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CASE REPORT

# Percutaneous Transumbilical Portal Vein Embolization in a Patient with a Ruptured Hepatocellular Carcinoma Supplied by the Portal Vein

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**Abstract** We describe a case of a ruptured hepatocellular carcinoma supplied by the portal vein that was successfully treated with portal vein embolization via a percutaneous transumbilical approach. A contrast material-enhanced computed tomographic (CT) scan showed the presence of a large hypervascular tumor on portal venous phase as well as right hepatic vein thrombosis and hemoperitoneum that prevented portal vein embolization by the use of the percutaneous and transjugular transhepatic approach. The use of percutaneous transumbilical portal vein embolization can be an alternative option in this situation.

**Keywords** Chemoembolization · Portal vein · Umbilical vein · Hepatocellular carcinoma

## Introduction

Chemoembolization is widely used in the management of an inoperable hepatocellular carcinoma (HCC) [1]. Whereas the liver receives a dual blood supply from the hepatic artery and portal vein, most HCCs are supplied by the hepatic artery. However, previous reports have documented vascular supply to HCC and dysplastic nodules from the portal vein by computed tomographic (CT) arterial portography [2–6].

Spontaneous rupture is a catastrophic complication of a HCC that is associated with a high mortality rate of up to 40%, as has been reported in Asian countries [7–9]. For the management of active bleeding from a ruptured HCC, several treatment modalities including transarterial embolization and emergency or staged liver resections have been proposed [7–9].

In this report, we describe a patient who sought care for a ruptured HCC supplied by the portal vein. A CT scan revealed the presence of a large tumor with enhancement as seen on portal venous phase and hemoperitoneum. Right hepatic vein thrombosis and ascites prevented portal vein embolization by the use of the percutaneous transhepatic and transjugular transhepatic approach. The patient was successfully treated with portal vein embolization via a percutaneous transumbilical approach.

## Case Report

A 45-year-old man infected with the hepatitis B virus was admitted to our hospital for abdominal pain that had lasted 3 days. The patient had asthenia, dyspnea, and tachycardia due to hypotension (blood pressure, 78/40 mmHg). The patient had a medical history of multiple HCC nodules that were detected 2 years earlier. Since the detection of the HCC lesions, the patient had undergone repeated transcatheter arterial chemoembolization (seven sessions, including the initial session) through the hepatic artery. The latest session was performed 4 months before admission.

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At admission, physical examination revealed abdominal distension with shifting dullness, whole abdominal tenderness, and rebound tenderness. Laboratory studies revealed the following abnormal values: A hemoglobin level of 9.0 g/dL (normal range, 12–16 g/dL) after two packs of RBC transfusion, an asparatate aminotransferase level of 76 IU/L (normal range, 0–40 IU/L) alanine aminotransferase level of 57 IU/L (normal range, 0–40 IU/L), alkaline phophokinase level of 77 IU/L (normal range, 30–115 IU/L) and total bilirubin level of 1.3 mg/dL (normal range, 0.2–1.2 mg/dL). The level of alpha-fetoprotein (AFP) was 40,710 ng/mL (normal range, 0–20 ng/mL). When we performed tapping for ascites, fresh blood was aspirated.

A contrast-enhanced CT image demonstrated the presence of a large inhomogeneous mass in hepatic segment VI. This mass displayed mainly low attenuation during the hepatic arterial phase and heterogeneous high attenuation during the portal venous phase (Fig. 1A, B). In addition, a hematoma with high attenuation around the liver was detected, which was consistent with the presence of a ruptured HCC (Fig. 1C). A CT scan also showed right hepatic vein thrombosis (Fig. 1C) and a recanalized paraumbilical vein (Fig. 1D).

As seen on celiac angiography, the right hepatic artery was occluded and no hypervascular mass was detected (Fig. 1E). To search for extrahepatic collateral arteries, we performed selective angiography of the right inferior phrenic artery, adrenal artery, renal artery, intercostal artery, superior mesenteric artery, and right internal mammary artery, which failed to reveal tumor staining. On the portography obtained from the superior mesenteric arteriography, a hypervascular mass was visualized in hepatic segment VI.

