CASE REPORT

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Rapidly Progressive Hepatocellular Carcinoma Mimicking Benign Portal Vein Thrombosis: A Case Report

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Portal vein thrombosis (PVT) is commonly associated with liver cirrhosis, irrespective of the presence of hepatocellular carcinoma (HCC). Given that malignant PVT is a poor prognostic factor in patients with HCC, it is important to differentiate malignant PVT from benign PVT. Because malignant PVT has been reported to be contiguous with parenchymal HCC, in most cases, the presence of PVT alone indicates a benign entity. We report the case of a patient with rapid progression of malignant PVT mimicking benign PVT but without definite parenchymal HCC on imaging modalities. **(Gut Liver 2013;7:116-119)**

Key Words: Hepatocellular carcinoma; Portal vein thrombosis; Disease progression; Computed tomography; Magnetic resonance imaging

INTRODUCTION

Portal vein thrombosis (PVT) is more prevalent in patients with liver cirrhosis and hepatocellular carcinoma (HCC) than the general population.[1] Benign PVT usually results from portal venous hypertension and venous stasis, whereas malignant PVT is formed by direct invasion of the portal vein by malignant neoplasms, such as HCC.[2] Several imaging features have been used to differentiate malignant PVT from benign PVT. Malignant PVT is generally accompanied by parenchymal mass, such as HCC in cirrhotic liver, whereas the presence of PVT alone is considered benign.

It is necessary to diagnose PVT in patients with HCC for curative treatment. HCC usually presents as a nodule in the liver parenchyma. However, occasionally the only manifestation of HCC is PVT.[3,4] In that situation, the diagnosis and treatment of HCC could be delayed. We describe a patient with rapidly progressive HCC who presented with PVT alone without hepatic parenchymal mass.

CASE REPORT

A 39-year-old man was admitted for hematemesis. Esophageal varices without active bleeding were detected on following endoscopy. To evaluate the etiology of the esophageal varices, contrast-enhanced abdominal computed tomography (CT) with 64 channel multi-detector row CT scanner was performed. Abdominal CT scan showed liver cirrhosis without parenchymal mass and PVT with mild enhancement in the mild dilated right portal vein (17 mm in diameter; Fig. 1A). He had diagnosed as hepatitis B-induced cirrhosis of Child-Pugh A classification. He had never known about hepatitis B infection before admission. Laboratory tests revealed the following: alpha fetoprotein (1,276 IU/mL; normal, <5.8), gamma-glutamyl transpeptidase (389 IU/L; normal, <75), aspartate amino transferase (25 IU/L; normal, <38), alanine amino transferase (8 IU/L; normal, <43), alkaline phosphatase (107 IU/L; normal, <117), total blilirubin (0.7 mg/dL; normal, <1.3), and direct bilirubin (0.2 mg/dL; normal, <0.4).

Although there was no delineated parenchymal mass on abdominal CT, an occult HCC was suspected because of the markedly elevated alpha fetoprotein. Therefore liver magnetic resonance imaging (MRI) was performed for evaluation of

