Comparison of ultrasonography, computed tomography and ^{99m}Tc liver scan in diagnosis of Budd-Chiari syndrome

S GUPTA, S BARTER, G W L PHILLIPS, R N GIBSON,
AND H J F HODGSON

From the Departments of Medicine and Radiology, Hammersmith Hospital, Royal Postgraduate Medical School, London

SUMMARY Ultrasonography, computed tomography and ^{99m}Tc liver scanning are all useful in diagnosis of patients with the Budd-Chiari syndrome. In a study to determine their comparative value characteristic findings were recorded in all nine patients at ultrasonography and in seven patients at computed tomography. In contrast ^{99m}Tc liver scan showed a characteristic pattern in only one of eight patients. In our experience intrahepatic venous abnormalities were seen better at ultrasonography than at computed tomography. In addition, abnormality in the direction of blood flow could be detected by pulsed Doppler examination. Ultrasonography is relatively inexpensive, readily accessible, does not require administration of radiation or contrast agents and therefore should be the primary non-invasive investigation of patients with Budd-Chiari syndrome, or those at risk of developing it.

The diagnosis of Budd-Chiari syndrome is generally made by either liver biopsy or hepatic venography.¹⁻³ Although it is often commented that liver biopsy may not be possible in many of these patients,⁴⁻⁵ this has been only occasionally so in our experience and that of others.^{1,2} The major disadvantages of liver biopsy and venographic studies, however, are that they are invasive, require expertise and may be available only in specialised centres. Furthermore, neither of these techniques is ideally suited for screening or monitoring the course of the illness. Budd-Chiari syndrome may be more common than is realised as a diagnosis may be made more often with greater awareness^{6,7} and therefore rapid, non-invasive means which might suggest the condition are desirable.

Traditionally, isotope liver scan has been utilised for this purpose and may show a characteristic pattern for central uptake in patients with Budd-Chiari syndrome.^{1,2,7-10} Both computed tomography^{5,11,12} and ultrasound^{5,12-16} have recently been found to be useful in the diagnosis of Budd-Chiari

syndrome. Of these two, ultrasound is advantageous because it is cheaper and widely available. Experience with ultrasonography in Budd-Chiari syndrome, however, is limited.

We have studied the diagnostic features at ultrasonography in nine patients of Budd-Chiari syndrome. A comparison with computed tomography and ^{99m}Tc liver scanning was carried out to determine which of the three should be the non-invasive investigation of choice.

Methods

PATIENTS

Studies were carried out on nine patients with Budd-Chiari syndrome. Four patients were of a recent onset, five had long standing disease. The diagnosis was made in all patients by hepatic venography and in addition liver histology in eight patients showed a characteristic pattern of sinusoidal dilatation and congestion along with perivenous hepatocellular atrophy.^{2,3} The clinical details of these patients are summarised in Table 1. There were four men and five women in the age range of 27-48 years.

Address for correspondence: Dr S Gupta, USC Liver Unit, Rancho Los Amigos Hospital, 7601 E. Imperial Highway, Downey, CA 90242, USA.

Received for publication 10 July 1986.

Table 1 Clinical characteristics of patients

	Age	Sex	Duration of symptoms before diagnosis (months)	Associated illnesses	Ascites	Hepatomegaly	Splenomegaly
1	40	F	6	Nil	+	+	-
2	45	M	20	Heterozygous α -1 antitrypsin deficiency	+	+	+
3	33	F	4	Nil	+	+	+
4	43	M	1	Systemic vasculitis	+	+	+
5	29	F	44	Nil	++	-	-
6	42	F	7	Nil	+++	+	+
7	27	M	2	Paroxysmal nocturnal haemoglobinuria	+++	+	++
8	48	M	3	Nil	+	+	-
9	28	F	1	Nil	-	++	-

The radiological investigations done in the five patients with chronic disease were reviewed. Ultrasonography, ^{99m}Tc liver scans, and venographic studies were done in all patients at the time of the initial referral and all but one patient also had computed tomography, which was carried out simultaneously in all patients. The duration of symptoms before the diagnosis was made is listed in Table 1. One patient in this group died (patient 2, Table 1), but the remaining four have been under regular follow up and all had recent ultrasound scans to verify previous findings. Ultrasonography was the initial investigation in the four patients of recent onset and the diagnosis was confirmed subsequently by angiography in all. Three of these patients had ^{99m}Tc liver scans and all had computed tomography of the abdomen. Follow up ultrasound scans were carried out within two to four weeks on all these patients.

