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COVERED METALLIC STENT FOR ISCHEMIC HILAR BILIARY STRICTURE

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ABSTRACT

Compared with surgery, endoscopic treatment is safe and highly effective for a postoperative hilar benign bile duct stricture (BDS). However, the long-term outcome of conventional placement of a single biliary stent for hilar benign BDS is generally poor. Although the placement of multiple biliary stents is preferred, multiple stenting in a BDS is difficult. Alternatively, single or multiple stent placement above the papilla ("inside stent") or fully covered self-expandable metallic stents (SEMSs) are feasible approaches for benign BDS. Nevertheless, controversy remains regarding whether and how to perform endoscopic biliary drainage for a hilar benign BDS. In patients with hilar benign BDS, endoscopic biliary drainage can be performed by placing conventional plastic stents across the papilla, plastic stents above the papilla, or fully covered SEMSs. Individualized treatment should be considered. We report the placement a fully-covered SEMS for a hilar benign biliary stricture after extended left hepatectomy.

Key Words: hilar biliary stricture, ischemic biliary stricture, ischemic biliary injury, metallic stent, transcatheter arterial chemoembolization

INTRODUCTION

Benign hilar bile duct strictures (BDSs) remain not only a diagnostic challenge but also a therapeutic challenge for which a multidisciplinary approach is often necessary. Various methods have been used to treat benign hilar BDS. For benign postoperative hilar BDS, surgical repair can be difficult, requires specific skills and experience, and is associated with worse short- and long-term outcomes compared with the repair of lesions below the hepatic confluence. An endoscopic approach is safe and effective for postoperative hilar BDS.[1] Although single stent placement is technically easy, proximal strictures are more difficult to treat and more frequently associated with recurrence.[2] Multiple stent placement is now the preferred technique for hilar BDSs[3] in many countries. Plastic stents have problems with patency. Self-expandable metallic stents (SEMSs), particularly fully covered SEMSs, have a longer patency than plastic stents.[4] Uncovered SEMSs should be avoided for hilar BDS because of tissue ingrowth and the impossibility of removal. In contrast, fully covered SEMS can be removed. However, covered SEMSs for hilar BDS is discouraged in cases exhibiting occlusion of side branches of the intrahepatic or contralateral hepatic bile ducts. Here, we present a patient with a hilar BDS caused by ischemic injury that was treated with covered SEMSs and complicated by a liver abscess.

CASE REPORT

In December 2010, a 58-year-old man was hospitalized because of fever, abdominal pain, and jaundice that had persisted for 7 days. He had been treated with extended left hepatectomy for hepatitis B virus-related hepatocellular carcinoma (HCC) with portal vein tumor thrombus in 1997. After surgery, he was also treated with repeated transarterial chemoembolization (TACE). His abdomen was soft, and no mass was palpable. Laboratory data showed remarkable liver dysfunction. Abdominal ultrasonography (US) revealed that the intrahepatic bile ducts were mildly dilated (largest diameter, 3 mm); the residual liver showed no evidence of HCC. Enhanced computed tomography (CT) showed mild dilatation of the intrahepatic bile duct and a soft tissue density in the hilum of the bile duct (Fig. 1). No portal lymphadenopathy was detected in the US or CT studies. Although a malignant hilar biliary stricture could not be completely ruled out, laboratory data and imaging studies suggested ischemic biliary strictures. A short stricture at the hilum of the bile duct was confirmed during endoscopic retrograde cholangiopancreatography (ERCP), and the guidewire could pass through the stricture (Fig. 2). However, selective biliary stenting through the stricture was impossible because of its severity. He rejected percutaneous transhepatic biliary drainage and was treated with antibiotic therapy only. A granulomatous tissue taken

from the stricture was proven histologically.

