—Case Reports—

Major Arterioportal Shunt Caused by Hepatocellular Carcinoma

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Abstract

A case of hepatocellular carcinoma (HCC) causing a major arterioportal (A-P) shunt is reported. The patient exhibited massive ascites and tested positive for hepatitis B surface antigen. An abdominal computed tomography (CT) examination showed a low-density lesion in the left lobe of the liver and an A-P shunt, but no tumor stain was visible. Upper gastrointestinal endoscopy revealed severe esophageal varices. Because the tumor marker level was abnormally high, an HCC causing an A-P shunt in a cirrhotic liver background with severe esophageal varices as a result of portal hypertension was diagnosed. We performed endoscopic variceal ligation to treat the severe esophageal varices and interventional radiology treatment for the A-P shunt and HCC, but the patient's condition was unchanged. Because the patient's liver function gradually improved, surgical treatment was selected. The patient underwent left hepatectomy. Pathological examination revealed a poorly differentiated HCC in a cirrhotic liver background. The postoperative course was uneventful, and the patient was discharged 2 weeks after the operation.The patient subsequently underwent transcatheter arterial embolization therapy for recurrent HCC in the right lobe of the liver, but the esophageal varices disappeared.
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Key words: hepatocellular carcinoma, arterioportal shunt, esophageal varices

Introduction

Hepatocellular carcinoma (HCC) has one of the worst prognoses of all malignancies because of the typically advanced stage of cancer and severe liver dysfunction at the time of diagnosis and the high recurrence rate after curative treatment. Furthermore, arterioportal (A-P) shunts created caused by such tumors can worsen the patient's condition by causing portal hypertension.

The present report describes a patient with HCC and a major A-P shunt whose condition improved after treatment.

Case Report

A 47-year-old man with massive ascites was referred to our hospital for treatment and to determine the cause of the ascites. The patient's

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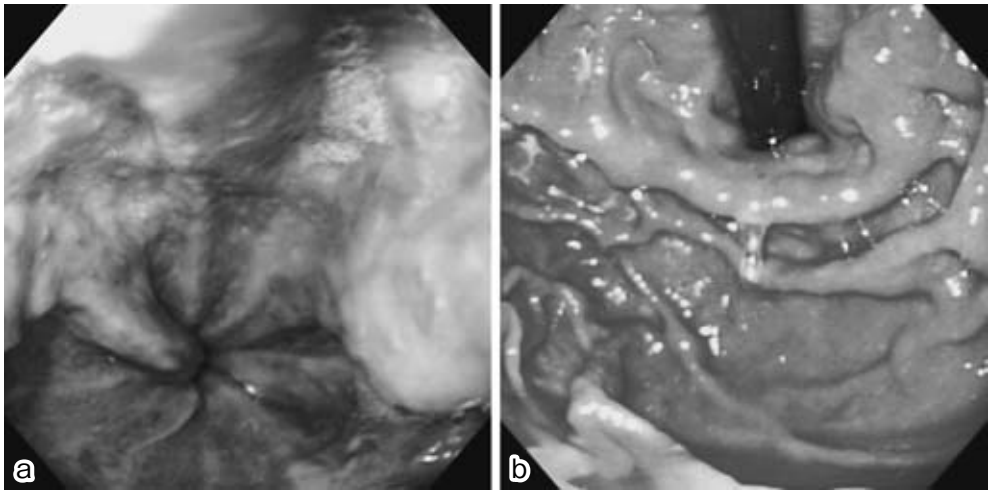

Fig. 1 Upper gastrointestinal endoscopic findings. **a:** Severe esophageal varices are visible. **b:** Gastric varices are not visible.