EQUIPMENT

Ultrasonography

Real time ultrasound examination was carried out using a Toshiba SAL 50A, 5 MHz linear array scanner or a Diasonics 400 CV 3.5 MHz mechanical sector scanner. In addition the direction of flow in venous collaterals was determined in three patients using a Diasonics 3.5 MHz cardiac probe with 3.0 KHz Doppler.

All examinations included assessment of the confluence of veins draining into the inferior vena cava, presence or absence of the hepatic veins, alterations in the calibre or the shape of the intrahepatic veins, presence of intrahepatic collaterals, enlargement of the caudate lobe and narrowing of the inferior vena cava. Presence of splenomegaly, ascites and extrahepatic venous collaterals including azygos, hemiazygos and umbilical vein were noted.

Computed tomography

Examinations were carried out on a Siemens Somatom 2 scanner with contiguous slices of 8 mm thickness and a scan time of five seconds. Scans were

done before and after injection of contrast medium (100 ml Urografin 310 M injected intravenously at the rate of 1 ml/sec). All patients also received 1 litre of diluted oral Gastrografin before the examination to opacify bowel loops.

The presence or absence of hepatic veins was noted and hypertrophy of caudate lobe was determined by using the previously described caudate/right lobe ratio.^{5,6} In addition presence of parenchymal abnormalities, distortion or narrowing of the inferior vena cava, presence of dilated azygos vein or collaterals, ascites and splenomegaly were noted.

^{99m}Tc liver scanning

Technetium -99m labelled sulphur colloid (Amersham International) was used for isotope scanning. Two millicuries (80 MBq) dose of the isotope was administered intravenously and scanning was started 10 minutes later. An International General Electric Maxi camera 400A on line to a Medtronics Data Systems computer was used to take views in anteroposterior, posteroanterior, right and left lateral positions. Tomographic sections were also generated.

Presence of liver or splenic enlargement, homogeneity of uptake of the isotope by the liver and caudate lobe enlargement were determined.

Results

The findings at radiological investigations are summarised in Table 2. There were abnormalities on ultrasound examination in all nine patients. The normal confluence of hepatic veins into the inferior vena cava could not be demonstrated in any patient, although it is generally an easily recognised feature in normal individuals (Fig. 1). Clearly defined, outflowing hepatic veins were absent although distorted middle and right hepatic veins were present in one patient (Fig. 2) and a left hepatic vein with an irregular calibre could be seen in two patients. In these three patients pulsed Doppler examination