Seven days after ERCP, he was hospitalized in our department because of recurrent cholangitis. Urgent ERCP revealed a severe hilar biliary structure. Although a 0.035-inch guidewire was successfully advanced across the stricture, a tapered ERCP catheter or wire-guided balloon catheter could not pass. We decided to use a Soehendra biliary dilation catheter (SBDC-6, -7 and -8.5; Wilson-Cook, Winston-Salem, NC, USA) for step-up dilation (Fig. 3). The dilation catheter was gently and carefully advanced under endoscopic and fluoroscopic guidance. He developed hemobilia in the intrahepatic bile ducts (Fig. 4) after step-up dilation. Various stents are available for this type of procedure (nasobiliary stents, single/multiple conventional plastic stents, and inside plastic stents); we selected a fully covered SEMS (WallFlex^{TM} stent; Boston Scientific, Tokyo, Japan) for this severe stricture complicated with hemobilia, for fear of early obstruction of plastic stents and due to the expectation of hemostasis in a single session (Fig. 5a). Immediately after SEMS placement, we observed the elimination of contaminated bile and blood through the SEMS (Fig. 5b). These findings improved spontaneously (Fig. 5c). Although he experienced moderate abdominal pain based on dilation of the SEMS, we treated him conservatively with a pain reliever until the following day. His cholangitis recovered immediately. Unfortunately, he developed

liver abscesses 2 weeks after SEMS placement. CT revealed a low-density area in segment VIII and a low-density area with air density in segment VI (Fig. 6). He underwent successful percutaneous drainage of the abscess. Two months later, ERCP showed communication between the liver abscess and the intrahepatic bile ducts (Fig. 7). An abdominal angiography revealed markedly decreased portal vein blood flow derived from TACE. Arterial flow to the remnant liver was supplied by only the subphrenic and intercostal arteries (Fig. 8). We ultimately removed the SEMS in consideration of reflux cholangitis. During percutaneous transhepatic drainage at the 12-month follow-up visit, there was no clinical evidence of liver abscess recurrence. However, severe ischemic hilar biliary structure was not ameliorated.

DISCUSSION

Ischemic hilar BDS after TACE is a very rare but serious complication [5-8]. It has been suggested that repeated ischemic injury from prolonged embolization of the biliary vascular plexuses may result in ductal stricture.[6-8] Pathologic features of a narrowed bile duct after repeated TACE consist of dense and intense fibrosis of an extensive periductal area associated with denudation of the epithelial layer, with relatively few signs of inflammation.[6] In the context of HCC, new-onset BDS is usually due to

mechanical compression by the tumor or portal lymphadenopathy. In our case, tumor and portal lymphadenopathy could not be detected by US or CT, occlusion of bilateral hepatic arteries was proven on angiography, and a biopsy specimen showed granulomatous tissue. Therefore, our patient is a typical case of ischemic injury from repeated TACE.

For postoperative benign hilar BDSs, surgical repair can be difficult and requires specific skills and experience.[1] However, the prognosis of endoscopic therapy as an alternative in patients with benign hilar BDS is poor, with success rates of 25% or lower.[2,3] In general, the outcome of endoscopic therapy for benign hilar BDSs depends on the number of stents,[3,10,11] but multiple stenting for BDSs is sometimes difficult. Fully covered SEMS placement can preserve a large diameter, although occlusion of intrahepatic/contralateral hepatic bile ducts is possible. Therefore, it is recommended that a fully covered SEMS be positioned at least 2 cm below the level of the hilar bile duct.[12-14] In our case, we chose a fully covered SEMS for BDS despite its position 1 cm below the level of the hilar bile duct, across the papilla of Vater, because he had been treated with an extended left hepatectomy. With this procedure, a covered SEMS is effective for BDS. Unfortunately, this procedure is not safe for ischemic liver because of the risk for reflux cholangitis and secondary liver abscess. An endoscopic therapeutic

strategy was difficult in this case; as a result, transhepatic drainage was effective. We should exercise caution regarding biliary stenting for ischemic liver and ischemia-induced hilar BDS, considering the peculiarities of this case. Preservation of the function of the sphincter of Oddi as a bacteriological barrier may contribute to prevention of reflux colonization of bacteria at the inner lumen of the stent. Thus, we should consider that a BDS treated with single/multiple plastic stent(s) or a metallic stent across the papilla of Vater presents a risk for reflux cholangitis,[15] when the BDS has been caused by ischemic change.

