Chin Med Sci J
September 2011

Vol. 26, No. 3
P. 190-193

CHINESE
MEDICAL SCIENCES
JOURNAL

CASE REPORT

Sclerosing Cholangitis after Transcatheter Arterial Chemoembolization: a Case Report

Kai Qu[1], Chang Liu[1]*, Qi-fei Wu[1], Bo Wang[1], Aasef Mohamed Ali Mansoor[1], Hao Qin[2], Qiang Ma[2], and Ya-min Liu[2]

[1]Department of Hepatobiliary Surgery, [2]Department of Cardiovascular Intervention, the First Affiliated Hospital, Medical School of Xi'an Jiaotong University, Xi'an 710061, China

Key words: sclerosing cholangitis; transcatheter arterial chemoembolization; bile duct; complication; treatment

SCLEROSING cholangitis represents progressing jaundice or/and paroxysmal symptom of cholangitis, finally developing to end-stage of liver disease. When compared with primary sclerosing cholangitis (PSC), there are no apparent differences in pathology and clinical manifestation in secondary sclerosing cholangitis (SSC). But unlike PSC, SSC always has underlying causes, the most common being biliary obstruction, surgical trauma and ischemic injury of bile duct during liver transplantation.[1,2] Repeated transcatheter arterial chemoembolization (TACE) leading to progressive SSC was rarely reported. Because of its rapid and irreversible progression, once SSC begins, it is difficult to deal with. Therefore, clinicians need to pay more attention to it.

CASE DESCRIPTION

A 23 years old man on physical examination was found to have a neoplastic lesion in the left liver lobe and alpha-fetoprotein (AFP) was 567.3 μg/L on May 2007. Whereafter, left lobe resection was performed and pathology confirmed it was a primary hepatocellular carcinoma (Stage II). One month after surgery the patient was followed up. His AFP level was significantly elevated at 443.0 μg/L, but no positive signs were found in CT scans. Considering hepatic tumor recurrence, we advised this patient to undergo hepatic arterial infusion.

After selective hepatic angiography, chemotherapeutic agents (epirubicin 60 mg and fluorodeoxyuridine 1.25 g dissolved into 150 mL saline solution) were injected. No fever, chills, abdominal pain, diarrhea and jaundice were observed after the procedure. The patient totally underwent another two interventional therapy procedures with a similar approach performed by the same professional radiologist (Table 1). Thirty months after surgery, we performed the 4th TACE and injected emulsion (epirubicin 50 mg+fluorodeoxyuridine 1.0 g+iodized oil 15 mL) and gelfoam particles (diameter, 500-1000 μm) for abnormal staining seen in the right hepatic lobe and the caudate lobe (Fig. 1).

One month after the 4th TACE, fever and progressive aggravating jaundice were observed in this patient. White blood cell count was elevated and AFP was 240 μg/L. Cholestasis parameters including alkaline phosphatase (1951.4 U/L), γ-glutamyl transpeptadase (828 U/L), total bilirubin (425.8 μmol/L), and direct bilirubin (175.2 μmol/L) were significantly elevated. Autoimmune parameters such

Received for publication November 25, 2010.

*Corresponding author Tel: 86-29-85263190, Fax: 86-29-85263190, E-mail: eyrechang@126.com

Table 1. Interventional treatment for four times

No.	Time (mon)	AFP level (μg/L)	DSA presentation	Interventional therapy
1	1	433.0	Right hepatic artery was normal and no tumor staining was seen	HAI (FUDR 1.25 g+Epi 60 mg)
2	17	620.4	Right hepatic artery was normal and no tumor staining was seen	HAI (FUDR 1.25 g+Epi 60 mg)
3	26	313.9	Right hepatic artery was thinner and abnormal staining was seen in the right lobe (Fig. 1A)	HAI (FUDR 1.0 g+Epi 40 mg)+TACE (Epi 20 mg+iodized oil 5 mL)
4	30	634.9	Right hepatic artery was thinner, and abnormal staining was seen in the right and the caudate lobe (Fig. 1C)	TACE (FUDR 1.0 g+Epi 50 mg+iodized oil 15 mL)+gelfoam particles

AFP: alpha fetoprotein; DSA: digital subtraction angiography; HAI: hepatic arterial infusion; FUDR: fluorodeoxyuridine; Epi: epirubicin; TACE: transcatheter arterial chemoembolization.

as antinuclear antibody, smooth muscle antibody, antimitochondrial antibody and perinuclear antineutrophil cytoplasmic antibodies were all negative. CT scans after the 4th TACE showed multiple round lesions with central necrosis (Fig. 2). Endoscopic retrograde cholangiopancreatography (ERCP) suggested diffuse intrahepatic and extrahepatic bile duct occlusions and rarefaction of the small bile ducts (Fig. 3A). Pancreatic ducts were entirely normal. Histological findings of needle biopsy of liver showed that there was an inflammatory response of infiltrated lymphocytes and plasma cells and fibroid degeneration in the portal regions. Cholestasis with interlobular bile duct proliferation was also observed (Fig. 4).