Table 2 Summary of various radiological investigations

Patient no	Hepatic venography	Liver biopsy	Liver scan	CT		Ultrasound					
				Abnormal parenchyma	Caudate/right lobe ratio	Intrahepatic veins	IVC	Confluence at IVC	Intrahepatic veins and collaterals	Caudate lobe	IVC
1	Stenosis and partial occlusion of main hep veins. Multiple collaterals. IVC patent	+	Slight enlargement left lobe	+	Enlarged caudate lobe (1.0)	Absent	Narrowed	Absent	Distorted middle and right hepatic vein	Enlarged	Narrowed
2	Major hep veins occluded. Spider-web collaterals. IVC displaced and narrowed	+	Central accumulation. Spleen enlarged	-	Enlarged caudate lobe (1.7)	Absent	Narrowed	Absent	Abnormal collaterals only	Enlarged	Narrowed
3	Complete occlusion hep veins. IVC narrowed	+	Areas of patchy uptake. Spleen enlarged	+	Enlarged caudate lobe (0.67)	Absent	Normal	Absent	Abnormal collaterals only	Enlarged	Normal
4	Occlusion of all except one narrowed vein. IVC normal	+	Patchy uptake. Spleen enlarged	-	Possible enlargement (0.56)	Inadequate study		Absent	Abnormal venous channels only	Enlarged	Normal
5	Complete occlusion hep veins. IVC patent	+	Slight liver enlargement	Not performed				Absent	Only one hep venous collateral draining into IVC	Enlarged	Normal
6	Narrowed and irregular left vein, dilated right vein. IVC patent	+	Patchy uptake. Spleen enlarged	-	Enlarged caudate lobe (0.88)	Absent	Normal	Absent	Enlarged and irregular hep veins	Enlarged	Normal
7	Occlusion of all hep veins. Spider-web collaterals. IVC normal	+	Slightly patchy uptake. Spleen enlarged	+	Enlarged caudate lobe (0.94)	Absent	Normal	Absent	Only stenosed, irregular left hep vein	Enlarged	Narrowed
8	Left hep vein occluded. Right hep vein normal but tributaries occluded. IVC patent	+	Patchy uptake	-	Enlarged caudate lobe (0.85)	Absent	Normal	Absent	Abnormal collaterals only	Enlarged	Normal
9	All hepatic veins occluded. IVC patent	-	Not performed	+	Enlarged caudate lobe (0.64)	Absent	Normal	Absent	Left hep vein only seen	Enlarged	Normal

Fig. 1 Transverse scan of liver showing normal confluence of hepatic veins and inferior vena cava (IVC).

Fig. 2 Transverse ultrasound scan of the liver in patient with Budd-Chiari syndrome, showing a tortuous intrahepatic venous collateral.

showed that the blood flow *via* their veins was in an abnormal direction, away from the hepatic vein ostia. The venous structures which could be demonstrated on the region of the ostia were prominent collateral network of small, tortuous vessels. This pattern was seen in all patients with long standing disease. The caudate lobe was enlarged in all cases (Fig. 3).

In three patients the inferior vena cava was narrowed and in two it was almost occluded by the enlarged caudate lobe. Less specific findings as a result of portal hypertension included enlarged retro-peritoneal collaterals from the azygos and hemi-azygos systems, patent umbilical vein, splenomegaly and ascites (Fig. 4). Five patients were known to have gastro-oesophageal varices at endoscopy but these were not detected by ultrasonography, known to be inferior to endoscopy in detecting varices.¹⁷ Follow up ultrasound scans in four patients with recent onset of symptoms showed development of abnormal intrahepatic venous structures and collaterals. It was difficult to quantitate rigorously the extent of collateral flow or the degree of caudate lobe enlargement.

Fig. 3 Longitudinal ultrasound scan in Budd-Chiari syndrome showing prominent caudate lobe (CL) between markers (patient's head end to left).

Computed tomography showed an enlarged caudate lobe with low attenuation elsewhere in seven patients and equivocal enlargement in one patient. In four patients the caudate lobe was enlarged with abnormal attenuation. Intrahepatic veins did not fill after administration of contrast in seven patients. In one patient postcontrast studies were inadequate to

Fig. 4 Longitudinal ultrasound scan through left lobe of liver in Budd-Chiari syndrome (patient's head end to left) showing abnormal intrahepatic venous channels caudally, and tortuous hemiazygous collaterals beneath the inferior surface of the liver.

evaluate venous filling. Other abnormalities were demonstration of narrowing of the inferior vena cava by the enlarged caudate lobe in two patients, ascites in four patients, enlarged azygos vein in two patients, and enlargement of spleen in five patients.

Of the three investigations ^{99m}Tc liver scan was the least specific. Central accumulation of the isotope as described characteristically with caudate lobe enlargement^{1,2,8-11} was seen in only one of eight patients. Liver or splenic enlargement was identified and areas of patchy uptake of the isotope were present in five patients.

If demonstration of abnormalities in hepatic veins or enlargement of caudate lobe are taken as specific diagnostic features, although caudate lobe enlargement is of course possible in other circumstances, ultrasonography and computed tomography were successful in making a diagnosis in all patients, and ^{99m}Tc liver scanning only 12.5%.