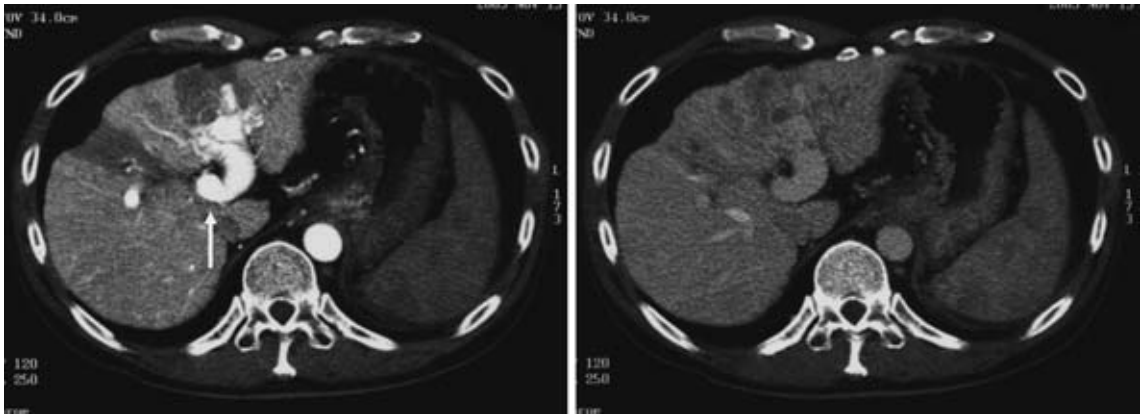

Fig. 2 Abdominal CT shows an A-P shunt but no tumor stain.

laboratory data showed a serum albumin level of 4.1 g/dL (normal: 3.8～5.5 g/dL), a total bilirubin level of 1.1 mg/dL (normal: 0.2～1.2 mg/dL), an aspartate aminotransferase level of 108 IU/L (normal: 10～28 IU/L), an alanine aminotransferase of 156 IU/L (normal: 5～33 IU/L), a γ-glutamyl transpeptidase of 198 IU/L (normal: 8～59 IU/L), a white blood cell count of 5,000 /μL, a red blood cell count of 481 × 10^4 /μL, a hemoglobin level of 15.4 g/dL, a hematocrit of 46.8%, a platelet count of 7.4 × 10^4 /μL, an α-fetoprotein level of 675.5 U/mL, a PIVKA-2 level of 5,827 U/mL, a positive hepatitis B surface antigen status, a negative hepatitis C virus antibody status, a prothrombin time of 73.5%, and an ICGR15 level of 7.5%. The clinical Child's classification status was Child's B.

Upper gastrointestinal endoscopy revealed severe esophageal varices but no gastric varices **(Fig. 1)**. Abdominal computed tomography (CT) examination showed a low-density lesion in the left lobe of the liver and an arterioportal (A-P) shunt, but no tumor stain was seen **(Fig. 2)**. Selective hepatic arteriography showed a major A-P shunt in the left lobe, but a tumor stain was not noted during the late phase because of the shunt **(Fig. 3a)**. Transarterial portography revealed a filling defect caused by thrombus in the portal vein stem and no left portal vein branch **(Fig. 3b)**. Edematous or necrotic parenchyma in the left lobe of the liver was seen with superparamagnetic iron oxide-enhanced magnetic resonance (MR) imaging. While the imaging results did not indicate the presence of a