For selective embolization of the portal vein that fed the tumor, the paraumbilical vein was punctured under ultrasonographic guidance, and a 4F sheath was inserted. A 4F catheter (Glidecath; Terumo, Tokyo, Japan) was advanced over a hydrophilic guide wire and was placed in the main portal vein. Direct portography revealed an exophytic hypervascular tumor supplied from segment VI portal branch (Fig. 1F). With the use of a microcatheter (Renegade Hi-Flo; Boston Scientific, Natick, MA, USA), the segment VI portal branch was superselected; gelatin sponge particles (Gelfoam; Upjohn, Kalamazoo, MI, USA) mixed with doxorubicin hydrochloride (Adriamycin RDF; Ildong Pharmaceutical, Seoul, Korea) were injected. Finally, the punctured paraumbilical vein was embolized with the use of a Nester coil (Cook, Bloomington, IN, USA) followed by sheath removal. Immediately after embolization, the blood pressure and heart rate were normalized and the hemoglobin level was stabilized (13.6 g/dl).

One week later, an ultrasound examination showed the usual HCC features of different echogenicity from the surrounding liver in segment VI, and there was no evidence of active bleeding. One month later, the alfa-fetoprotein level had decreased to 720 ng/ml. As seen at contrast-enhanced CT examination, the size of the mass in segment VI had also decreased 2 months later.

## Discussion

It is well known that an HCC is a hypervascular tumor supplied via the hepatic artery. As a result, transcatheter arterial chemoembolization—a mixture with lipiodol and/or several anticancer agents—has been widely performed with good therapeutic results for HCCs [1]. Moreover, transcatheter embolization has been used as an effective treatment to achieve hemostasis of a ruptured HCC in an emergency [7–9].

Several researchers have used CT during arterial portography and have demonstrated that some lesions, such as large regenerating nodule, dysplastic nodule, and well-differentiated small HCCs, were supplied not only by the hepatic artery but also by the portal vein [2–6]. These investigators have suggested that in the course of dedifferentiation from the normal liver parenchyma to an HCC showing sequential hemodynamic changes, such lesions may be in an intermediate phase where cells are nourished from both a hepatic artery and portal vein [2, 4].

For a patient who has history of repeated chemoembolization, the developing mechanism of an HCC supplied by the portal vein is thought to be quite different. Goseki et al. [10] have suggested that the some HCC cells were nourished by the portal blood flow directly throughout the viable tumor mass during at least some period after chemoembolization. Choi et al. [5] have reported a case where all of the tumor nodules had been exclusively nourished from the portal vein after repeated chemoembolization. These investigators insisted that the source of the blood supply in a nodule totally changed to the portal vein from the hepatic artery during the developmental process of the HCC as a result of hepatic arterial damage from repeated chemoembolization. Similarly, in our case, the latest celiac angiography demonstrated the presence of a poorly seen right hepatic artery, although the initial celiac angiography manifested a well-identified right hepatic artery with abundant blood flow. The newly developed HCC was totally supplied by the portal vein.

When celiac angiography showed occlusion of the right hepatic artery caused by repeated chemoembolization, we thought the tumor should supply the extrahepatic collateral arteries as occlusion of the hepatic artery exaggerated the formation of extrahepatic collateral arteries. Thus, we performed selective angiography of the right inferior phrenic artery, adrenal artery, renal artery, intercostal artery, superior mesenteric artery, and right internal

