
Radionuclide Venography in Budd-Chiari Syndrome with Intrahepatic Vena-Caval Obstruction

Miau-Ju Huang, Yun-Fan Liaw, and Kai-Yuan Tzen

Department of Nuclear Medicine and Liver Unit, Chang-Guang Memorial Hospital, Taipei, Taiwan

Radionuclide imaging of the inferior vena cava (RIVC) was performed by injecting [^{99m}Tc] phytate into a dorsal pedal vein, as an initial diagnostic procedure for eight patients with clinical features of Budd-Chiari syndrome. In five of them, membranous occlusion of the inferior vena cava (IVC) was proved by contrast venography and subsequent surgery. The other three patients, with histologically verified hepatocellular carcinoma, were proved to have tumor-induced narrowing or occlusion of the IVC by contrast venography. The RIVC findings include a sharply truncated inferior vena cava with marked hang-up of activity, extensive collaterals, and delayed visualization of the heart. Our results indicate that RIVC is as accurate and specific as contrast venography, by demonstrating the occlusion of the IVC and collateral circulation from the functional aspect. This simple and noninvasive method could therefore be used as a first-line test in patients with unexplained edema, ascites, superficial abdominal venous collaterals, and even in patients with hepatocellular carcinoma, for the detection of obstruction in the inferior vena cava.

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Budd-Chiari syndrome is a clinical entity resulting from an obstructive lesion of hepatic vein(s) or the inferior vena cava (IVC) in its hepatic or suprahepatic portion. The condition can result from a variety of causes, including clotting disorders, tumors, oral contraceptives, pregnancy, infections, and trauma (1-4). Membranous obstruction of the IVC is generally believed to result from congenital malformation, although thrombosis should also be considered. It is the most clinically promising form of Budd-Chiari syndrome because it is potentially curable by surgery (5-8). This condition is rare in the West, occurring more frequently in Orientals (6-9). Because it usually displays delayed onset and a slowly progressive course, early diagnosis and adequate treatment offer a good prognosis (6-8,10).

Contrast venography is currently the standard confirmatory diagnostic tool for obstruction of the inferior vena cava (11-13). However, contrast venography is an invasive procedure that often induces thromboembolic

processes (14-15). A noninvasive diagnostic approach is therefore desirable. Radionuclide venography, on the other hand, has been used satisfactorily in the study of IVC obstruction due to surgery or thromboembolic disorders (15,16). It was recommended as an adjunctive or even an alternative procedure in assessing patients suspected of having occlusion of the IVC (15). We have applied this procedure to evaluate patients suspected of having Budd-Chiari syndrome with IVC obstruction. This paper presents our findings and discusses the diagnostic value of radionuclide imaging of the IVC (RIVC) in comparison with contrast venography (CV).

MATERIALS AND METHODS

Eight patients with clinical features of Budd-Chiari syndrome—such as progressive engorgement of superficial veins from lower abdomen up to the thorax, long-standing ascites, intermittent edema, and stasis dermatitis—had an RIVC study as their initial diagnostic procedure. CV was performed by simultaneous catheterization and injection of contrast medium into the right

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For reprints contact: Miau-Ju Huang, MD, Dept. of Nucl. Med., Chang-Gung Memorial Hospital 199, Tung Hwa North Rd., Taipei, Taiwan 105.

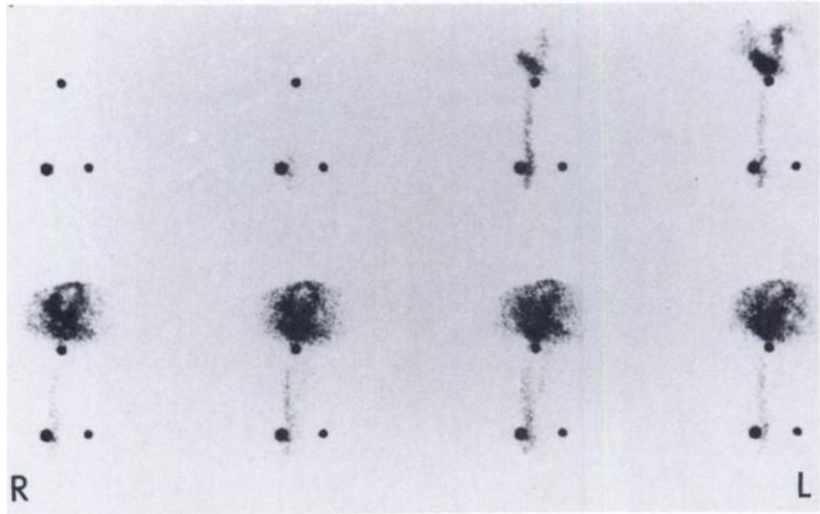


FIGURE 1
Normal radionuclide images of the IVC. Heart is visualized within 4 sec after initial visualization of inferior vena cava

atrium and IVC. Five of the patients were proved to have membranous occlusion of the IVC by CV and subsequent surgical intervention. The other three patients with histologically verified hepatocellular carcinoma (HCC) were proved by CV to have tumor-induced narrowing or occlusion of the IVC.