Ursodeoxycholic acid (UDCA) (10 mg/kg body weight) and antibiotics were used in our patient. To relieve jaundice of this patient, we performed endoscopic nasobiliary drainage (ENBD) and flushing of the bile duct with a continuous infusion of saline solution. After ENBD, both clinical manifestation and biochemical values of this patient were mitigated (Fig. 5). The patient was discharged on the 15th day after removed ENBD tube and with remarkable improvement of bile duct stricture (Fig. 3B). A seventeen months follow-up of patient showed that, at present, he is in a relatively stable condition without clinical sign of hepatic failure.

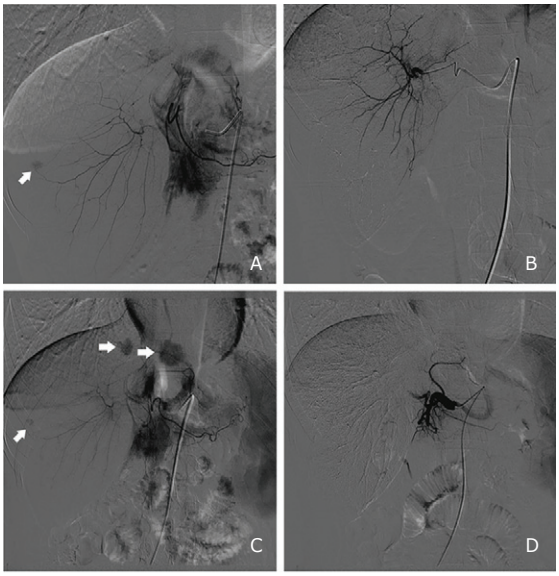

Figure 1. DSA presentations of the 3rd and 4th TACE.
A. Abnormal staining was seen in the right lobe in the 3rd TACE (arrow).
B, D. Presentations of hepatic angiography after the 3rd and 4th TACE, respectively.
C. Abnormal staining was seen in the right hepatic lobe and the caudate lobe in the 4th TACE (arrows).

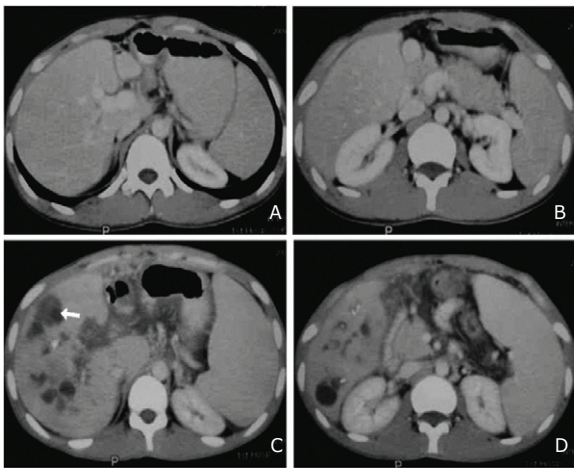

Figure 2. CT scan images pre- and post-4th TACE.
A, B. Transverse CT images before the 4th TACE are normal.
C, D. Transverse CT images one month after the 4th TACE show multiple low-density lesions with sharp margin in the hepatic parenchyma (the size of biggest one is 3.3 cm×2.5 cm, arrow).

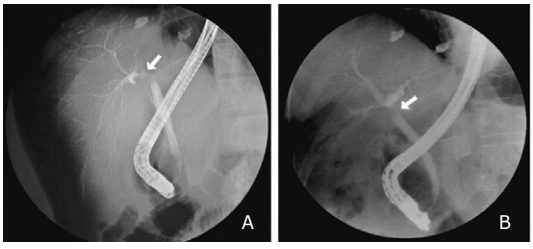

Figure 3. Endoscopic retrograde cholangiopancreatography (ERCP) shows common hepatic duct at the level of the hilum denotes a stricture (about 0.5 cm in length, arrow), and intrahepatic bile ducts are thin and their route is stiffened (A). ERCP shows the stricture becomes wider after endoscopic nasobiliary drainage (B, arrow).