A Case of Spontaneous Rupture of Hepatocellular Carcinoma Supplied by the Right Renal Capsular Artery Treated by Transcatheter Arterial Embolization

Joo Yeon Jang1, Ung Bae Jeon1, Jin Hyeok Kim1, Tae Un Kim1, Hwaseong Ryu1, Mong Cho2, Young Mi Hong2, Maeran Kim1

Departments of Radiology and Internal Medicine, Pusan National University Yangsan Hospital, Pusan National University College of Medicine, Busan, Korea

We present a case of spontaneous rupture of hepatocellular carcinoma with poor liver function managed by transcatheter arterial embolization (TAE). The patient’s bilirubin level was 2.1 mg/dL, albumin level was 2.4 g/dL, and prothrombin time international normalized ratio was 2.1. In addition, the patient had also developed a large number of ascites. The tumor was supplied by the right renal capsular artery, as observed on angiography. With successful TAE, no hepatic failure occurred. We believe TAE can be a safe and effective treatment option, even in patients with poor liver function, if tumors are supplied only by extrahepatic collateral vessels. (J Liver Cancer 2019;19:59-63)

Keywords: Hepatocellular carcinoma; Spontaneous rupture; Renal capsular artery; Extrahepatic collateral; Transcatheter arterial embolization

INTRODUCTION

Transcatheter arterial embolization (TAE) is considered a useful treatment in patients with spontaneously ruptured hepatocellular carcinoma (HCC). However, we must consider the patient’s liver function before we embolize the hepatic artery. We report a case of spontaneously ruptured HCC with poor liver function that was successfully managed by TAE via the right renal capsular artery.

CASE REPORT

1. Clinical findings

A 53-year-old man with hepatitis B virus infection and alcoholic liver cirrhosis visited the emergency room (ER) in our hospital. He was diagnosed with HCC 3 years ago and received transarterial chemoembolization (TACE) five times, at another hospital. However, he decided to obtain supportive care because his liver function was class C according to Child-Pugh’s classification. Around that time, he visited our ER for regular paracentesis. On that day, he experienced abdominal distension and dyspnea; bloody ascites were diagnosed by paracentesis. His blood pressure was 80/40 mmHg. Laboratory values on the same day showed a hemoglobin level of 7.3 g/dL, platelet count of 89×10^3/µL, serum bilirubin level of 2.1 mg/dL, albumin level of 2.4 g/dL, prothrombin
time international normalized ratio of 2.1, aspartateaminotransferase level of 43 IU/L, and alanine aminotransferaselevel of 25 IU/L. At that time, there was no evidence ofhepatic encephalopathy. Therefore, his Child-Pugh score wasclass C at 11 points.

2. Imaging findings

Abdomen computed tomography showed an infiltratinghypodense mass at the posterior inferior segment (segment6) of the right hepatic lobe with tumor thrombosis in theright posterior branch and a large number of ascites (Fig.1A). In addition, extravasation of contrast media from thismass and hemoperitoneum were observed in the arterialphase (Fig. 1B).

3. Diagnosis and treatment

Celiac arteriography using a 5F catheter (Rosch hepatic;Cook Medical Inc., Bloomington, IN, USA) demonstrated atumor stain in the right lobe of the liver, but without evidece of extravasation (Fig. 2A). Considering that the tumoris located at the periphery of the liver and adjacent to theright kidney, we suspected that the tumor might be suppliedby a parasitic blood supply, such as the right renal capsularartery. Right renal arteriography depicted contrast extrava-sation from the right renal capsular artery (Fig. 2B). Selectivearteriography through the right renal capsular artery using a microcatheter (Progreat, Terumo, Tokyo, Japan) and a mi-crowire (Transend, Boston Scientific, Marlborough, MA,USA) revealed a tumor stain and extravasation (Fig. 2C). Thisartery was embolized with a mixture of 2.0 mL of io-dized oil (Lipiodol Ultrafluid, Guerbet, Aulnay-sous-bois,France) and 0.5 mL of n-butyl-2-cyanoacrylate (Histoacryl,B. Braun Melsungen AG, Melsungen, Germany) glue. Con-sequently, the tumor stain and extravasation disappeared(Fig. 2D). After the procedure, the patient’s vital signsnormalized. His bilirubin value increased to 4.6 mg/dL tran-siently, then dropped to below 3.2 mg/dL, and no hepaticfailure occurred after TAE. Ten days later, he was discharged. He visited our ER repeatedly because of a large number ofascites and hepatic encephalopathy. He was admitted due to spontaneous bacterial peritonitis. During the hospital stay, the patient developed pneumonia and died about 2 months after undergoing TAE.