Three patients with functional Class III congestive heart failure (CHF) with ascites, and 21 consecutive patients with liver cirrhosis, were also studied for comparison. Thirteen of the 21 cirrhotic patients had ascites, but without other signs of Budd-Chiari syndrome. Three of them, with massive intractable ascites, were proved by CV to have a patent IVC.

RIVC was performed as follows:

1. Place radioactive markers at locations 5 cm to right

and left of the umbilicus and tip of the xyphoid process.

2. Tie tourniquets at the knee and ankle above the injection sites.

3. Inject 3–5mCi technetium-99m phytate into a dorsal pedal vein. If leg edema is prominent, the tracer is injected simultaneously into dorsal pedal veins on both sides.

4. Obtain scintiphotos every 2 sec for 1 min.

5. Perform conventional hepatic scintigraphy as usual, at 15 min after injection. Normally, the IVC is seen a few seconds after injection, and is located directly on the midline. The heart will be visualized within 4 sec following the first visualization of the lowest segment of the IVC. No collaterals will be demonstrated in normal subjects (Fig. 1).

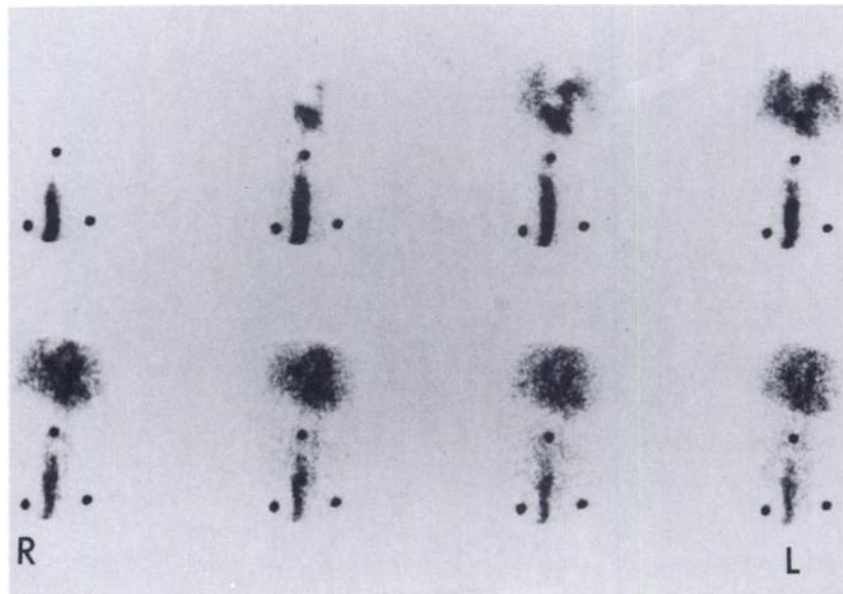


FIGURE 2
RIVC in patient with Budd-Chiari syndrome due to hepatocellular carcinoma. Note truncated inferior vena cava with hangup of radiotracer below tip of xyphoid process.

TABLE 1
Findings of Radionuclide Venography in Budd-Chiari Syndrome and Other Conditions

	Truncated IVC* with hang-up	Delayed visualization of the heart	Collaterals
Budd-Chiari syndrome			
Mem† (n = 5)	5	5	5
HCC (n = 3)	3	3	2
Total (n = 8)	8	8	7
Control			
Cirrhosis (n = 23)	0	0	0
CHF‡ (n = 3)	0	3	0

* IVC: inferior vena cava.
† Mem: membranous type.
‡ HCC: hepatocellular carcinoma.
§ CHF: congestive heart failure.

RESULTS

The RIVC findings of the eight Budd-Chiari patients with IVC obstruction are shown in Table 1. A truncated IVC with hangup of radioactivity (Fig. 2), marked collaterals (Fig. 3), and delayed visualization of the heart are three main signs indicating mechanical or functional

obstruction of the IVC. At least two signs were observed in all of the eight patients. The signs disappeared after surgical correction of the membranous IVC obstruction. Delayed visualization of heart was also observed as a single sign in the three patients with functional Class III CHF. None of the three above-mentioned positive signs was observed in cirrhotic patients, regardless of the presence or absence of ascites (Table 1). Complete correlation between RIVC and CV is shown in Table 2.

DISCUSSION

This study has confirmed that RIVC can accurately assess the patency of the inferior vena cava (15,16). Our data also indicated that this simple, noninvasive procedure was as accurate as the invasive CV and superior to ultrasonography, another noninvasive method (7-8), in the demonstration of the obstructive lesion in the inferior vena cava. Furthermore, the sequence of images in RIVC, such as delayed visualization of the heart, can demonstrate the functional occlusion of venous flow in the IVC, which is impossible by any other diagnostic modality.

