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Radionuclide venography as a clue to the diagnosis of Budd-Chiari syndrome

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A patient was examined with radionuclide venography (RVG) to investigate unilateral leg oedema which might be due to deep vein thrombosis. RVG with Tc-99m MAA demonstrated no findings to suggest deep vein thrombosis of the right leg. However, collateral flow derived from the left common iliac vein and truncated inferior vena cava (IVC) were revealed. Contrast venography confirmed the obstruction of IVC and collateral flow from the left common iliac vein to the left ascending lumbar vein. It also showed the obstruction of hepatic veins and the patient was finally diagnosed as Budd-Chiari syndrome. Although unilateral leg oedema is an atypical symptom in Budd-Chiari syndrome, the findings on RVG led us to conduct further imaging studies to reach the diagnosis.

Key words: radionuclide venography, Budd-Chiari syndrome, inferior vena cava

INTRODUCTION

BUDD-CHIARI SYNDROME in Japan is mainly caused by membranous obstruction of IVC in its hepatic portion.[1,2] Unlike the Budd-Chiari syndrome in western countries mainly caused by thrombosis of the hepatic vein and IVC, the features of the condition in patients with membranous obstruction are often chronic and not immediately life-threatening.[2] Ascites, hepatomegaly and oedema are signs often seen in the syndrome but they are not always evident because of slow progression of the disease.[1–4] Moreover, symptoms are not always specific. These aspects of the syndrome make early diagnosis difficult.

We report a case of unilateral leg oedema as a presenting symptom but a final diagnosis of Budd-Chiari syndrome. The lack of typical signs of Budd-Chiari syndrome meant that deep vein thrombosis causing the unilateral leg oedema was first suspected in this case. The findings on RVG indicating IVC obstruction led to the further investigation and subsequent confirmation of the diagnosis.

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CASE REPORT

A 48-year-old male was admitted to our hospital for the treatment of arterio-venous malformation (AVM) of the brain. He had a past history of thrombocytopenia for which he had been treated for twenty years. On admission, slight hepatic dysfunction (total bilirubin 1.9 mg/dl, ZTT 13 K.U., TTT 4.7 M.U., GPT 23 IU/ml, GOT 44 IU/ml, γ-GPT 200 IU/ml) and oedema of the right leg were noted. He had suffered the latter for one week. Ultrasound of the legs and pelvic cavity was carried out and no evidence of thrombosis was found. By keeping the leg raised, the oedema almost disappeared three days after admission. Angiography of the brain was performed for treatment of the AVM on the 8th day after admission. After the angiography, right leg oedema reappeared.

Radionuclide venography (RVG) with Tc-99m MAA was carried out for the investigation of suspected deep vein thrombosis. A small amount of radiotracer (111 MBq) was injected into the dorsal pedal vein on both sides. Preset count images of the legs and the abdomen were obtained. RVG (Fig. 1) showed normal deep and superficial veins of the right leg. The left leg also showed no evidence of venous obstruction. However, the ascent of the radiotracer through IVC was obstructed at the level of the mid-abdominal region and collateral flow from the left iliac vein was demonstrated. These findings strongly suggested obstruction of the IVC although rapid dilution of venous flow from the lower legs could not be excluded.

