Laboratory tests result showed normocytic and normochrome anemia with hemoglobin level of 8.5 mg/dL, leukocytosis 12,300 mg/dL, elevated transaminase enzymes i.e. AST 61 U/L, ALT 42 U/L, and hypoalbuminemia with albumin serum level 2.8 g/dL. The USG showed anechoic structure 5.5 cm at the 7-8th segment and hypoechoic round structure sized 3.5 cm at the 7th segment, which revealed suspected liver hematoma.

During hospitalization in emergency room and intermediate ward of Cipto Mangunkusumo hospital, he was assessed with hematemesis melena and erosive gastritis was suspected to be the etiology. Such condition was assumed because he had taked non steroid anti inflammatory drugs (NSAID), mefenamic acid, during his first and second hospitalization, and duodenal ulcer which was found at previous esophagogastroduodenoscopy (EGD). He was then treated with 40 mg of intravenous omeprazole once daily and 10 cc of sucralfat four times daily.

Hematemesis melena still occurred along with recurrent episodes of hypovolemic shock. Blood transfusions were administered several times. The surgeon stated that there were no signs of acute abdomen and no indication for surgery. After being hemodinamically stable, patient was moved to the regular ward. While being treated at regular ward, the hematemesis melena still occurred in amount of approximatelly ± 300 cc/day. Ocreotide acetate 2 ampules/4 hours, omeprazole drip infusion 8 mg/hours, intravenous tranexamid acid 3 x 500 mg/day were administered. Such treatment was not effective to stop the bleeding. Hemostasis parameter showed that the bleeding time and clotting time were in normal range, prothrombin time 12.3 seconds (k: 12.1), activated partial thromboplastin time 35.2 seconds (k: 34.9), fibrinogen level was 467 mg/L, D-dimer 1,200 ng/L. Five hundred milliliters of fresh frozen plasma transfusion was delivered.

On the sixth day at regular ward, he suddenly felt severe colic abdominal pain on the right upper quadrant with massive melena ± 600 cc. There were no tenderness, no muscular defence, and bowel sounds were normal. Immediately, surgeons were contacted and they concluded that there were no signs of acute abdomen, and no indication for immediate surgery procedure. Jaundice was seen in the sclerae of both eyes. The bilirubin level was increased i.e. total bilirubin level was 3.47 mg/dL; direct bilirubin level was 2.95 mg/dL; and indirect bilirubin level was 0.52 mg/dL).

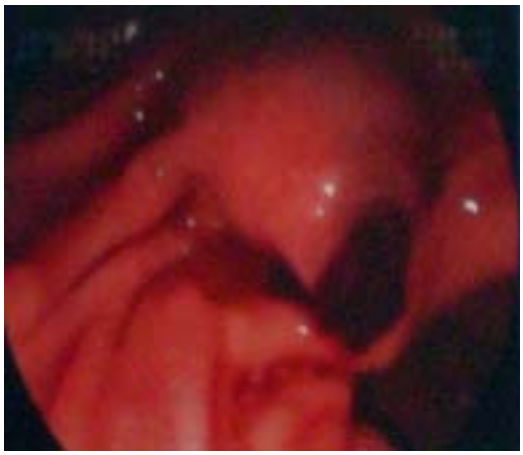

Figure 1. Esofagogastroduodenoscopy showed bleeding from ampulla of Vater

Patient underwent EGD procedure on the next day, which subsequently confirmed the diagnosis of hemobilia. There was bleeding seen at ampulla of Vater. This capture was suspected from intra hepatic gall tract or liver hematoma. Surgery had been planned but the patients and his family had refused the procedure. Patient was taken home and died two days after.