In an animal study, Geoghegan *et al*.[16] reported that stent placement above the intact sphincter of Oddi ("inside stent") is useful for prolongation of stent patency. Uchida *et al*.[17] supported these findings in a clinical study. Pedersen *et al*.[18] conducted a randomized controlled trial of stent placement above and across the sphincter of Oddi in patients with malignant bile duct stricture. They reported no significant difference between stents placed above and across the sphincter of Oddi, because there was a greater tendency for stent dislocation in the "inside stent" group. However, they concluded that the patency period might be increased if the migration of stents placed inside the common bile duct could be avoided. Although migration and removal of the inside stent may be major problems, we prefer inside biliary stenting for ischemic liver

and BDS. Recently, several investigators have reported the feasibility of fully covered SEMS for hilar BDS. Interestingly, Tee *et al.*[19] and Hu *et al.*[20] described a prototype short, removable, fully covered SEMS with a retrieval suture for post-liver transplantation. Poley *et al.*[21] showed the efficacy of a conventional fully covered SEMS in combination with a contralateral plastic stent for postoperative injury and hilar BDS induced by liver transplantation. Although short, fully covered SEMSs are not at present commercially available in Japan, the most suitable stenting in this case was a short fully covered SEMS above the papilla, which has the possibility of removal. Further studies are needed to demonstrate the long-term safety of biliary stenting with an inside SEMS for benign hilar BDS.

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Figure legends

Fig. 1. Abdominal computed tomography (CT) image.

CT showing mild dilatation of intrahepatic bile ducts and soft tissue density in the hilum of the bile duct.

Fig. 2. Endoscopic retrograde cholangiographic image.

Endoscopic retrograde cholangiography showing the hilar biliary stricture.

Fig. 3. Endoscopic retrograde cholangiographic and duodenoscopic image during biliary dilation.

A Soehendra biliary dilation catheter was gently and carefully advanced to the stricture of the bile duct of the hilum.

Fig. 4. Endoscopic retrograde cholangiographic image after biliary dilation.

Endoscopic retrograde cholangiography showing the hemobilia in the intrahepatic bile duct above the stricture.

Fig. 5. Endoscopic retrograde cholangiographic and duodenoscopic image after biliary stenting.

a: Endoscopic retrograde cholangiography and duodenoscopy showing that a fully covered metallic stent was placed successfully.

b: Duodenoscopy showing elimination of contaminated bile and blood through the fully

covered metallic stent.

c: Duodenoscopy showing disappearance of contaminated bile and blood.

Fig. 6. Abdominal CT image, 2 weeks after placement of a fully covered metallic stent.

