Hemobilia Causing Acute Biliary Pancreatitis after Percutaneous Liver Biopsy

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INTRODUCTION

Hemobilia caused by iatrogenic causes such as liver biopsy occurs more frequently with the popularity of invasive procedure. However, pancreatitis associated with hemobilia is unusual because the fresh bile perfusion keeps the bile duct not to be obstructed by blood clots. We report the case of a 63-yr old woman who presented with acute biliary pancreatitis 4 days later after liver biopsy. On duodenoscopic examination, blood clots and fresh blood coming from ampulla were observed. According to literature, two cases of pancreatitis associated with hemobilia induced by liver biopsy were reported and managed with arterial embolization. However, our patient was dramatically improved only with biliary drainage. A case of biliary pancreatitis associated with hemobilia induced by liver biopsy is presented.
CASE REPORT

A 63-year-old woman had been admitted for percutaneous liver biopsy due to $3 \times 4$ cm hepatic mass of left lobe. Laboratory findings were within the normal range (Table 1). She undertaken sonography-guided liver biopsy with the 18 gauzed needle and confirmed as angiomyolipoma. She was discharged and followed-up as an outpatient. Four days later after liver biopsy, she admitted again because of nausea, vomiting and boring epigastric pain. On physical examination, her vital sign was stable but she complained of severe tenderness over the epigastrium. At that morning, she passed voluminous tarry stool. Laboratory studies on second admission were as follows: WBC 10,000/mm$^3$, Hb 12.9 g/dl, platelet 201,000/mm$^3$, PT 1.12INR, aPTT 39.7 sec, AST 267 U/L, ALT 138 U/L, bilirubin 2.1 mg/dl, alkaline phosphatase 337 U/L, r-GT 271 U/L, amylase 1,052 U/L and lipase 1,594 U/L (Table 1). Gallbladder, which was normal before liver biopsy, was distended and showed an echogenic polyoid lesion on abdominal ultrasonogram. Pancreas was mildly enlarged and edematous. An abdominal CT scan of pre-enhanced phase showed round high attenuated lesion within the gallbladder. It was not noted on CT scan of the first admission and considered as blood clots (Fig. 1). ERCP was undertaken to find the cause of tarry stool and biliary pancreatitis. On duodenoscopic examination, blood coming from the ampullary orifice and tubular shaped blood clots just near the ampulla were found (Fig. 2). Pancreatography was normal but cholangiography demonstrated a round polyoid filling defect within the gallbladder (Fig. 3). Endoscopic sphincterotomy and nasobiliary drainage were performed for drainage and decompression of bile duct. One day after ERCP, acute cholecystitis has developed. She presented high

Table 1. Laboratory findings of the patient at the time of first admission for liver biopsy and second admission for acute pancreatitis

<table>
<thead>
<tr>
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<th>First admission</th>
<th>Second admission</th>
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<tbody>
<tr>
<td>WBC (mm$^3$)</td>
<td>5,500</td>
<td>10,000</td>
</tr>
<tr>
<td>Hb (g/dl)</td>
<td>13.4</td>
<td>12.9</td>
</tr>
<tr>
<td>Platelete (mm$^3$)</td>
<td>201,000</td>
<td>210,000</td>
</tr>
<tr>
<td>GOT/GPT (U/L)</td>
<td>43/35</td>
<td>267/138</td>
</tr>
<tr>
<td>ALP/r-GT (U/L)</td>
<td>154/36</td>
<td>337/271</td>
</tr>
<tr>
<td>Bilirubin (mg/dl)</td>
<td>0.6</td>
<td>2.8</td>
</tr>
<tr>
<td>Amylase/Lipase (U/L)</td>
<td>64/71</td>
<td>1,052/1,594</td>
</tr>
<tr>
<td>Protein/Albumin (g/dl)</td>
<td>7.3/3.5</td>
<td>7.1/3.7</td>
</tr>
<tr>
<td>PT/aPTT (INR/sec)</td>
<td>1.02/39.2</td>
<td>1.12/39.7</td>
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Fig. 1. CT finding at second admission shows high attenuated round lesion within the gallbladder (left) in contrast with normal looking gallbladder (right) at first admission.
fever over 39°C and intense right upper quadrant pain with Murphy sign. Emergent percutaneous cholecystostomy was undertaken. We sucked blood clots up from the gallbladder via percutaneous cholecystostomy tube (Fig. 4) and then initial polypoid lesion of the gallbladder was disappeared on repeated cholecystogram (Fig. 3). Blood chemistry returned to normal and drained bile became clear. She was discharged after removal of drainage tube and has continued to be asymptomatic.

**DISCUSSION**

The term, hemobilia, was originally introduced to designate bleeding into the biliary tract after trauma to the liver, but it is now used to denote bleeding into the bile duct from any causes. Diagnosis of hemobilia can be made by endoscopy from the appearance
of blood coming from the ampulla of Vater. Patients classically present with melena accompanied by biliary pain and jaundice. Strikingly, our patient presented with acute biliary pancreatitis suggested by boring epigastric pain, abnormal liver function test and marked elevation of serum amylase and lipase level. The occurrence of acute pancreatitis following hemobilia was first mentioned by Dean. A review of literature reveals eight well-documented pancreatitis associated with hemobilia. Only two cases out of them occurred after liver biopsy. Usually, fresh bile perfusion and intrinsic fibrinolytic capacity of bile may spontaneously dissolve blood clots in bile duct. If bile flow is not enough to wash the bile duct, however, persistence of blood clots may cause acute pancreatitis. In those cases, the pancreatitis is thought to be induced by the same manner in gallstone pancreatitis.

Previous reported cases of pancreatitis associated with hemobilia were improved after invasive treatment of angiographic gelfoam embolization. In our case, blood loss was not so serious as to require transfusion because hemobilia was originated from minimally damaged hepatic mass, not from arterioportal fistula or pseudoaneurysm. Therefore, she could be managed only with biliary drainage procedure.

In summary, for those patients with acute pancreatitis after liver biopsy, biliary pancreatitis associated with hemobilia should be considered even though there is no evidence of gallstone or sludge. Biliary drainage procedure may be a simple and effective treatment modality if the amount of bleeding is not profuse.

REFERENCES