Discussion

Before the advent of ultrasonography^{5,12-15} and computed tomography,^{5,12,13} non-invasive screening and monitoring of patients with Budd-Chiari syndrome was carried out by ^{99m}Tc liver scanning.^{1,2,8,11} ^{67}Ga liver scanning has recently been reported to be useful.¹⁸ In our experience, however, the characteristic central accumulation of the isotope is seen rarely and liver scans are a relatively poor means of making a diagnosis, perhaps because of variable preservation of liver function in different lobes.^{2,13,19}

This study indicates that the diagnostic accuracy of ultrasound in Budd-Chiari syndrome is similar to angiography. Absence of hepatic veins at the normal venous confluence at the inferior vena cava, abnormal intrahepatic collaterals and enlarged caudate lobe are of maximum discriminant value. Computed tomography is similarly successful in suggesting the diagnosis.^{5,12,13} The overall pattern of venous abnormalities and intrahepatic collaterals, however, is relatively clearer at ultrasonography and in suitably trained hands a diagnosis can possibly be made with greater conviction.

There are a number of other reasons for which ultrasound theoretically may prove to be advantageous in the diagnosis of Budd-Chiari syndrome. It has been recognised recently that in this condition venous occlusion may not be total and partial thrombosis has been described both in patients¹⁹⁻²¹ and in experimental conditions.¹⁹ In these circumstances an inability to cannulate the venous ostia at venography, or non-filling of hepatic vein side branches, both of which are taken as evidence of venous occlusion, may generate erroneous information which could be corrected by ultrasonography. Narrowing or dis-

placement of the inferior vena cava by an enlarged caudate lobe in some patients may simulate lesions such as tumours or abscesses. Venography alone does not differentiate between such lesions and further imaging by computed tomography or arteriography is often necessary.²¹ In the Far East venous webs are relatively common cause of Budd-Chiari syndrome. The ultrasonographic appearances of webs in the inferior vena cava are not known to us, and as webs are very thin,²² it is conceivable that they might only be detected with difficulty, but proximal dilatation of the hepatic veins might be expected. Another advantage of ultrasound is the demonstration of features of portal hypertension such as enlarged retroperitoneal veins or a patent umbilical vein, which may reflect the severity of venous occlusion of liver damage, and also associated portal vein or splenic vein thrombosis are obviously not demonstrable by hepatic venography alone.

When ultrasound examination was repeated over two to four weeks in patients with recent onset of symptoms, it was possible to see evolution of the lesions, notably with development of intrahepatic collaterals. The direction of flow in collaterals was also demonstrable using pulsed Doppler ultrasound, which showed abnormal intrahepatic channels with blood flowing away from the site of the hepatic vein ostia. Although we have no evidence from this study, it is possible that ultrasound monitoring of the development of collaterals might correlate well with a good outcome from conservative management, or alternatively define a group of individuals with poor collateral development, in whom surgical intervention might be required. We hope that prospective studies will investigate this.

The evidence in this study, however, shows that ultrasound is an effective tool in the detection of Budd-Chiari syndrome, clearly superior to isotope liver scan, and detection of normal ultrasonic findings in a patient in whom the diagnosis of Budd-Chiari has been considered should obviate the need for angiographic demonstration of the hepatic veins.