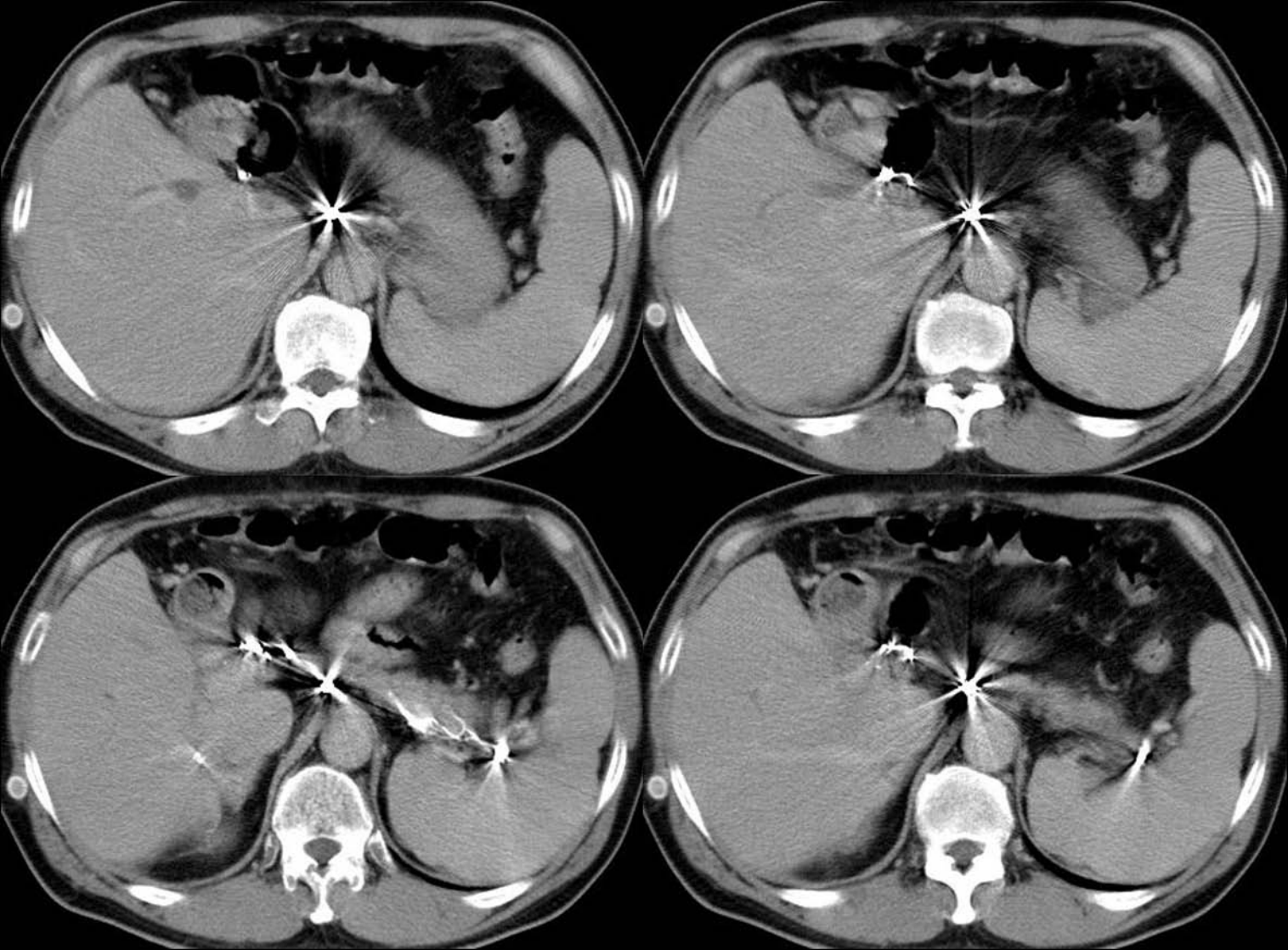
CT showing a hepatic abscess in segments VI and VIII of the liver.