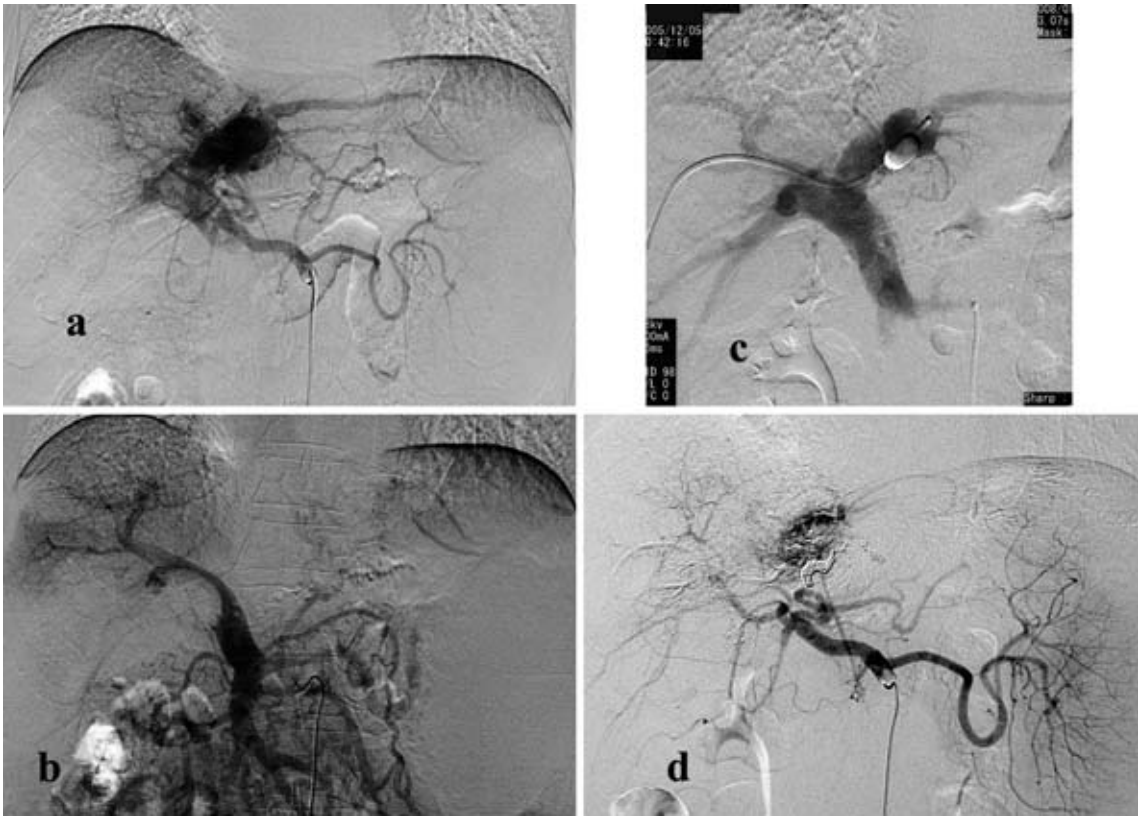

Fig. 3 **a:** Selective hepatic arteriography shows a major A-P shunt in the left lobe but no tumor stain. **b:** Transarterial portography reveals a filling defect in the portal vein stem and no left portal vein branch. **c, d:** TAE with a coil in a balloon-occluded left portal vein was performed for the treatment of the major A-P shunt.

tumor, the tumor marker level was abnormally high; thus, HCC with an A-P shunt in a cirrhotic liver background, complicated by severe esophageal varices as a result of portal hypertension, was diagnosed.

First, we performed endoscopic variceal ligation (EVL) to treat the severe esophageal varices. The ascites was simultaneously treated by performing transcatheter arterial embolization (TAE) with Gelfoam for the major A-P shunt. Despite the TAE, the A-P shunt did not disappear. Consequently, we next performed TAE using a coil in a balloon-occluded left portal vein to treat the major A-P shunt **(Fig. 3c, d)**. After treatment, the filling defect caused by the thrombus in the portal vein stem persisted, and the esophageal varices did not improve. Because no improvement in the A-P shunt was seen, subsequent therapy was required.

Five months later, the massive ascites had become controllable, so the patient underwent left hepatectomy **(Fig. 4)**. Pathological examination revealed a poorly differentiated HCC in the segment 4 area of the liver; the nontumorous lesion was caused by cirrhosis. The postoperative course was uncomplicated, and the esophageal varices improved. The patient was discharged 2 weeks after the operation. Thereafter, the patient underwent TAE therapy for the treatment of recurrent HCC in the right lobe.

