Case Report

Massive hemobilia in a cirrhosis patient after percutaneous liver biopsy

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ABSTRACT

Hemobilia is commonly seen in clinic after liver injury or other diseases, but it is uncommonly secondary to percutaneous liver biopsy, especially leading to significant hemorrhage. We reported a case that a patient with post hepatitis C cirrhosis has received percutaneous liver biopsy at our hospital. After that, the patient suffered from discontinuous abdominal pain and jaundice which were caused by the obstruction of blood clots due to hemobilia, but there was no performance of upper gastrointestinal bleeding. Not until the symptoms of hemorrhagic shock emerged did the diagnosis of hemobilia was confirmed. Urgently surgical exploration eliminated blood clots in biliary tract relieving biliary obstruction. And then the hemorrhage was restrained by selective transcatheter arterial embolization. Pseudoaneurysm and arterioportal fistula were also found by hepatic arteriography. We learn from this case that rational choice of treatments is of importance, whether surgery, arterial embolization, conservative therapy or the combination of them, which mainly rests on the situation.

Keywords: Hemobilia, Percutaneous liver biopsy, Complications, Arterial embolization, Surgical management

INTRODUCTION

Hemobilia means that blood presents abnormally in bile ducts through passages between vessels and adjacent biliary tracts. It’s usually secondary to biliary tract infection, biliary calculi, tumor or trauma (including iatrogenic trauma). Percutaneous Liver Biopsy (PLB) is a golden standard for diagnosing acute and chronic liver diseases.1 This invasive procedure may accompany with hemorrhage complications, of which hemobilia is a relatively rare type.2,3 It can be life-threatening once leading to massive bleeding and hypovolaemia. We reported a cirrhosis patient with hemobilia after PLB was successfully cured by surgical exploration and selective Transcatheter Arterial Embolization (TAE).

CASE REPORT

The thirty-eight years old man was admitted with abdominal distension and hepatalgia. He was diagnosed as post hepatitis C cirrhosis for positive hepatitis C antibody and liver cirrhosis showed by Computer Tomography (CT). The patient agreed to accept ultrasound-guided percutaneous liver biopsy to know the degree of cirrhosis on Jun. 24th, 2014. Before the biopsy, the patient’s blood routine, blood coagulation and liver function were all in normal range. The pathological diagnosis was moderate chronic hepatitis (G2 S3) and early cirrhosis.

Paroxysmal epigastric cramps attacked the patient after the procedure, and became more frequently on the third...
night when his sclera began to be yellow. The patient denied hematemesis, melena and fever all through. On physical examination, the major signs were moderate icteric sclera and epigastric tenderness which was absent during the intervals of abdominal pain. Laboratory analysis revealed obstructive jaundice (AST 77 U/L, AST 116 U/L, GGTP 572 U/L, total bilirubin 75.7 μmol/L, direct bilirubin 63.8 μmol/L). Serum levels of amylase and blood test were normal. Magnetic resonance cholangiopancreatography (MRCP) showed that an intrahepatic arteriovenous malformation (AVM) located in right posteriorinferior hepatic lobe; abnormal hepatic perfusion during arterial phase; cholecystolithiasis and cholecystitis (Figure 1). The patient suddenly turned pale and weak on the fifth day after PLB. There were obvious signs of hemorrhagic and infectious shock according to clinical parameters (heart rate increased; hemoglobin dropped from 107 g/L to 84 g/L within eight hours; white blood cell count rose to 14.69×10^7/L, and the percentage of neutrophils was 89.8%).

![Image](image1.png)

**Figure 1:** Magnetic resonance cholangiopancreatography (MRCP). A: Coronal slice image of enhanced MRI on the 5th day after liver biopsy showed the arteriovenous malformation (AVM) locating at the puncture site; B: Contrast-enhanced MRI image on the 5th day after liver biopsy shows the hemocholecyst and hemorrhage in liver.

Biliary duct exploration was performed urgently, in which we saw massive blood clots casting filling up the gallbladder, right hepatic duct and choledoch. Operators removed the gallbladder and swept away blood clots. Stones and the source of hemorrhage haven’t been found. A T-tube was placed in the common bile duct at the end of surgery. The patient was sent to ICU after surgery where 600 ml red blood cells suspension was transfused. Bloody fluid was drained out from the T tube discontinuously on the first postoperative day. We speculated the bleeding didn’t subside, so hepatic angiography and TAE were conducted the next day. A pseudoaneurysm (about 3 mm in diameter) and a low shunt arterioportal fistula that located in the sixth hepatic segment were obviously showed by angiography, which was the source of bleeding (Figure 2). Gelfoam slurry and spring coils were used to stem the feeding artery. Then Digital Subtraction Angiography (DSA) verified the completion of embolization. The interventional treatment achieved hemostasis successfully: the T tube stopped draining bloody fluid; biochemical indicators and blood routine remained stable. A month later, the patient recovered well and the T tube was pulled out after cholangiography.