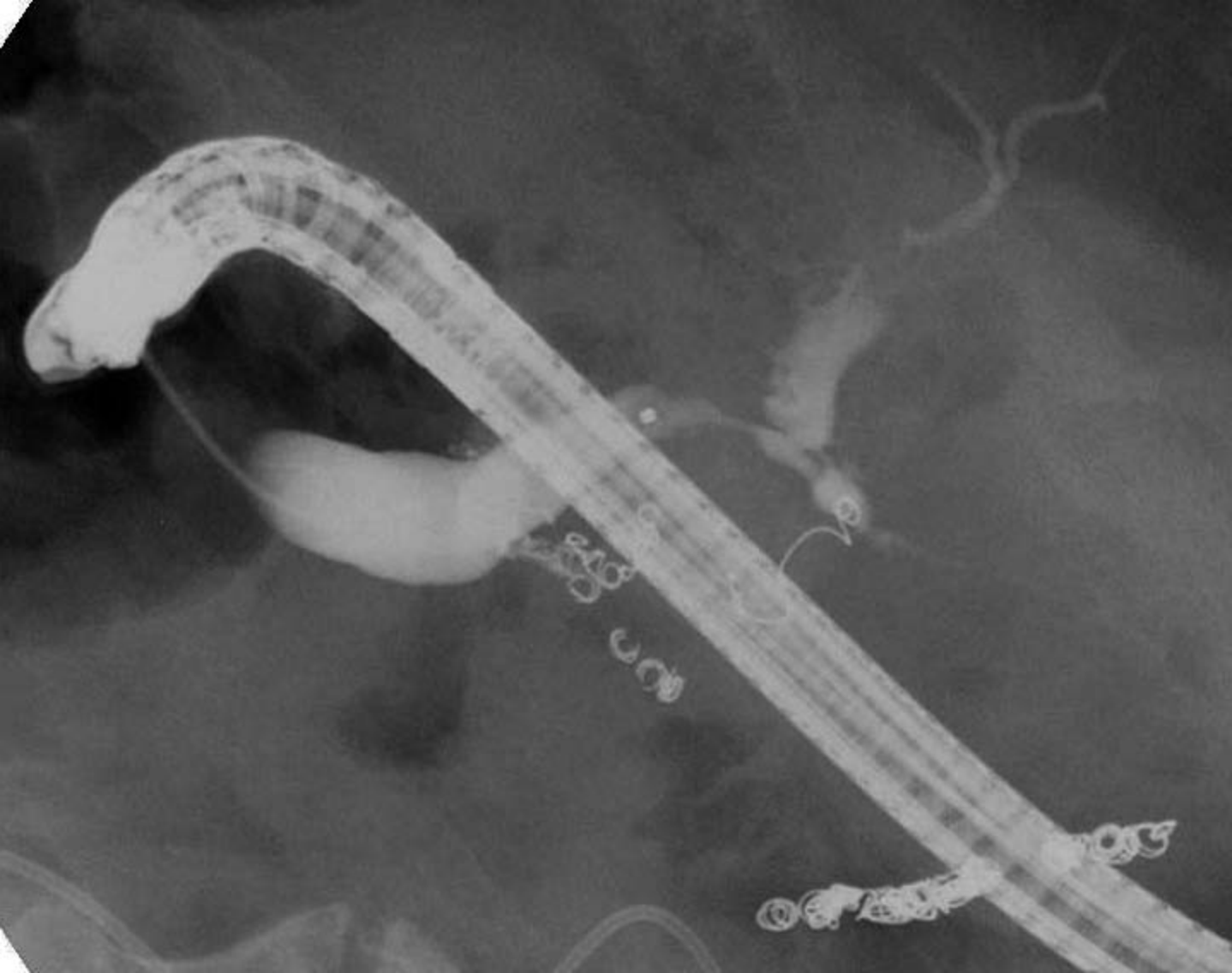
Fig. 7. Endoscopic retrograde cholangiographic image.

