CASE REPORT

Portal Vein Occlusion after Biliary Metal Stent Placement in Hilar Cholangiocarcinoma

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Acute symptomatic portal vein obstruction related to biliary stenting is an extremely rare but life-threatening complication. This usually occurs in patients with either tumor invasion into the portal vein or pre-existing portal vein thrombus. Therefore, the portal vein should be carefully evaluated before placing a biliary metallic stent in such patients. We describe a case of acute portal vein obstruction after placing metallic biliary stents in a patient with a periductal-infiltrating type of hilar cholangiocarcinoma. (*Gut and Liver* 2008;2: 47-50)

Key Words: Portal vein; Stent; Cholangiocarcinoma

INTRODUCTION

Portal venous hypertension caused by extrahepatic obstruction comprises 5-10% of all cases of portal hypertension.¹ Neoplasms, such as hepatocellular carcinoma, pancreatic cancer, and bile duct cancer, are responsible for 15-24% of the cases with extrahepatic portal venous occlusion.¹⁻³ Biliary stent placement in these patients may further aggravate portal vein obstruction.⁴ We present an unusual case of fatal acute portal vein occlusion after biliary metallic stent insertion in a patient with unresectable extrahepatic bile duct cancer.

CASE REPORT

A 62-year-old man was admitted to the hospital with a 2-week history of jaundice. He lost 4 kg body weight in 10 days prior to the hospitalization. The patient reported of having intermittent right upper quadrant pain, anorex-

ia, and whole body itching, but denied having fever, chills, and myalgias. His past medical history included diabetes mellitus for which he had been taking oral medication for 12 months. Physical examination on admission showed mild obesity without acute distress. Blood pressure was 130/ 70 mmHg, pulse 76/min, and temperature 36.4°C. His abdomen was soft with normal bowel sounds. He had mild right upper quadrant tenderness without guarding or rebound tenderness. There was no palpable spleen, abdominal mass, or ascites. Admission laboratory studies revealed a hematocrit of 38%, a white blood cell count of 5,700/mm³, and a platelet count of 234,000/mm³. Liver test showed as follows: total bilirubin 12.4 mg/dL, direct bilirubin 8.4 mg/dL, alkaline phosphatase 777 U/L, γ -glutamyl transferase 206 IU/L, AST 38 U/L, ALT 23 U/L, total protein 6.8 g/dL, and albumin 3.6 g/dL. Serum electrolytes, coagulation, and hepatitis studies were normal. Tumor markers, CEA and CA19-9, were elevated to 12.8 ng/mL and 155.9 U/mL, respectively. No abnormality was seen on upper gastrointestinal endoscopy and colonoscopy. A computed tomography (CT) of the abdomen on the second hospital day revealed a long segmental wall thickening of the bile duct at level of both hepatic ducts and common bile duct with bilateral intrahepatic ductal dilatation, which suggested either an inflammatory process or malignancy (Fig. 1A). A subsequent percutaneous transhepatic cholangiogram (PTC) showed the possibility of periductal infiltrating hilar cholangiocarcinoma (Fig. 2). Brush cytology taken during endoscopic retrograde cholangiopancreatography (ERCP) confirmed adenocarcinoma. On the 13th hospital day, self-expandable Zilver[®] biliary stents (Wilson-Cook, Bloo-

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Fig. 1. Preprocedural CT scan taken on the second hospital day. (A) Wall thickening of both hepatic ducts and the common bile duct is evident with bilateral intrahepatic ductal dilatations (black arrow). (B) A luminal protruding lesion suggesting tumor invasion is evident in the middle portion of the portal vein (black arrow).

Fig. 2. Transhepatic cholangiogram showing long-segment narrowing with slight upstream dilatation, which is consistent with an infiltrating cholangiocarcinoma and a Bismuth type IV hilar stricture.

