

# Successful Transcatheter Chemoembolization for Acute Jaundice in a Patient with Advanced Hepatocellular Carcinoma and Portal Vein Tumor Thrombosis: A Case Report

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## Abstract

A 54-year-old man with suspected cirrhosis and a hepatic tumor on positron emission tomography presented to our hospital for assessment and treatment in January 2007. Laboratory tests and diagnostic imaging revealed that the patient had cirrhosis due to hepatitis B virus infection and advanced hepatocellular carcinoma (HCC) along with portal vein tumor thrombosis (PVTT) (T4N1M0, Child's B). After hospitalization, the serum levels of total and direct bilirubin increased markedly within several days (26.0 and 20.0 mg/dL), and biliary obstruction by the tumor appeared to have caused this sudden jaundice. To treat the biliary obstruction, selective transcatheter chemoembolization (TACE) was performed via the feeding arteries of the tumor in the anterior segment of the right lobe. After TACE, total bilirubin decreased to 7.0 mg/dL, and the patient survived for 4 more months.

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**Key words:** hyperbilirubinemia, hepatocellular carcinoma, portal vein tumor thrombosis, transcatheter arterial chemoembolization

## Introduction

For the treatment of hepatocellular carcinoma (HCC) in patients with cirrhosis and a total bilirubin level greater than 3.0 mg/dL or portal vein tumor thrombosis (PVTT) or both, some investigators have recommended intra-arterial chemotherapy and avoidance of further interventions, such as embolotherapy<sup>1–4</sup>. Our patient with advanced HCC had severe jaundice and PVTT. Since it seemed

likely that the tumor had caused biliary obstruction and the sudden onset of jaundice, transcatheter arterial chemoembolization (TACE) was performed selectively via the feeding vessels of the tumor. We found that TACE was effective for treating jaundice in this patient.

## Case Report

A 54-year-old man had been found to have chronic hepatitis due to hepatitis B virus (HBV) infection 6

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Table 1

WBC	4,600 / $\mu$ L	Na	137 mEq/L	HBsAg	( + )
RBC	$432 \times 10^4$ / $\mu$ L	K	3.0 mEq/L	HBsAb	( - )
Hb	13.8 g/dL	Cl	98 mEq/L	HBeAg	( - )
Ht	39.7 %	BUN	12.0 mg/dL	HBeAb	( + )
Plt	$7.5 \times 10^4$ / $\mu$ L	Cre	0.70 mg/dL	HBcAb	( + )
AST	258 IU/L	TP	8.0 g/dL	HBV-DNA	6.6 LC/mL
ALT	165 IU/L	Alb	3.6 g/dL	HCVAb	( - )
LDH	219 IU/L	T-Cho	147 mg/dL	NH <sub>3</sub>	138 $\mu$ g/dL
ALP	916 IU/L	TG	240 mg/dL	PT	88.1 %
ChE	159 IU/L	Glu	308 mg/dL	AT3	129.1 %
$\gamma$ -GTP	1,129 IU/L	HbA1c	8.8 %	AFP	352.1 ng/mL
T-Bil	9.9 mg/dL	CRP	0.51 mg/dL	PIVKA-2	3,645 mAU/mL
D-Bil	7.7 mg/dL				



Fig. 1 Computed tomography (CT) shows multiple hepatic tumors in segments 5 to 8, occlusion of the right portal vein by tumor, and intrahepatic bile duct dilatation.

years earlier and had been followed up without any symptoms. In January 2007, 18F-fluorodeoxyglucose positron emission tomography suggested cirrhosis and hepatic tumors, and the patient presented to our hospital for further evaluation and treatment in February 2007. The patient had smoked 30 cigarettes per day and had drunk 1 L of beer per day for 30 years. His medical history included appendicitis and diabetes mellitus. His mother and third brother also had chronic hepatitis due to HBV.

On admission, physical examination revealed jaundice. The liver was palpable at 3 fingerbreadths below the right costal margin, and the spleen was palpable at 2 fingerbreadths below the left costal margin. Spider nevi were detected on the chest wall, but ascites, peripheral edema, and other signs of chronic liver disease were not found. Laboratory

studies revealed obstructive jaundice and moderate liver damage associated with HBV infection and type II diabetes mellitus (**Table 1**). Esophagogastric varices (Lm, F<sub>2</sub>, Cb, RC<sub>3</sub>, Lg +) were demonstrated by upper gastrointestinal endoscopy<sup>5</sup>. Abdominal computed tomography (CT) revealed multiple tumors occupying most of the right lobe of the liver with PVTT and concomitant intrahepatic bile duct dilatation (**Fig. 1**). On the basis of these findings, HCC (T4N1M0, Child's B) was diagnosed<sup>6</sup>.

