Doppler Ultrasonography of the Liver: What Every General Radiologist Should Know

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Learning objectives

The purposes of this exhibit are:

- To review the bases on how to adequately perform a Doppler study in order to correctly interpret it.

- To review the normal hemodynamic for each of the major hepatic vessels (hepatic artery, hepatic vein, and portal vein) that should be explored in a Doppler ultrasound of the liver and to recognize their abnormal waveform patterns in the context of the main pathologies.

Background

Doppler ultrasound of the liver is a noninvasive technique, operator-dependent, which may provide relevant information in the study of common conditions such as portal hypertension, passive hepatic congestion and complications of liver transplantation.

To correctly interpret a Doppler study, we must know how to adequately perform it and the normal hemodynamic of the hepatic vessels.

Normal waveforms have characteristic appearances, and the majority of liver diseases cause only a limited number of abnormal waveform patterns.

Basic concepts in Doppler ultrasonography (US):

Three basic levels of US should be performed Fig. 1 :

- 1st level: **gray-scale** US (B-mode imaging).

- 2nd level: **color Doppler** examination (produces an image that shows blood flow in vessels).

- 3rd level: **spectral Doppler** examination (over a vessel of interest, produces a spectral Doppler waveform).

The waveform is obtained from a small sample volume that is placed in the center of the vessel (ideally, in the midportion of the lumen for optimal estimation of laminar flow) by the sonographer.

An angle indicator line is subjectively placed parallel to the vessel. The **Doppler angle** (between the actual Doppler beam and the Doppler interrogation line) should be **less than 60°** to avoid errors into the final velocity calculation.

Every spectral waveform has morphologic features that provide information regarding:

- **Direction**: whether the waveform lies above (blood flow toward the transducer) or below (blood flow away from the transducer) the baseline.

- **Velocity** (distance from the baseline at any given point on the curve)

- **Acceleration** (the slope of the curve, rate of change in velocity).

The terms ***antegrade*** (in the forward direction) and ***retrograde*** (in the reverse direction) refer to flow with respect to its expected direction in the circulatory system. Thereby, antegrade flow may be either toward the transducer (e.g. hepatic artery) or away from the transducer (e.g. hepatic vein).

Findings and procedure details

Normal arterial and venous hemodynamics:

Arteries normally have a **pulsatile** flow with marked undulation in the waveform (steep slopes and a wide vertical range between inflections).

There are low and high resistance arteries. This is confirmed by calculating a Resistive Index (RI):

- **Low-resistance arteries** are internal carotid arteries, hepatic arteries Fig. 2 , renal arteries and testicular arteries (vessels to parenchymatous organs). The lowest point (trough) of the waveform at end diastole is high therefore there is relatively more flow during diastole. They all have normal **RIs ranging from 0.55 to 0.7**.

- **High-resistance arteries** include external carotid arteries, extremity arteries and fasting mesenteric arteries. The trough is low and there is relatively less flow during diastole. They normally have **RIs over 0.7**.

Normal veins have a **phasic** waveform Fig. 3 (mild undulation, shallow slopes and a small vertical range between inflections) and they are low-resistance vessels.

Hemodynamics of the normal vessels of the liver:

The hepatic artery Fig. 2 is a **low-resistance** vessel (because the liver requires continuous blood flow), with a **pulsatile** waveform. In general, low-resistance arteries normally have an RI of 0.55–0.7; however, wider normal ranges of 0.55–0.81 have been reported for this vessel. Any measured RI above or below the normal range may represent disease (high resistance is less specific for disease than is low resistance).

The normal hepatic venous waveform Fig. 4 has historically been called **triphasic**; in reality, however, it is biphasic with **predominantly antegrade** flow and four inflection points (there is a complex alternating antegrade-retrograde flow variations, created by pressure variations related to the cardiac cycle).

In the portal venous Fig. 3 , physiologic flow is always **antegrade** and **hepatopetal** (toward the transducer, waveform above the baseline). Flow velocity in this vessel is relatively low (16–40 cm/sec) compared with that in the vessel coursing next to it (the hepatic artery). Normal **phasicity** may range from low to high. Abnormally low phasicity results in a nonphasic waveform, whereas abnormally high phasicity results in a pulsatile waveform.

Portal hypertension:

Portal hypertension is caused by cirrhosis in the vast majority of cases. However, there is an exhaustive list of causes, divided into prehepatic (e.g. portal vein thrombosis), intrahepatic (e.g. cirrhosis from any cause), and posthepatic (right-sided heart failure, tricuspid regurgitation, Budd-Chiari syndrome) causes.

The most specific findings for portal hypertension are development of **portosystemic shunts** (e.g. a **recanalized umbilical vein** Fig. 5 , which originates from the left portal vein and courses inferiorly through the falciform ligament and along the anterior abdominal wall) and slow or reversed (hepatofugal) flow. Abnormally **slow portal venous flow** is diagnostic for portal hypertension when peak velocity is less than 16 cm/sec.