![Image](image2.png)

**Figure 2:** Hepatic angiography. A: the angiography showed the artery in liver and a pseudoaneurysm was corresponding to the position of puncture site; B: the angiography showed the arteriovenous fistula in the right liver; C: There was no obvious arteriovenous fistula after the hepatic artery. The spring coil completely clogged the fistula artery.

**DISCUSSION**

Hemobilia generally emerges in liver injury or after biliary tract infection as a kind of bleeding complication, but seldom after PLB. A large series of liver biopsy complications had been reported by Piccinino et al., in which hemobilia occurred in only 4 of 68276 biopsies. Though the pathogenesis of hemobilia is simple and well-studied, the risk of deterioration and fatality can’t be underestimated in clinic.

Fibrotic liver tissues in patients with cirrhosis may lose a kind of squeezing action making it hard to stop bleeding spontaneously. For cirrhosis patients, poor blood coagulation function also makes massive hemobilia more likely to happen. The patient described above had obvious intermittent epigastrum cramp after PLB, which was once misinterpreted as gastrointestinal cramp. The intermittent epigastrum cramp might be due to arterial steal phenomenon imitating intestinal angina in the described case. We didn’t suspect it of hemobilia following liver biopsy until the AVM was found by MRCP and the patient performed as early stage of hemorrhagic and infectious shock. Some studies suggested that the symptoms of hemobilia appear almost five days after biopsy while most complications after PLB occur within 24 hours. Atypical and tardive symptoms as well as inconspicuous imaging tests make it hard to diagnose hemobilia promptly and correctly.

In the process of blood loss and accumulating in bile duct, hypovolemia may develop into hemorrhagic shock, and the high biliary pressure can cause cholangitis and infectious shock. So the foremost therapeutic schedule of hemobilia should be conventional treatments such as fluid resuscitation, instant laboratory analyses and blood transfusion. Relieving biliary pressure and
obstruction by endoscopic or percutaneous biliary decompression is equally important to prevent from acute pancreatitis and cholangitis. If the patient’s condition deteriorates rapidly with conservative treatment, immediate surgical management will be imperative. Surgical indications also include failure or complications of medical treatment, arterial embolization and biliary decompression. More importantly, surgical management should be decided cautiously when patient can’t tolerate the surgery, the rest of liver is unable to compensate or other serious complications happen. The emergency surgery in our case was life-saving for the shock patient to eliminate blood clots in bile tract, and it confirmed the diagnosis simultaneously. Ligating the right hepatic artery in the surgery had been considered, but we were afraid of postoperative hepatic failure in this cirrhosis patient. While there was still a small amount of blood diffused from biliary tract intermittently after surgery, TAE was carried out immediately.

TAE is a safe and effective method of hemostasis for solid organ hemorrhage, and also the optimal therapy for arteriovenous fistula and arteriobiliary fistula. Comparing to surgery management, selective arterial embolization can reduce the harm for hepatic function. Gelfoam slurry, spring coil and polyvinyl alcohol particles are commonly used as embolic materials. Spring coil is more preferred in arteriovenous fistula or arteriobiliary fistula. These materials can be used alone or in combination for better hemostatic effect in different situations. Either surgery or artery embolization was enough to treat patients with massive hemobilia in most cases. Murugesan et al. concluded their algorithm of diagnosis and therapeutics for patients suspected of hemobilia. We hold the same view that conservative treatment, surgical management and interventional therapy shouldn’t be regarded as independent options, as these methods have their definitive roles in different situations, sometimes combination therapy is required.

In conclusion, if an invasive operation involving liver or biliary tract is inevitable, appropriate patient selection, preprocedure evaluation, and postprocedure observation should be attached importance. Prompt and definitive diagnosis for hemorrhage complications is the pivotal issue. Purposeful treatment plan should be made as soon as possible. It should be extra vigilant in the clinic that the symptoms of hemobilia may not occur right after trauma or invasive procedures, and sometimes it can be changeable or asymptomatic.

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