References

- 1 Tavill AS, Wood EJ, Krel J, Jones EA, Gregory M, Sherlock S. The Budd-Chiari syndrome: correlation between hepatic scintigraphy and the clinical, radiological and pathological findings in nineteen cases of hepatic venous outflow obstruction. *Gastroenterology* 1975; **68**: 509-18.
- 2 Mitchell MC, Boitnott JK, Kaufman S, Cameron JL, Maddrey WC. Budd-Chiari syndrome: etiology, diagnosis and management. *Medicine* 1982; **61**: 199-218.
- 3 Clain D, Freston J, Krel L, Sherlock S. Clinical diagnosis of Budd-Chiari syndrome. A report of six cases. *Am J Med* 1967; **43**: 544-54.
- 4 Maddrey W. Hepatic vein thrombosis (Budd-Chiari syndrome). *Hepatology* 1984; **4**: 445-65.
- 5 Baert AL, Fevery J, Marchal G, *et al*. Early diagnosis of Budd-Chiari syndrome by computed tomography and ultrasonography: report of five cases. *Gastroenterology* 1983; **84**: 587-95.
- 6 Harbin WP, Robert NJ, Ferrucci JT. Diagnosis of cirrhosis based on regional changes in hepatic morphology. A radiological and pathological analysis. *Radiology* 1980; **135**: 273-83.
- 7 Gupta S, Cuss FM, Crofton ME, Baron JH, Hodgson HJF. Budd-Chiari syndrome and alpha-1 antitrypsin deficiency. *Hepato-Gastroenterol* 1985; **32**: 253-4.
- 8 Meindok H, Langer B. Liver scan in Budd-Chiari syndrome. *J Nucl Med* 1976; **17**: 365-8.
- 9 Thijs LG, Heidendal GAK, Huijgens PC, Mol JJ, Verkuyl M. The use of nuclear medicine procedures in the diagnosis of Budd-Chiari syndrome. *Clin Nucl Med* 1978; **3**: 389-92.
- 10 Staab EV, Hartman RC, Parrott JA. Liver imaging in the diagnosis of hepatic venous thrombosis in paroxysmal nocturnal haemoglobin-uria. *Radiology* 1975; **117**: 341-8.
- 11 Rossleigh MA, Uren RF, Morris JG. Radionuclide liver scan. A screening test for the Budd-Chiari syndrome. *Med J Aust* 1984; **140**: 234-6.
- 12 Rossi P, Sposito M, Simonetti G, Sposato S, Cusumano G. CT diagnosis of Budd-Chiari syndrome. *J Comput Assist Tomogr* 1981; **5**: 366-9.
- 13 Yang PJ, Glazer GM, Bowerman RA. Budd-Chiari syndrome: computed tomography and ultrasonographic findings. *J Comput Assist Tomogr* 1983; **7**: 148-50.
- 14 Cooper RA, Picker RH, Fulton AJ, Lunzer MR. The ultrasonic appearance of the liver in hepatic venous outflow obstruction (Budd-Chiari syndrome). *J Clin Ultrasound* 1982; **10**: 35-6.
- 15 Makuuchi M, Hasegawa H, Yamazaki S, Moriyama N, Takayasu K, Okazaki M. Primary Budd-Chiari syndrome: Ultrasonic demonstration. *Radiology* 1984; **152**: 775-9.
- 16 Powell-Jackson P, Karani J, Ede R, Meire H, William R. Diagnosis of Budd-Chiari syndrome by liver ultrasound and colloid scintigraphy. [Abstract]. *Gut* 1985; **26**: A568.
- 17 Subramanyam BR, Balthazer EJ, Madamba MR, Raghavendra BN, Horri SC, Lefleur RS. Sonography of portosystemic venous collaterals in portal hypertension. *Radiology* 1983; **146**: 161-6.
- 18 Garty I, Horovitz I, Kenyan A. The use of gallium-67 liver imaging for the early diagnosis of Budd-Chiari syndrome. *J Nucl Med* 1984; **25**: 320-2.
- 19 Saint-Marc Girardini MF, Zafrani ES, Prigent A, *et al*. Unilobar small hepatic vein obstruction: possible role of progestogen given as oral contraceptive. *Gastroenterology* 1983; **84**: 630-5.
- 20 Maguire R, Doppman JL. Angiographic abnormalities in partial Budd-Chiari syndrome. *Radiology* 1977; **122**: 629-35.
- 21 Nakamura H, Tamaka J, Horii S, Yashioka H, Kuroda C. Partial Budd-Chiari syndrome. *J Comput Assist Tomogr* 1982; **6**: 833-5.
- 22 Takeuchi J, Takada A, Hasumura Y, Matsuda Y, Ikegami F. Budd-Chiari syndrome associated with obstruction of the inferior vena cava: A report of seven cases. *Am J Med* 1971; **51**: 11-20.