DISCUSSION

Hepatocellular carcinoma is a hypervascular tumor that al-most always occurs in patients with liver cirrhosis. It is the
fifth-most common cancer in the world and its incidence is gradually increasing. The reported incidence of spontaneous rupture of HCC varies from 2.9% to 26%.²,³,⁴,⁸

The mortality rate in the acute phase of rupture is around 25-75% of cases. This complication is the third-most common cause of death in HCC patients, after neoplastic progression and liver failure.⁹

The mechanism of spontaneous ruptured HCC is not fully understood. Some investigators believe that disruption of a friable feeding artery or development of a tear in the surface of a tumor subjected to high pressure can cause rupture. Rapid growth of the tumor and necrosis are associated with increased intratumoral pressure caused by progressive or sudden occlusion of branches of hepatic veins due to tumor invasion.⁴,⁶,¹⁰

Several studies have reported that tumor size is significantly greater in spontaneously ruptured HCC than in non-ruptured HCC.⁷,¹¹ When HCC protrudes beyond the original liver margin, the risk of rupture may be higher than that of tumors surrounded by normal liver parenchyma.⁴,⁷,¹²,¹³ Chen et al.⁷ reported that HCC in the left lobe may be more prone to rupture than that in the right lobe.

The symptoms of HCC rupture include the sudden onset of abdominal pain (66.4%), abdominal distention (16%), and shock (6.7%).¹⁴ Spontaneous rupture is usually a critical and life-threatening condition because of hypovolemic shock.

Figure 2. Transcatheter arterial embolization. (A) Celiac arteriography demonstrates the tumor stain in the right lobe of the liver without evidence of extravasation. (B) Right renal arteriography depicts contrast extravasation (arrow) from right renal capsular artery (arrowhead). (C) Selective arteriography through the right renal capsular artery reveals a tumor stain (arrowhead) and extravasation (arrow). (D) After embolization, the tumor stain and extravasation disappeared.
due to massive blood loss into the peritoneal cavity. The death rate ranges from 1% to 22%.8,14

HCC generally receives blood supply from the hepatic artery, however, various collateral blood supplies have also been reported.15 The prevalence of parasitic blood supply is unclear. The right renal capsular artery has also been described as an extrahepatic collateral vessel to the liver.15,16

TAE is a useful treatment for unresectable HCC.17,18 It causes ischemic necrosis of the tumor tissue in hypervascular HCC. TAE is widely used for various types of HCC unless the main portal vein is occluded. In usual TACE, an iodized oil containing an anticancer drug is used in combination with gelatin sponge particles to act as anti-cancer therapy and to cease bleeding. We use only a gelatin sponge in cases of emergent hepatic artery embolization for HCC rupture in our hospital. In our case, due to bleeding occurring from the parasitic vessel, the sole function of which was to supply the tumor, n-butyl-2-cyanoacrylate was indicated. However, when the parasitic vessel supplies the tumor and the surrounding liver parenchyma, embolic material should be gelatin sponge particles alone.

Acute hepatic failure occurs after TAE in 2.1% of cases.19 The risk factors for acute hepatic failure associated with TAE are poor hepatic functional reserve, a history of multiple embolization procedures, and high doses of chemotherapeutic agents.19 Generally, TAE is not performed in patients with poor liver function, especially hyperbilirubinemia (≥2.0 mg/dL).20 Okazaki et al.2 argued that emergency TAE for ruptured HCC is contraindicated in patients with hyperbilirubinemia (≥3.0 mg/dL). However, selective TAE reduces the risk of hepatic failure because it minimizes normal liver damage. It should also be noted that the parasitic blood supply is usually distributed only to the tumor, and not to surrounding hepatic parenchyma. For this reason, TAE via parasitic vessels is safer than that via hepatic arteries in terms of normal liver injury. In our patient, the tumor feeders were both the hepatic artery and right renal capsular artery. However, contrast leakage was observed only in the right renal capsular artery. Therefore, we occluded that artery safely without concerns of acute hepatic failure. In conclusion, we believe that if HCC rupture develops from only extrahepatic collateral vessels, TAE is an effective procedure, even in patients with poor liver function.

ACKNOWLEDGEMENTS

This study was supported by a 2018 research grant from Pusan National University Yangsan Hospital.

Conflicts of Interest

The authors have no conflicts to disclose.

REFERENCES