DISCUSSION

The phenomenon, which now has been known as hemobilia was first recorded in XVII century by a well-known anatomist from Cambridge, Francis Glisson and his description was published in Anatomia Hepatic in 1654.[3] The term "traumatic hemobilia" was suggested by Sandblom in 1948 to designate hemorrhage into the bile ducts as a result of trauma to the liver.[5]

Hemobilia is a rare clinical condition that has to be considered in differential diagnosis of upper GI bleeding. In Western countries, the leading cause of hemobilia is hepatic trauma with bleeding from an intrahepatic branch of the hepatic artery into a biliary duct, mostly it is iatrogenic in origin, e.g. needle biopsy of the liver or percutaneous cholangiography. Less common causes include hepatic neoplasm; rupture of a hepatic artery aneurysm, hepatic abscess, choledocholithiasis and in Asia countries, additional causes include ductal parasitism by *Ascaris lumbricoides* and oriental cholangiohepatitis.[3] Post-traumatic hemobilia is the most common etiologic category, accounting for 55% of cases in Sandblom's collected series and 40% to 85% of cases in more recent reports that reflect a rise in the incidence of blunt and penetrating liver injury due to violent crime and vehicular accidents and the increased use of percutaneous biopsy and intubation techniques for managing hepatobiliary diseases. The liver is the most commonly injured abdominal organ, and hemobilia may complicate the clinical course of as many as 3% of patients with substantial liver injuries.[6]

The classic clinical presentation of hemobilia, the Quinke triad, include one symptom and two sign: (1) upper abdominal pain; (2) upper gastrointestinal bleeding; (3) jaundice.[3] The patient had all of them. Other clinical presentation of hemobilia include biliary colic in 70% of patients, jaundice in 60%, and GI bleeding in all patients, which may range from occult to massive bleeding. The classic triad of pain, jaundice, and bleeding is present in only 32% to 40% of patients.[6]

The underlying cause of haemobilia in this case is a hepatic ruptur which occurred after an accidental liver injury from motor vehicle accident in 49 days prior to the hospital admission. Hepatic trauma occurs in approximately 5% of all admissions in emergency rooms. The anatomic location and the size of the liver make the organ even more susceptible to trauma and it is frequently affected in penetrating injuries. Motor vehicle accidents are the most common etiology for blunt liver injury followed by pedestrians and car collisions, falls, motorcycle crashes and penetrating injuries. Hemobilia is uncommon complication and occurs in less than 3% of liver injuries. Nowadays perihepatic packing is the most accepted method in the management of major liver injuries. The decision to pack must be made early at the time of the exploration in order to provide better chances of survival for patients with liver trauma. Abbreviated and extremely necessary procedures only done to keep patient alive, like packing are called "damage control".[7] The patient had have twice perihepatic packing as the treatment of his liver injury.

Diagnosing hemobilia is difficult, as it can occur several days after liver trauma and veiling the cause. After blunt abdominal trauma, hemobilia usually occurs much later than after penetrating injuries. Hematoma and bile accumulation occur more gradually, inhibiting coagulum formation and inducing further necrosis. Eventually, fistula formation occurs between these collections, the hepatic vessels and bile ducts. After penetrating trauma this process of cavitations and fluid formation is exceptional. Longer delays in these circumstances have only been described following rupture of pseudoaneurysms into the biliary system.[8] Hemobilia after hepatobiliary operation is a rare form of upper GI bleeding but has a high mortality rate. Bleeding may be delayed occurring weeks or months after surgery.[9] Delays presentation were documented by Krige at al, the mean delay between the initial liver injury and the diagnosis of hemobilia was 23.5 days wiith range of 1-120 days, Durban found mean delay of 16 days with range of 7-211 days.[10]

EGD should be the initial diagnostic test in patients manifesting an upper GI bleeding after hepatic trauma. Gastritis or ulcer will be discovered in the majority of cases. Endoscopic diagnosis of hemobilia is made when bleeding exiting from the ampulla of Vater. However, bleeding is usually intermittent and is endoscopically observed in about 12-30% of cases.[11,12] We assumed that hemobilia has already existed when the patient underwent EGD at the private hospital; however, the procedure didn't reveal the bleeding and only found gastritis and duodenal ulcer.