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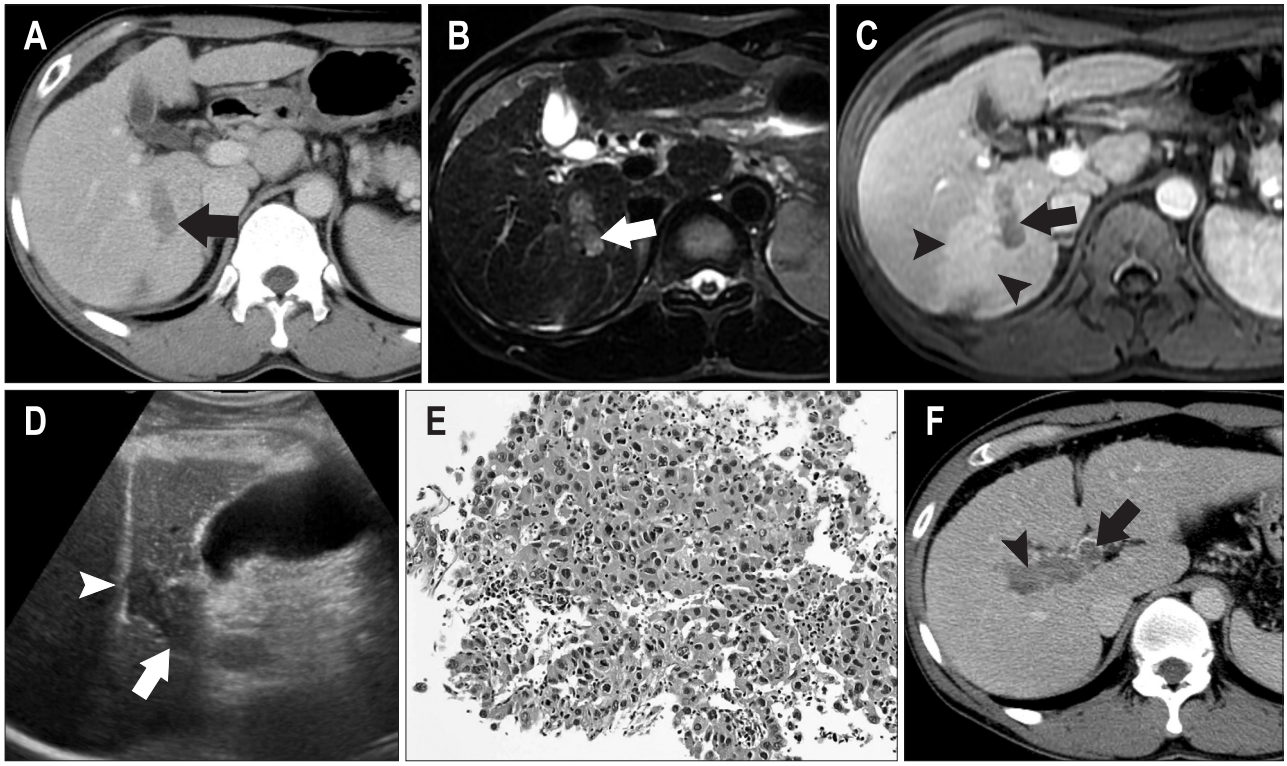

Fig. 1. (A) A contrast-enhanced computed tomography (CT) shows portal vein thrombosis (PVT) (black arrow) within the posterior segmental branch of the right portal vein. Focal capsular atrophy was seen in combination with PVT, and there was no enhancement in that area. (B) A T2-weighted image shows PVT (white arrow) with high signal intensity within the posterior segmental branch of the right portal vein. There was no parenchymal tumor surrounding the PVT. (C) A Gadolinium-enhanced T1-weighted image shows mild enhancing PVT (black arrow) with low signal intensity in the posterior segmental branch of the right portal vein. A wedge-shaped enhancement (black arrowheads) is shown in the posterior segment of the right hepatic lobe, which was the result of the secondary-enhanced parenchymal blood supplied by the right hepatic artery due to the decreased portal flow. (D) An ultrasound image shows hypoechoic PVT (white arrow) within the right portal vein of the cirrhotic liver. The 18-gauge cutting needle (white arrowhead) within the malignant PVT is shown during the percutaneous ultrasound-guided biopsy. (E) The needle biopsy sample showed a trabecular arrangement of tumor cells with abundant eosinophilic cytoplasm, suggestive of hepatocellular carcinoma (H&E stain, ×100). (F) A follow-up preoperative contrast-enhanced CT image shows the increased extent of malignant PVT (black arrow) along the left portal vein and the anterior segmental branch of the right portal vein. The more distinct enhancing area (black arrowhead) within the malignant PVT is shown within the more dilated portal vein.

HCC. Liver MRI also revealed only PVT with mild enhancement within mild dilated right portal vein (17 mm in diameter) in cirrhotic liver without evidence of parenchymal mass (Fig. 1B and C). There was no evidence of hepatic tumor, such as a HCC, on CT and MRI. Conservative treatment that focused on control of bleeding and prevention of recurrent variceal hemorrhage was done during the next 1 month. We decided to perform percutaneous ultrasound-guided biopsy of PVT and following liver MRI was performed before ultrasound-guided biopsy.