Fig. 3. Contrast-enhanced CT scan at 21 days after placement of biliary stents revealing an asymmetric oval filling defect suggesting portal vein thrombosis (black arrow).

mington, IN, USA), 10×80 mm in left hepatic duct and 8×40 mm in right hepatic duct, were successfully placed through the preformed percutaneous transhepatic biliary drainage route after balloon dilatation up to 8 mm. There were no procedure-related complications. After the placing of the biliary stents, he complained of right upper quadrant pain, quadrant pain, which subsided with oral non-opioid analgesics. On the 28th hospital day, 15 days after placement of biliary stents, the patient only complained of nonspecific abdominal discomfort. He had a normal serum ALT of 31 IU/L, total bilirubin 2.2 mg/dL, but persistently elevated alkaline phosphatase of 664 IU/L.

However, six days later, he was re-admitted with severe

epigastric pain that was not relieved by intravenous analgesics and rectal enema. A follow-up CT revealed a filling defect in portal vein as well as newly developed ascites, diffusely thickened gastric wall and portal vein obstruction (Fig. 3). Endoscopic findings showed newly developed esophageal varices and congested gastric mucosa. A direct splenoportal venogram showed complete occlusion of the main portal vein (Fig. 4). A subsequent whole body PET-CT scan revealed multiple mediastinal and intra-abdominal nodal metastases (Fig. 5). A decision as to treat him with intravenous heparin was made as a result of group discussion with an interventional radiologist and a general surgeon. Although heparin was administered for 11 days, the Doppler sonogram did not show any im-

Fig. 4. Percutaneous transabdominal splenoportogram obtained 22 days after placement of metallic stents shows contrast medium filling the splenic and inferior mesenteric veins but not the main portal vein. Y-shaped metallic stents inserted through the left hepatic duct are evident on the left side.

provement, and the patient expired from sepsis. Hypercoagulation studies for levels of homocysteine, fibrinogen, prothrombin time, aPTT, protein C and protein S, antithrombin III activity, and lupus anticoagulant showed results within normal range.

DISCUSSION

Portal vein obstruction can result from thrombosis as well as invasion or constriction by a malignant tumor.⁵⁻⁷ Portal vein involvement by neoplasm is classified as either extrinsic portal vein occlusion or intraluminal tumor thrombus. The former is usually caused by pancreatic cancer, gallbladder cancer, and metastatic lymph nodes while the latter is usually caused by hepatocellular carcinoma and some alpha-feto protein-producing gastric cancers.⁶ In addition, acute portal vein thrombosis may also occur as a complication of post-ERCP pancreatitis in relation to internal drainage for malignant biliary obstruction.⁸

In our case, metallic stents placed in the biliary tract may have contributed to portal vein obstruction and/or thrombosis. Since it had been only 3 weeks after the biliary stenting procedure, it was unlikely that the progression of the tumor caused the complete obstruction during the observational period. However, biliary stenting alone is not believed to produce symptomatic portal vein obstruction in the absence of preformed thrombosis, tumor invasion or compression, or procedure-related pan-

Fig. 5. PET-CT scan taken after placement of metallic stents, showing widespread metastasis.

creatitis,⁵ and it can be suggested that clinical aggravation might have attributed to this result in the present case. To clarify the cause of portal vein obstruction in our patient, we re-evaluated the initial CT and found an intra-luminal protruding lesion in the main portal vein, either a preformed thrombus or vascular tumor invasion, which has been overlooked in the first place (Fig. 1B).

A pathological study showed that 6.8% of the surgical specimens of cholangiocarcinoma showed portal vein or hepatic vein invasion.⁹ Although there have been no studies on the prevalence of portal vein invasion or thrombus in patients with unresectable tumors, several case reports have demonstrated the efficacy of portal vein stenting in patients with malignant portal vein stenosis.^{10,11} A case report described an advanced gallbladder carcinoma treated with combined biliary and portal venous stent placement.¹⁰ The clinical usefulness of these procedures has also been demonstrated in a Japanese study that included 13 cases of malignant pancreatic or biliary neoplasm.¹¹ However, acute symptomatic portal obstruction following biliary stent placement is very unusual, despite the fact that internal drainage employing metallic stents has been widely used for malignant biliary obstruction. A further case of portal vein obstruction was described as an acute complication after the placement of two metallic biliary stents in a patient with malignant hilar obstruction.⁴ Although the portal vein obstruction was successfully treated with subsequent portal venous stent placement, pre-existing portal vein tumor invasion was overlooked as happened in our case.