CV provides the most clear-cut anatomical information and can demonstrate collaterals as well (16). However, the intrusion of a catheter, combined with the high pressure during the injection of contrast medium, could overcome a partial obstruction and make the existing collaterals invisible, as well as risk dislodging a thrombus (15). We therefore recommend RIVC as the

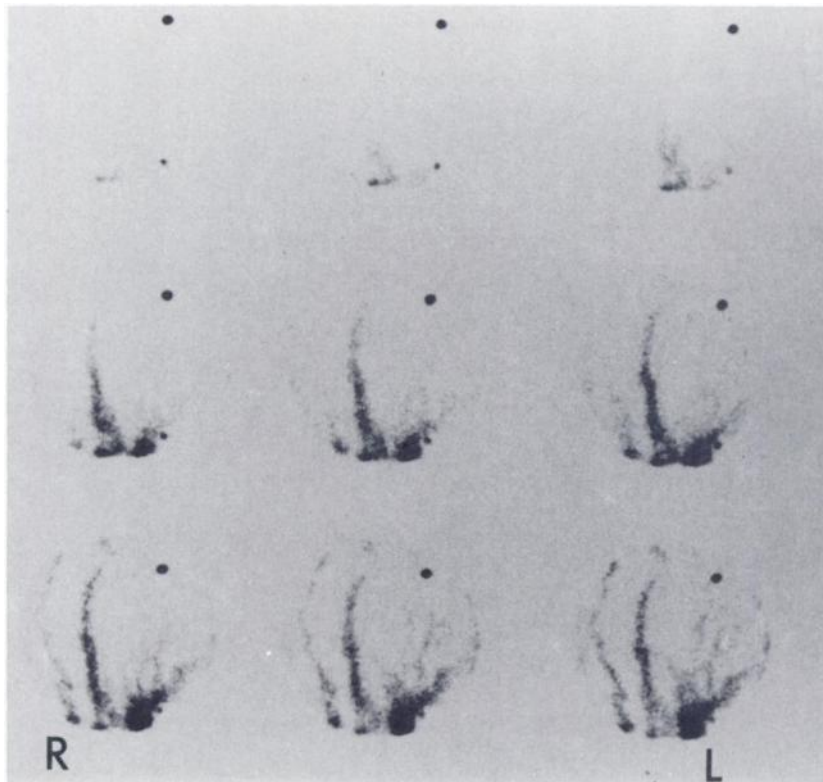


FIGURE 3
RIVC in patient with membranous type of Budd-Chiari syndrome. Extensive collaterals are visualized instead of inferior vena cava

TABLE 2
Correlation of RIVC with CV in Diagnosis
of IVC Obstruction

		RIVC	
		+	-
CV	+	8*	0
	-	0	3†

* Budd-Chiari syndrome.

† Liver cirrhosis with ascites.

RIVC: radionuclide imaging of the inferior vena cava.

CV: contrast venography.

IVC: inferior vena cava.

first-line study for IVC patency, and CV as a confirmatory study in preparation for surgical intervention.

Typically, the clinical features of Budd-Chiari syndrome with IVC occlusion are long-standing jaundice, progressive engorgement of the superficial veins, and recurrent leg edema, ascites, or even congestive cirrhosis. The condition is therefore frequently indistinguishable from liver cirrhosis due to other causes (7,9). Our data indicate that RIVC in patients with liver cirrhosis, with or without ascites, was normal and could be clearly differentiated from that of IVC occlusion (Table 1). Congestive heart failure can conceivably induce delayed visualization of the heart in RIVC, as shown in this study. Our patients with Budd-Chiari syndrome all had positive signs other than merely delayed visualization of the heart, thus simplifying differential diagnosis. Therefore, RIVC was not only very sensitive but also highly specific in the detection of Budd-Chiari syndrome with IVC obstruction.

Conventional hepatic scintigraphy has some value in the diagnosis of Budd-Chiari syndrome, by demonstrating selective colloid uptake by the caudate lobe in patients with occluded hepatic vein but patent IVC (8, 10). This condition accounts for 31% of Budd-Chiari syndromes in Orientals (6), and conceivably cannot be detected by contrast inferior venocavography or RIVC per se. However, conventional hepatic scintigraphy was included as a routine part of RIVC described in this study. This practice would detect most of the patients with Budd-Chiari syndrome. In addition, Budd-Chiari syndrome induced by liver disease, for example hepatocellular carcinoma or cirrhosis, could also be detected by the same procedure.

Accordingly, this simple, safe, accurate, and reproducible procedure may be used as a first-line procedure in assessing patients suspected of having occlusion of the IVC (15), including Budd-Chiari syndrome. We also recommend adding this procedure to conventional hepatic scintigraphy for patients with clinically suspected liver cirrhosis, particularly those with intractable ascites.

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