Follow-up MRI showed that the extent of enhancing PVT with high T2 signal intensity was more increased in the right portal vein and the PVT diameter was greater (22 mm) than the CT findings on admission. There was also no parenchymal mass on follow-up MRI. Percutaneous ultrasound-guided biopsy of the PVT was performed (Fig. 1D). Vascularity was detected within the PVT on color Doppler ultrasound. The PVT was pathologically confirmed to be a HCC (Fig. 1E). There was no evidence of extrahepatic involvement on imaging modalities, thus we decided to perform a right hepatectomy for curative treatment. However, the right hepatectomy could not be done, because, 20 days later, the pre-operative CT revealed that the PVT in the right portal vein had extended to the left and the main portal vein had a greater diameter (23 mm; Fig. 1F). At that time, the level of alpha fetoprotein was markedly elevated to 17,500 IU/mL. The interval between first admission and preoperative CT scan was just 50 days. The patient died after 1 year despite chemotherapy with five cycles of etoposide and epirubicine.

DISCUSSION

Several imaging findings have been used for discriminating between malignant and benign PVT. When the PVT diameter is greater than or equal to 23 mm or PVT neovascularity is present, the sensitivity of CT for identification of malignant PVT is

86%.[5] Furthermore, malignant PVT is almost always contiguous with or directly in contact with a parenchymal HCC.[5] These findings such as PVT neovascularity, marked expansion of portal vein and continuation between PVT and parenchymal HCC have been described as the differentiating points between benign and malignant PVT on contrast-enhanced CT or MRI.[5] In the current case, however, the initial abdominal CT scan showed that the thrombus in the posterior branch of the right portal vein was 17 mm in diameter. There was no discernible parenchymal HCC around the thrombosed portal vein. Mild enhancement of the PVT on contrast-enhanced CT and MRI was misinterpreted as a secondary enhanced effect of the hepatic arterial supply to the hepatic parenchyma due to the PVT. Therefore, the presumptive diagnosis was benign PVT. However, a high level of alpha fetoprotein on laboratory test suggested the presence of HCC or malignant PVT. We should have been suspicious of the possibility of malignant PVT in our case because the abdominal CT on the first admission showed mild enhancement of the PVT in the arterial phase and the laboratory data demonstrated a high level of alpha fetoprotein. The mild enhancement of the PVT suggested neovascularity within PVT and corresponded to CT criteria for malignant PVT. Follow-up CT and MRI showed that the diameter of the PVT had increased and the PVT was more distinctly enhanced.

The mechanism underlying a malignant PVT without definite parenchymal HCC has not been elucidated. One possible explanation is that a very small HCC, which exists around a portal vein, directly invades an adjacent portal vein in the early stage. To the best of our knowledge, there has been only one pathologic proven report of malignant PVT in a patient with liver cirrhosis, but without a demonstrable parenchymal mass.[4] The previous report[4] also demonstrated that HCC only presented as malignant PVT without parenchymal tumor and the serum alpha fetoprotein was markedly elevated more than 1,000 IU/mL. However, as compared to the previous report, our case showed extremely rapid progression of malignant PVT and the patient was relatively young. Moreover, in this case report we presented cross-sectional imaging findings in detail, which included the enhancement pattern and temporal change of the PVT.

The cause of rapid progression of malignant PVT in our case was unknown. It is known that male gender, age younger than 40 years, and hepatitis B virus in patients with HCC are poor prognostic factors.[6] It is also known that alpha fetoprotein is a tumor marker for HCC, with a higher level indicating possibility of greater tumor activity.[7] We suggest that these findings might have been factors for rapid progression of the malignant PVT in our case.

There are no specific or distinct different findings in the pathologic specimen compared to a typical HCC. Dramatic expansion of HCC into the main or lobular portal vein branches is often seen in HCC patients with portal vein invasion.[8] In the current case, the initial diagnosis was benign PVT and the PVT rapidly progressed along the main and left portal veins, therefore we missed an opportunity to perform a right hepatectomy as curative treatment. The prognosis for patients who have HCC with malignant PVT is extremely poor. The median survival of untreated HCC with PVT has been reported to be 2.7 months, whereas survival in those without PVT is 24.4 months.[9] In our case, the patient died after 1 year despite chemotherapy. Early detection of HCC and aggressive treatment, such as hepatectomy including portal tumor thrombectomy, can improve survival rates, thus it is important to detect malignant PVT in patients with liver cirrhosis.[10] Our case suggests that PVT alone can indicate malignant PVT, especially when the alpha fetoprotein is markedly elevated.

In conclusion, we report a case with rapid progression of malignant PVT that was not accompanied by discernible parenchymal HCC on CT and MR imaging.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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