Ideally, the next step is to try and localize the bleeding vessel. Ultrasound examination at this stage is of little help and may even be misleading when the bile ducts are full of blood. If the patient is hemodynamically stable, CT angiography shall be performed subsequently. Such investigation may not only detect the bleeding site but also demonstrate lesions like a liver abscess or tumor, which then we can proceed with operation. If the CT scan is negative or the patient is unstable, then he or she should have selective angiography of the hepatic artery and embolization of the bleeding site. This is an important investigation because preoperative localization of

bleeding site is mandatory to avoid the trauma when we are looking for a bleeding lesion in a friable and opaque organ like the liver.[12] Abdominal CT can show liver injuries that predispose to hemobilia and confirm hemobilia by showing accumulation of blood in the biliary tree and gallbladder. However, abdominal CT should not play a primary role in patients with suspected hemobilia when EGD is available. Otherwise, appropriate therapy may be delayed.[11] Yoshida et al reported that the initial diagnosis was made with angiography in 28% cases, endoscopy in 12%, intraoperatively in 34%, and with the noting of blood in various biliary drains in 12% cases. Anecdotal reports indicate that technetium-labeled erythrocyte scans and the presence of fecal occult blood occasionally support or suggest the diagnosis, but a reliance on these tests for diagnosis cannot be justified.[6] Although EGD, USG, CT, magnetic resonance imaging (MRI), and endoscopic retrograde cholangio-pancreatography (ERCP) may be used to obtain additional information when hemobilia is suspected, the accurate sites of bleeding cannot be located by such modalities and sometimes patients especially those in pre shock or shock status are intolerable to those examinations. Selective celial and hepatic arteriography as a rapid, safe, and accurate option is helpful to determine the bleeding site.[13]

The goals of treatment in hemobilia management are to stop the bleeding and to restore bile flow past clots. Some modalities to stop bleeding include angiography with embolization, surgical intervention, observation, and electrocoagulation or photo-coagulation. Surgical therapy may be most helpful in hemobilia cases after blunt liver trauma since debridement, drainage, and vessel ligation are important. Studies of liver healing in patients with substantial hepatic trauma would support the placement of drains to reduce the possibility of bile pooling. Selective hepatic artery ligation is a surgical manuver that may be useful in some hemobilia cases. Finally, surgical therapy should be considered as the treatment of choice when the underlying cause of hemobilia constitutes an independent indication for such treatment, such as cases associated with cholelithiasis, cholecystitis, or resectable neoplasms. However, an operation carries an obvious risk of morbidity.

Embolization is not without substantial risk. Hepatobiliary necrosis occurs in 6% cases, abscess formation in 9%, bleeding in 6%, and gallbladder fibrosis in 2% cases following arterial embolization.[6] The death rate for operations to treat hemobilia is about 20%, which it not only because the bleeding may be massive but also because the diagnosis and bleeding control is delayed by the necessity for organizing endoscopy, CT scanning and angiography.[12]

In a study of 15 patients with massive hemobilia, transarterial embolization, hepatic artery ligation and open drainage were effective as treatment of non-surgical and surgical procedures. However, the former two procedures might not be successful if sudden and severe hemobilia developed, or when an aberrant hepatic artery existed. Main hepatic artery should better be isolated before removal of the percutaneous transhepatic biliary drainage tube during operation.[14]

Xu et al conclude conclude that selective arteriography and transcatheter embolization could be one of the best diagnostic and treatment options, especially for those patients with suspected hemobilia after hepatobiliary surgery.[13] It is safe, less invasive and effective if performed in a targeted selective or superselective way. The effect of conservative treatment and traditional surgical methods such as hepatic lobectomy, ligation of the hepatic artery or branches and bile duct drainage is elusive for massive hemobilia, while giving a high mortality rate.[13]

The review from Southampton searched the English language literature and found 222 cases worldwide. Management was aimed at stopping bleeding and relieving biliary obstruction. In 43% cases, the management was conservative and 36% cases were managed by transarterial embolization (TAE). Surgery was indicated when laparotomy was performed for other reasons and for failed TAE. The mortality rate was 5%. They concluded that the incidence of iatrogenic hemobilia has risen considerably but the bleeding is often minor and can be managed conservatively. When more urgent intervention is required, TAE is usually the treatment of choice. There is no evidence that the conservative management of accidental liver trauma increases the risk of haemobilia.[15] In this case, surgery had been planned but patients and his family refused to go through the procedure because of financial problem.

CONCLUSION

This is a case of hemobilia which occurs 49-days after accidental hepatic trauma and twice perihepatic packing. Endoscopic diagnosis of hemobilia is made when there is bleeding exiting from the ampulla of Vater. Surgery had been planned but patient and his family had refused the procedure.

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