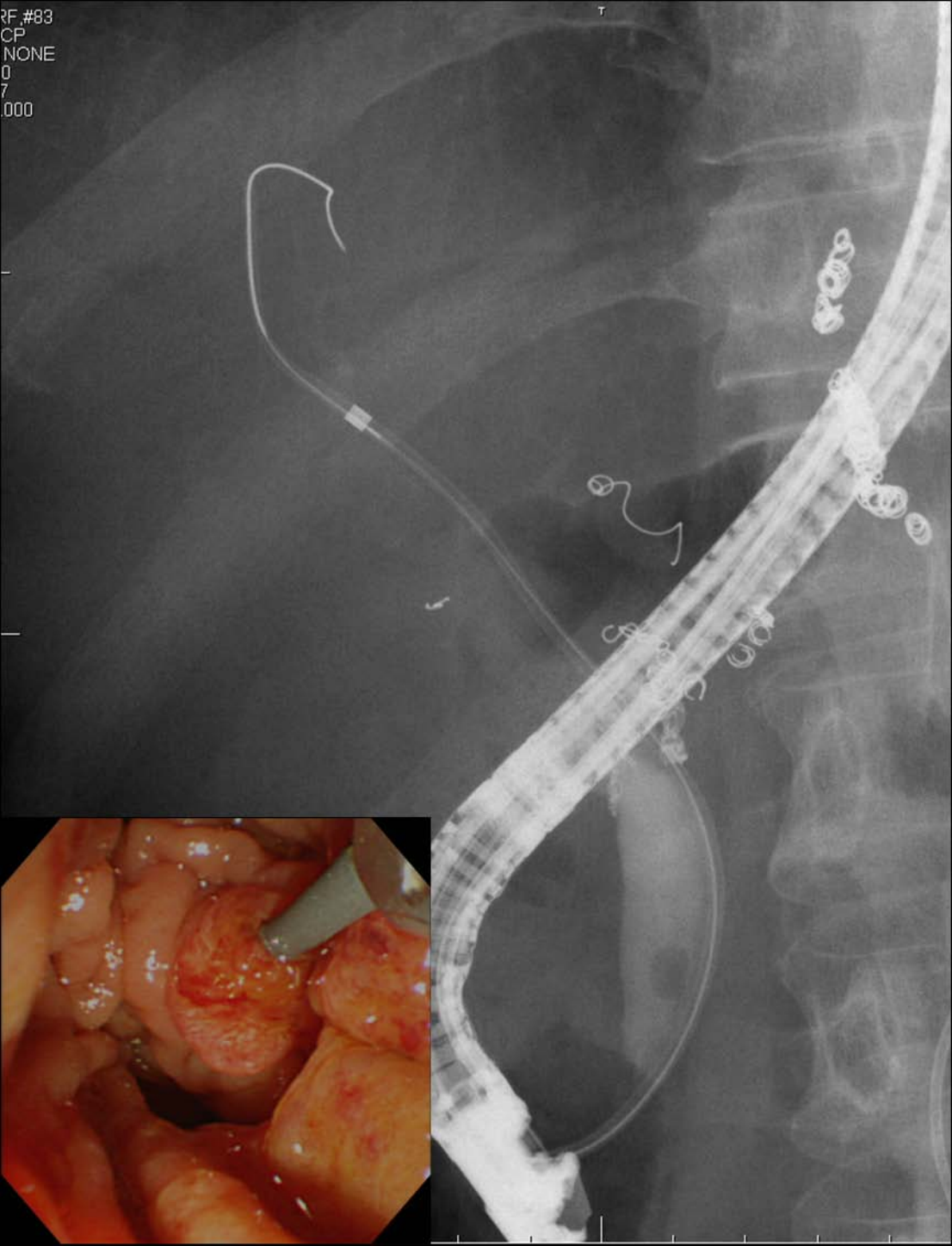
Endoscopic retrograde cholangiography showing the communication between the liver abscess and the intrahepatic bile ducts.

Fig. 8. Abdominal angiography image.