Discussion

In cirrhotic liver, vascular casts have demonstrated that fibrous septa comprise a dense network of vessels with some anastomoses between branches of the hepatic artery and portal vein[1]. HCC is frequently associated with A-P shunts. Severe A-P shunt leads to or aggravates portal hypertension, causing life-threatening conditions, such as esophagogastric varices, refractory ascites,

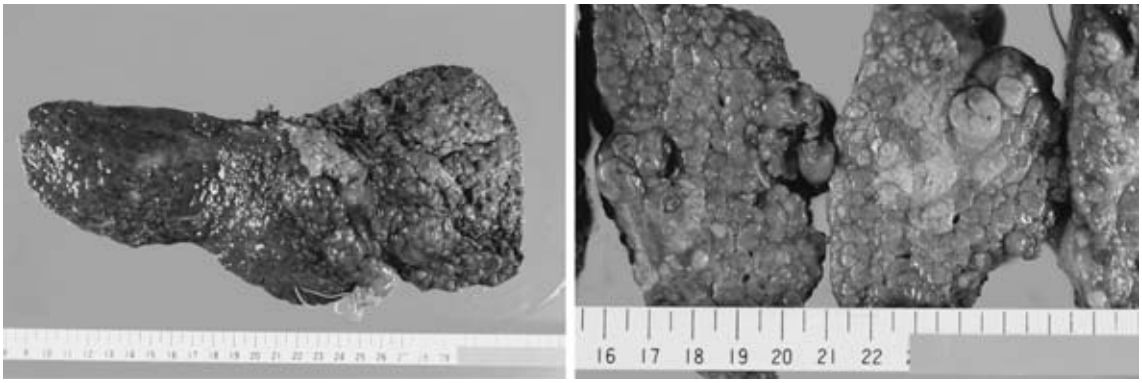

Fig. 4 Macroscopic appearance of the tumor. A poorly differentiated HCC in segment 4 area of the liver; the nontumorous lesion was cirrhotic liver.

refractory diarrhea, and hepatic encephalopathy[2,3]. A-P shunt is detected as a hyperenhancing focus during the arterial phase on dynamic studies and as a portal venous perfusion defect on CT during arterioportography. Nontumorous A-P shunts in patients with chronic liver disease vary in CT appearance and exhibit isoattenuation during the delayed phase, most of which disappear after 4 months[4]. Tumorous A-P shunts are seen as areas of reduced signal loss, whereas most nontumorous A-P shunts are seen as areas of normal signal loss, similar to normal liver parenchyma, on MR imaging using superparamagnetic iron oxide[5].

In the present case, the patient presented with ascites. A CT examination showed a low-density lesion in the left lobe of the liver and an A-P shunt, but a tumor stain was not visible. An MR examination also did not contribute to the diagnosis of HCC. The tumor marker level, however, was abnormally high, and no evidence of type B liver cirrhosis was present; consequently, the presence of HCC was strongly suspected. Rupture of esophageal varices is a life-threatening condition. Kokubu et al.[6] have shown that EVL is effective for the treatment of esophagogastric varices in patients with tumor thrombus of the portal vein trunk associated with HCC. After performing EVL to treat the severe esophageal varices, we selected interventional radiology (IVR) as the next treatment because of the patient's intractable ascites.

To decrease portal hypertension caused by severe A-P shunts in patients with HCC, the A-P shunts must be treated. Transcatheter embolization is an effective treatment for variceal bleeding caused by portal hypertension arising from A-P fistulae, because it reduces arterial flow into the varices[7]. Gelfoam, steel coils and ethanol are the most commonly used embolic materials[8,9]. In the present patient, we attempted to perform TAE using oil in a balloon-occluded left portal vein to treat the major A-P shunt. Unfortunately, the A-P shunt was resistant to several rounds of IVR. Once the ascites was controlled with conservative treatment and the liver function improved, however, we attempted a surgical cure.

The histologic pattern in the present case was a poorly differentiated type. We suspect, but cannot prove, that shunts may develop more easily as a result of poorly differentiated tumors, rather than well differentiated tumors.

In the present case, our treatment strategy was effective and produced no major complications; surgical resection appeared to help improve the patient's condition.

In conclusion, we have presented a patient with HCC and an A-P shunt in whom long-term survival was achieved with various treatments. We believe that survival can be prolonged, even in patients with a poor prognosis, by choosing treatments appropriate for the patient's clinical condition.

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