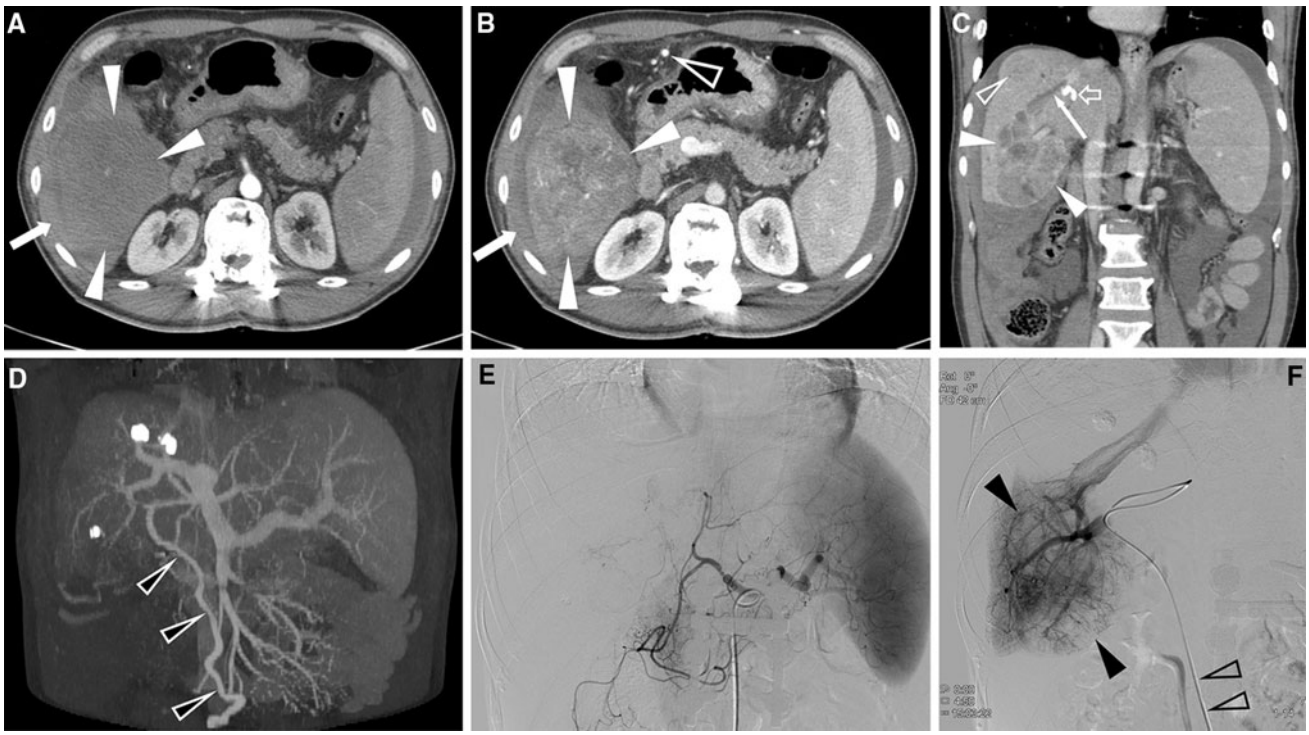


**Fig. 1** A case of a 45-year-old man with a ruptured HCC supplied by the portal vein is presented. **A** A transverse hepatic arterial phase CT scan shows a hematoma (*arrow*) of high attenuation around the liver, and a large mass (*arrowheads*) of low attenuation in segment VI of the liver. Note ascites of low attenuation around the spleen. **B** A transverse portal venous phase CT scan demonstrates a large mass (*arrowheads*) of heterogenous high attenuation. Note the dilated enhancing paraumbilical vein (*open arrowhead*) and hematoma (*arrow*) around the liver. **C** A coronal delayed-phase CT scan shows a large low attenuating mass (*arrowheads*) in segment VI with a tumor thrombus (*arrow*) in the right hepatic vein. Note another small tumor (*open arrowhead*) in segment VIII and iodized oil retention resulting from previous chemoembolization (*open arrow*). Note the hematoma of high attenuation around the liver and ascites of low attenuation around the spleen. **D** An anteroposterior view maximal intensity projection image shows the course of the paraumbilical vein (*arrowheads*) from the left portal vein. **E** Celiac angiography shows occlusion of the right hepatic artery and no demonstrable tumor staining. **F** A percutaneous transumbilical portogram reveals a large hypervascular mass (*arrowheads*) in segment VI, which is supplied by the portal vein. Note the catheter placed through the paraumbilical vein (*open arrowheads*)

mammary artery. However, there was no tumor staining as seen at hepatic and extrahepatic collateral angiography, and we were first puzzled about what to do next. After a careful review of CT scan that indicated the presence of a hypervascular tumor on portal venous phase and not on hepatic arterial phase, we decided to perform indirect portography obtained from the superior mesenteric arteriography, which revealed tumor staining fed by the portal vein.

Spontaneous recanalization with blood flow in the umbilical vein has been reported to occur in 26% of patients with cirrhosis of the liver and portal hypertension [11]. The use of the percutaneous transumbilical approach was reported for esophageal varix embolization more than two decades ago [12]. The transumbilical approach has an advantage over the percutaneous transhepatic approach because it can obviate intra-abdominal hemorrhage in a patient with tense ascites or a coagulation disorder [12]. Intra-abdominal hemorrhage may infrequently occur when the paraumbilical vein is punctured percutaneously, as the paraumbilical vein is located in the intraabdominal wall adjacent to the umbilicus and is not located in the intraperitoneal space. In the present case, because the patient had a large hematoma, the percutaneous transhepatic approach was not selected, and tumor thrombosis of the right hepatic vein made the use of the transjugular approach impossible. Instead, the luminal diameter of the paraumbilical vein was sufficient for insertion of an angiocatheter.

In conclusion, we report a patient with a ruptured HCC supplied by the portal vein. We advocate that when a CT scan shows a tumor with enhancement on the portal venous phase, radiologists should keep in mind that the tumor could be supplied by the portal vein. The use of the percutaneous transumbilical approach can be a safe alternative route for portal vein embolization when hepatic vein thrombosis and ascites disturb portal vein embolization by the use of the percutaneous and transjugular transhepatic approach.

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**Conflict of interest** The authors declare that they have no conflict of interest.

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