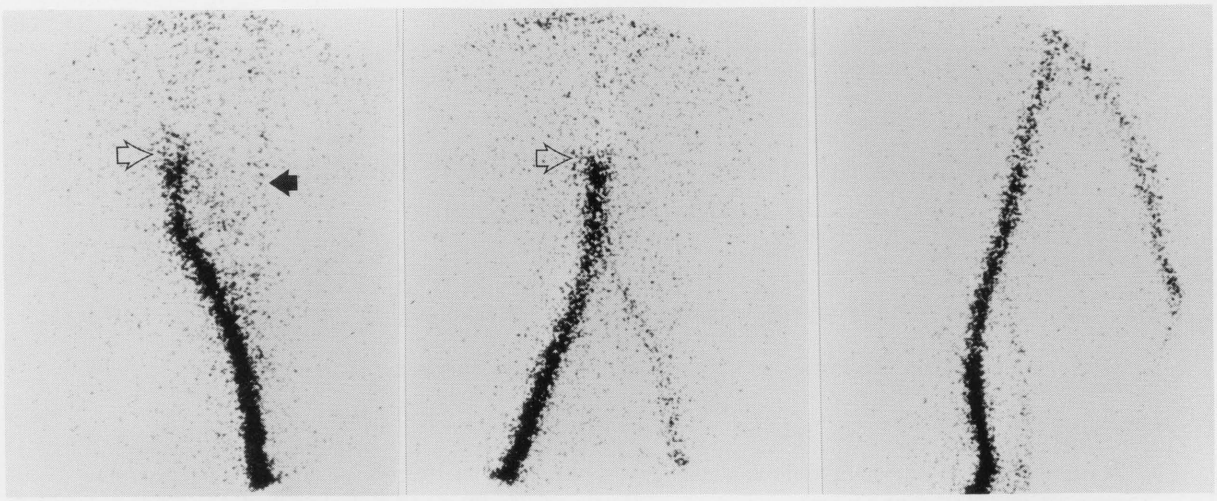

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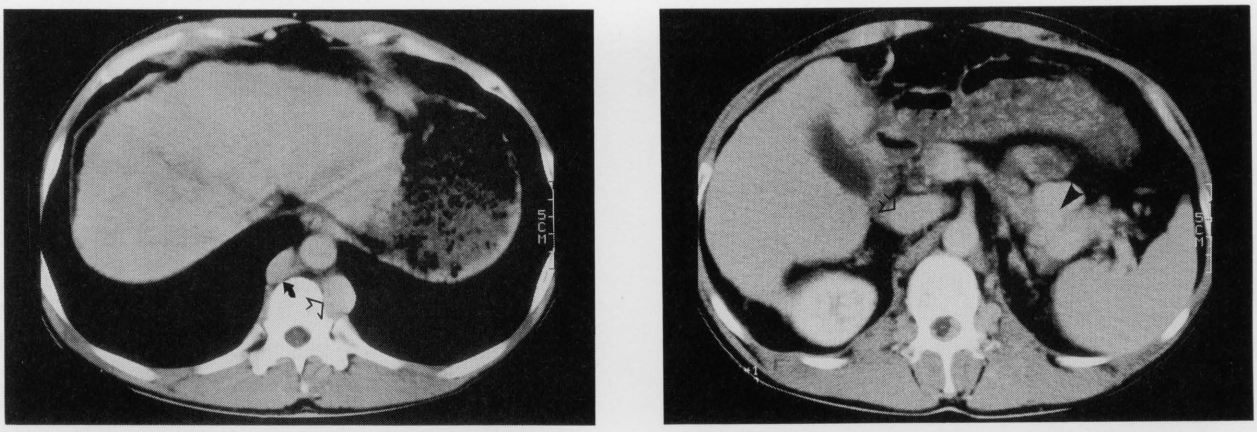
Fig. 1 Radionuclide venography. A: Left leg. No evidence of thrombosis is shown. Truncated inferior vena cava (open arrow) with hold-up of tracer and collateral (arrow) from left common iliac vein are noted. B: Right leg. The superficial vein and the deep vein are clearly visualized and truncated inferior vena cava is noted.



A B

Fig. 2 Computed tomography. A: Above the hepatic portion. The surface of the liver is serrated. No IVC is visualized, and dilated azygos (arrow) and hemiazygos veins (open arrow) are noted. B: Below the hepatic portion. Marked dilated splenic vein (arrow) is noted. IVC (open arrow) is shown beside the aorta.

Radionuclide angiography with Tc-99m labeled red blood cells was indicated to differentiate the obstruction from abnormally rapid mixture of venous flow due to arterio-venous shunting.

Radionuclide angiography of the abdomen showed no abnormality in the arterial phase and increased activity was revealed at the left side of the abdominal aorta in the blood pool image. This activity was noted to appear at about 30 seconds after injection. Hemangioma-like tumor or dilated veins were suspected.

Computed tomography (Fig. 2) of the abdomen showed marked dilated splenic vein, lumbar vein, azygos vein and hemiazygos vein. IVC was not evident above the hepatic portion. The surface of the liver was serrated and its shape showed atrophy which suggested liver cirrhosis.

Contrast venography (Fig. 3) confirmed obstruction of the IVC and collateral flow from the left iliac vein into the ascending lumbar vein. Several collaterals from the IVC were demonstrated at the site of obstruction. Some of them were connected with the obstructed hepatic veins deviating blood flow to the right atrium through the superior vena cava. Other collaterals to lumbar, azygos and hemiazygos veins from abdominal organs such as the kidneys were also demonstrated. The patient was finally diagnosed as having Budd-Chiari syndrome in view of the findings on contrast venography.