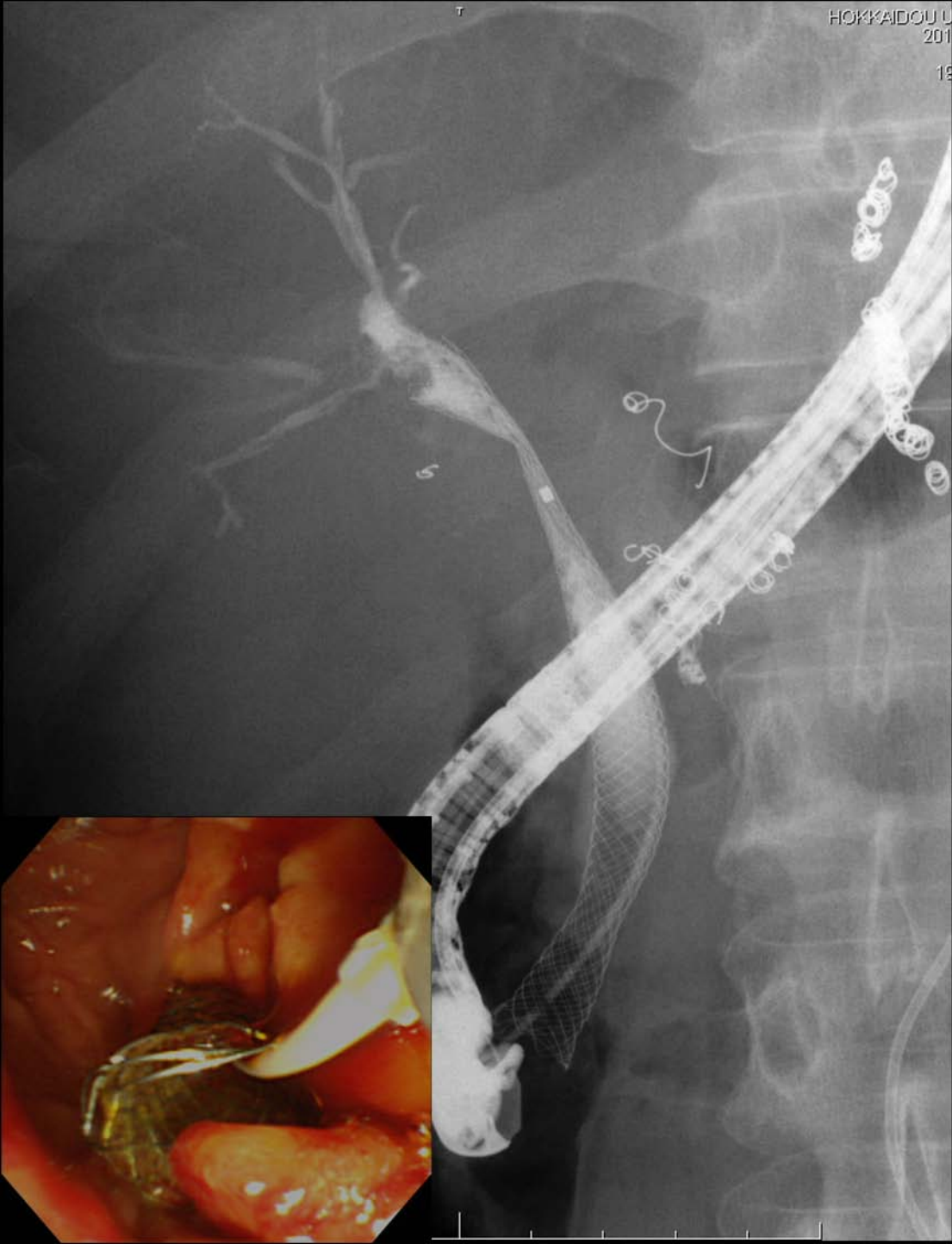
Angiography showing markedly decreased portal vein blood flow and arterial flow supplied from only the subphrenic and intercostals arteries.