After hospitalization, the serum levels of total and direct bilirubin increased markedly and reached 26.0 and 20.0 mg/dL within a few days. We attributed the profound jaundice to biliary obstruction caused by expansion of the HCC. Portography showed dilatation of the portal vein, tumor thrombosis in the main portal vein, and a complete absence of portal venous flow in the right lobe of the liver (**Fig. 2**). Because the HCC comprised multiple tumors, surgical treatment was considered too difficult. After confirming the feeding vessels for the tumor with angiographic CT, we concluded that TACE could be selectively performed via these vessels. To treat the biliary obstruction, we therefore performed selective TACE (10 mL of lipiodol, 50 mg of farnorubicin, and 60 mg of gelfoam) for the HCC in the anterior segment of the right lobe (**Fig. 3**). After TACE, serum bilirubin decreased to 7.0 mg/dL, serum levels of alkaline phosphatase and  $\gamma$ -guanosine triphosphatase decreased, and liver function showed no further deterioration (**Fig. 4**). The patient was discharged in May 2007 but died of ruptured esophageal varices in August 2007.

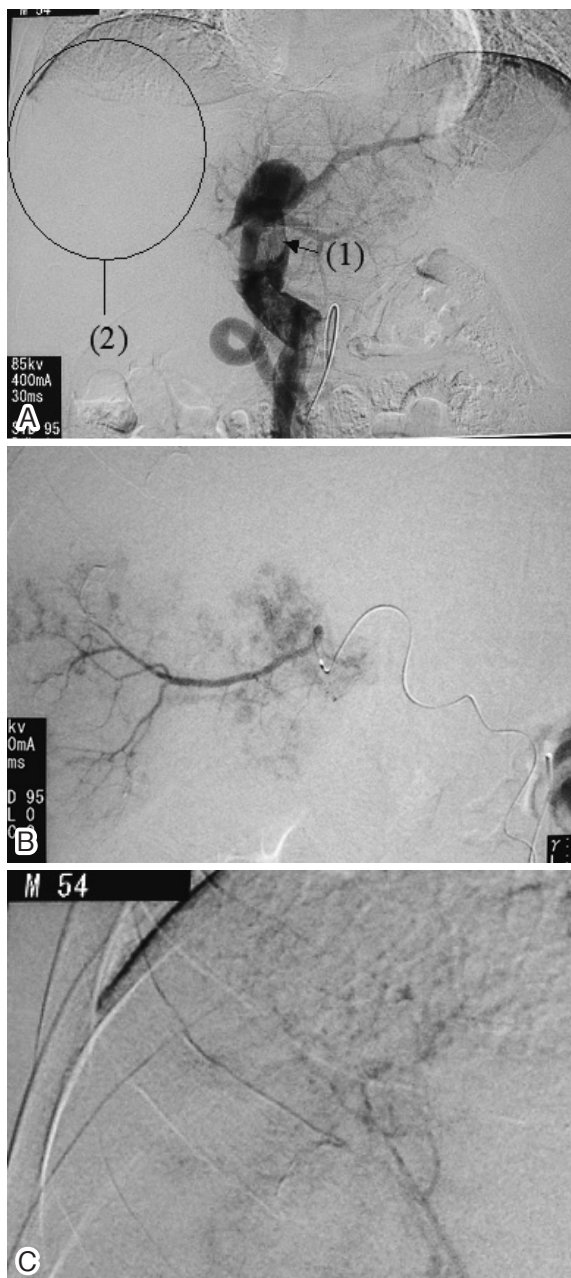


Fig. 2 (A) Portography reveals dilatation of the portal vein, tumor thrombosis in the main portal vein (1), and complete absence of portal venous flow in the right lobe (2). Hepatic artery angiography shows selective enhancement of A5 (B) and A8 (C), and these vessels have associated tumor stains. Therefore these vessels were suspected to be tumor feeders.