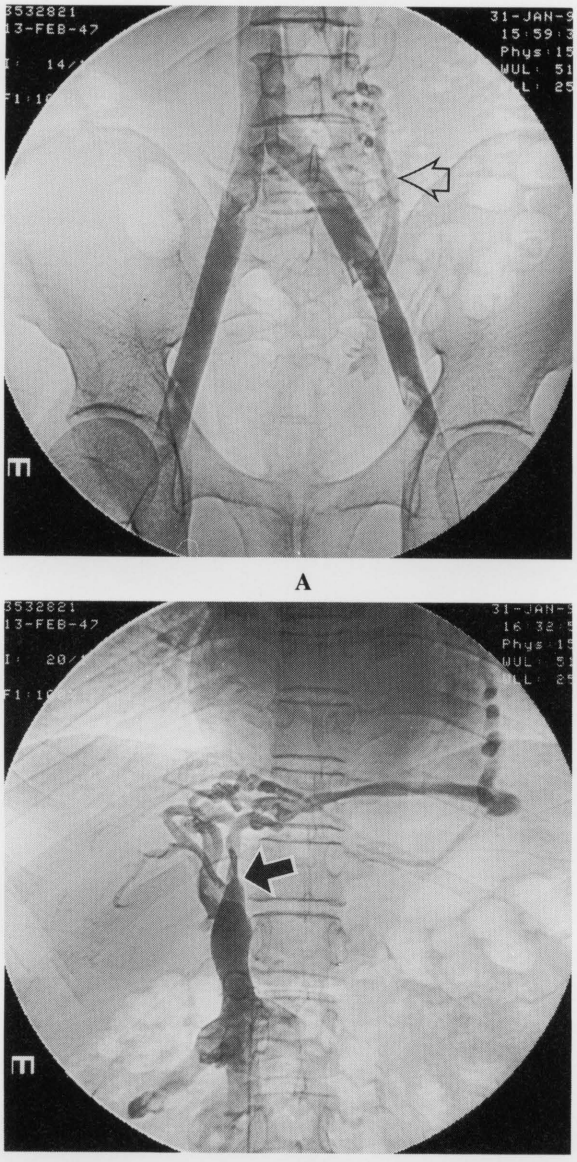


Fig. 3 Contrast venography. A: Contrast medium was released at bilateral external iliac veins. Only collateral (open arrow) from common iliac vein is noted. B: Inferior vena cava is obstructed in the hepatic portion (arrow). Several collaterals are noted at its obstruction site.

DISCUSSION

The symptoms of our case were not recognized as the signs of Budd-Chiari syndrome at the time of admission. His initial symptom of the syndrome was the unilateral leg oedema. Leg oedema is the typical initial sign of a syndrome due to IVC obstruction but usually involves both legs. This unilateral leg oedema made us suspect deep vein thrombosis of the leg rather than IVC obstruction. Slight hepatic dysfunction was the only sign to suggest the syndrome besides leg oedema. Truncated IVC on RVG was the first indication of IVC obstruction in this case. It led us to further investigations. Contrast venography confirmed obstruction of the IVC and the hepatic veins, and the patient was finally diagnosed as Budd-Chiari syndrome.

The notable finding on RVG in our case was unilateral collateral flow on the left side. This flow was confirmed to be the collateral vessels from the left iliac vein to the left ascending lumbar vein. We hypothesize that IVC obstruction caused oedema of the right leg but collateral flow of the left side prevented the left leg from developing the oedema.

The typical findings of the syndrome on RVG are described as no visualization of IVC or sharply truncated IVC with hold-up of radiotracer.[5–7] Collaterals are also demonstrated and they are usually bilateral.[5–7] In our case, truncated IVC was demonstrated but collateral was unilateral. Sy et al.[5] described a case with a partially occluded IVC in which RVG showed collaterals in the left paralumbar plexus and the lack of other abdominal channels usually associated with complete caval occlusion. The findings were similar to our case. They[5] also showed a series of RVG with several-week intervals in patient with IVC occlusion caused by thrombi. These studies demonstrated the evolution of collaterals with progression of the disease. The finding of RVG in our case may be one of the possible variations or may have occurred in the process of the evolution of collaterals.

In conclusion, RVG is a non-invasive and useful method as a first line study for Budd-Chiari syndrome. A patient might show atypical symptoms but characteristic findings on RVG can be a clue to the diagnosis of the syndrome.

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