Although the main cause of portal vein obstruction is

not clear, it is reasonable to think that vascular tumor invasion was followed by further secondary thrombus formation for the following reasons; first, the vascular and biliary lesion positioned adjacent to each other, and secondly, the portal vein lesion developed a thrombus-like appearance after intervention. Initial and follow up CT findings of asymmetric filling defect in the main portal vein support this assumption. Nonetheless, it is still unclear whether the aggravation of portal vein obstruction has occurred as a consequence of balloon dilatation of bile duct or of the metal stent itself. Microperforation of tumor mass after balloon dilatation might have led to further formation of thrombus. We decided to perform systemic anticoagulation therapy because we estimated the duration of portal venous thrombosis to be more than 2 weeks, in which case local thrombolytic treatment would be of little benefit.

As a retrospective speculation, a question can be raised why double plastic stents were not inserted at first instead of the bilateral metallic stents through PTC. We suspected that the patient's life expectancy could be more than 6 months at the time of metallic stent insertion. Moreover, we did not anticipate biliary stents to obstruct the portal vein at the time of the procedure. Considering the PET-CT findings that showed widespread metastases in our patient, a more proper palliative option for biliary drainage should have been chosen.

The present case highlights that metallic stent insertion in a patient with diffuse infiltrative bile duct carcinoma may compress the portal vein and lead to serious obstructive vascular complications, especially when there is pre-existing portal vein invasion or thrombus formation. Recognition of these with CT or endoscopic ultrasonogram prior to the stenting can be helpful in determining the risks involved with the biliary drainage procedure.

REFERENCES

1. Macpherson AL. Portal hypertension due to extrahepatic portal venous obstruction: a review of 40 cases. *J R Coll Surg Edinb* 1984;29:4-10.
2. Cohen J, Edelman RR, Chopra S. Portal vein thrombosis: a review. *Am J Med* 1992;92:173-182.
3. Sugiura N, Matsutani S, Ohto M, et al. Extrahepatic portal vein obstruction in adults detected by ultrasound with frequent lack of portal hypertension signs. *J Gastroenterol Hepatol* 1993;8:161-167.
4. Peynircioglu B, Cho KJ, Cwikiel W. Portal hypertension and obstructive jaundice after hepatic interventions: reports of two unusual complications. *J Vasc Interv Radiol* 2007;18:567-571.
5. Valla DC, Condat B. Portal vein thrombosis in adults: Pathophysiology, pathogenesis and management. *J Hepatol* 2000;32:865-871.
6. Tanaka A, Takeda R, Mukaiharu S, et al. Tumor thrombi in the portal vein system originating from gastrointestinal tract cancer. *J Gastroenterol* 2002;37:220-228.
7. Sobhonslidsuk A, Reddy R. Portal vein thrombosis: a concise review. *Am J Gastroenterology* 2002;97:535-541.
8. Vimalraj V, Jeswanth S, Kannan DG, Krishnan CS, Surendran R. Acute portal vein thrombosis and massive necrosis of the liver. An unusual complication after stenting for chronic pancreatitis. *JOP* 2006;7:660-664.
9. Koh KC, Lee H, Choi MS, et al. Clinicopathologic features and prognosis of combined hepatocellular cholangiocarcinoma. *Am J Surg* 2005;189:120-125.
10. Tanaka J, Andoh H, Yoshioka M, Furuya T, Asanuma Y, Koyama K. Palliative treatment with metallic stents for unresectable gallbladder carcinoma involving the portal vein and bile duct. *J Hepatobiliary Pancreat Surg* 2000;7:331-335.
11. Yamakado K, Nakatsuka A, Tanaka N, et al. Portal venous stent placement in patients with pancreatic and biliary neoplasms invading portal veins and causing portal hypertension: initial experience. *Radiology* 2001;220:150-156.