### Discussion

In this patient, the T-Bil/D-Bil levels increased rapidly after hospitalization. A comparison of the laboratory results and imaging findings suggested



Fig. 3 Abdominal CT after TACE reveals selective accumulation of lipiodol in the anterior segment of the right lobe of the liver.

that the likeliest cause of the jaundice was biliary obstruction due to tumor growth. We thought that the profound jaundice should be treated urgently. Angiography clearly demonstrated the feeding arteries of the HCC, and we concluded that TACE could be performed selectively via these vessels with minimal damage to functioning hepatocytes. Because this patient had good liver function (Child's B, liver damage grade A), we performed selective TACE via the feeding arteries. The jaundice and serum levels of alkaline phosphatase and  $\gamma$ -guanosine triphosphatase decreased after the relief of biliary obstruction, and the patient survived for more than 4 months. It is possible that if we had been able to depict the feeding vessels of the tumor causing PVTT and perform TACE, the death of this patient (through rupture of esophageal varices) might have been prevented.

According to guidelines for the treatment of HCC, patients who have grade A liver damage and more than 4 tumors should be treated with TAE (TACE) or arterial infusion chemotherapy<sup>7</sup>. Although some investigators have found that TACE can prolong the survival of patients with unresectable HCC<sup>8-11</sup>, others have reported that TACE (or TAE) is contraindicated in such patients with PVTT<sup>1-4</sup>. However, our patient showed good liver function (Child's class B) except for jaundice, and the feeding vessels of the tumor were clearly visualized; therefore, selective TACE could be performed via those vessels without any adverse events. It

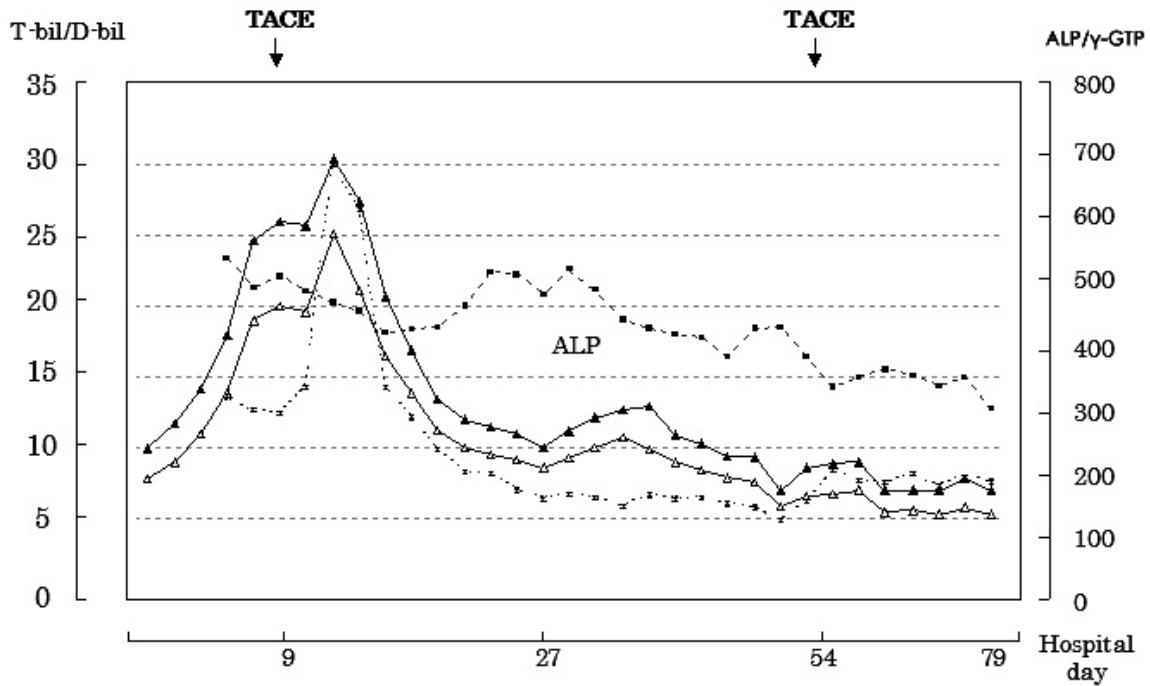


Fig. 4 Clinical course. (▲) T-Bil, total-Bilirubin; (△) D-Bil, direct bilirubin; (∗) ALT, alanine aminotransferase; (■) ALP, alkaline phosphatase; TACE, transcatheter arterial chemoembolization.

appeared that the patient's survival was prolonged, and tolerable quality of life was maintained<sup>10,11</sup>.

In conclusion, if the blood supply to the liver via the portal vessels and hepatic arteries and the vessels feeding the tumor are clearly visualized, it may be possible to perform TACE with minimal damage to the functioning hepatocytes in patients who have high risk condition for embolotherapy.

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