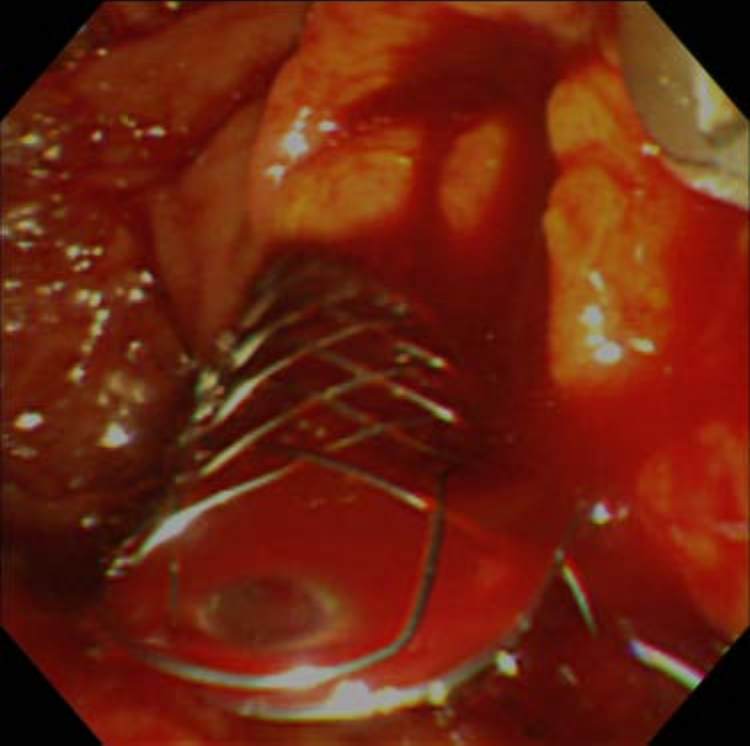


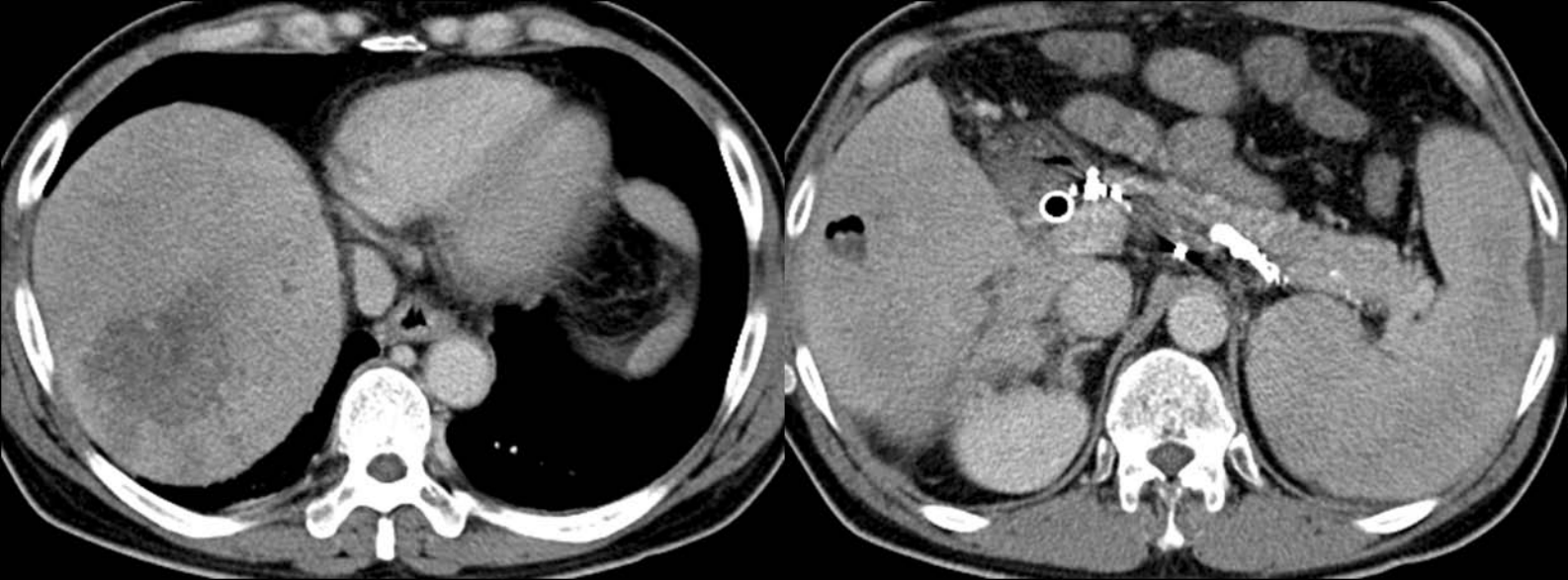



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