Absent flow in the portal vein may be due to stagnant flow (portal hypertension) or occlusive disease caused by bland or malignant thrombosis Fig. 6 . If the portal vein is only partially blocked (nonocclusive intraluminal filling defects), there will be some degree of flow, which may be increased at the stenosis. Recanalization occurs in some cases of thrombosis but, more frequently, if portal vein thrombosis persists, portal flow is reestablished via **cavernous transformation** Fig. 7 (portal vein undergoes fibrosis and a tangle of tortuous collateral veins are seen along the usual course of the portal vein).

A **dilated portal vein** (portal vein diameter exceeding 13 mm) is also a possible finding in portal hypertension.

Splenomegaly and **ascites** are nonspecific and may be seen in other pathologic conditions.

Hepatic venous congestion:

Congested liver because of right-sided Congestive Heart Failure (CHF) manifests as **dilated hepatic veins** at gray-scale US Fig. 8 (easily distinguished from cirrhosis, which manifests as compressed hepatic veins), with **turbulent flow** appearance on color Doppler and **pulsatile waveform** on pulsed Doppler (whereas cirrhosis causes a decreased hepatic venous phasicity and spectral broadening Fig. 8).

Flow in the hepatic veins is pulsatile when both the antegrade and retrograde velocities

are increased relative to physiologic states (this creates a waveform with dramatic fluctuations). There are two conditions that can create a pulsatile hepatic venous waveform, both of which are also associated with a **pulsatile portal venous** waveform: tricuspid regurgitation and right-sided heart failure without tricuspid regurgitation (these two entities can be differentiated by analyzing the hepatic venous waveform).

The congested liver usually has a **hypoechoic parenchyma** and there can be some ancillary findings like **ascites**, **pleural effusion**, **thickened visceral walls** (gallbladder, bowel, stomach), **splenomegaly** and **cardiomegaly**.

Complications of liver transplantation:

Orthotopic liver transplantation is the only definitive treatment for irreversible acute liver failure and chronic liver disease.

After transplantation, **normal hepatic arterial RI ranges from 0.55 to 0.8** Fig. 9 (however, in the immediate postoperative period, some patients have an elevated RI that returns to a normal waveform within a few days).

High hepatic arterial resistance (RI >0.8) may be seen in cold ischemia posttransplantation and any stage of **transplant rejection**. Low hepatic arterial resistance (RI <0.55) is usually associated with proximal arterial narrowing, which in case of liver transplant may mean a stenosis in the hepatic artery anastomosis.

Stenosis flow dynamics**:** Velocities are increased within a stenotic portion of a vessel, and the RI is increased when the stenosis is downstream but decreased when the stenosis is upstream. A waveform whose contour is affected by an upstream stenosis (**proximal stenosis**) is described as a **tardus-parvus waveform** (late and low appearance of the peak of the waveform).

Conclusion

An understanding of the basic principles of vascular Doppler US is required to successfully perform liver Doppler US.

The major teaching points of this exhibit are:

- The three steps of a Doppler ultrasound study: gray-scale, color Doppler and spectral Doppler. The relevance of the Doppler angle for the proper estimation of flow velocity.

- The patterns and characteristic normal waveforms of hepatic arteries, hepatic veins and portal veins, and their alterations in situations of portal hypertension and passive congestion of liver. The relevance of the study of the hepatic artery after liver transplantation and the post-stenotic parvus tardus waveform.

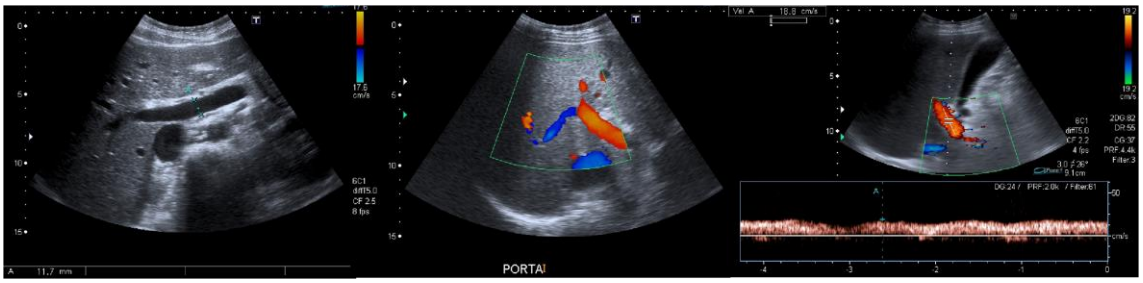

Fig. 1
Annotation:
Three levels of US in a normal portal vein: gray-scale, color Doppler and spectral Doppler.

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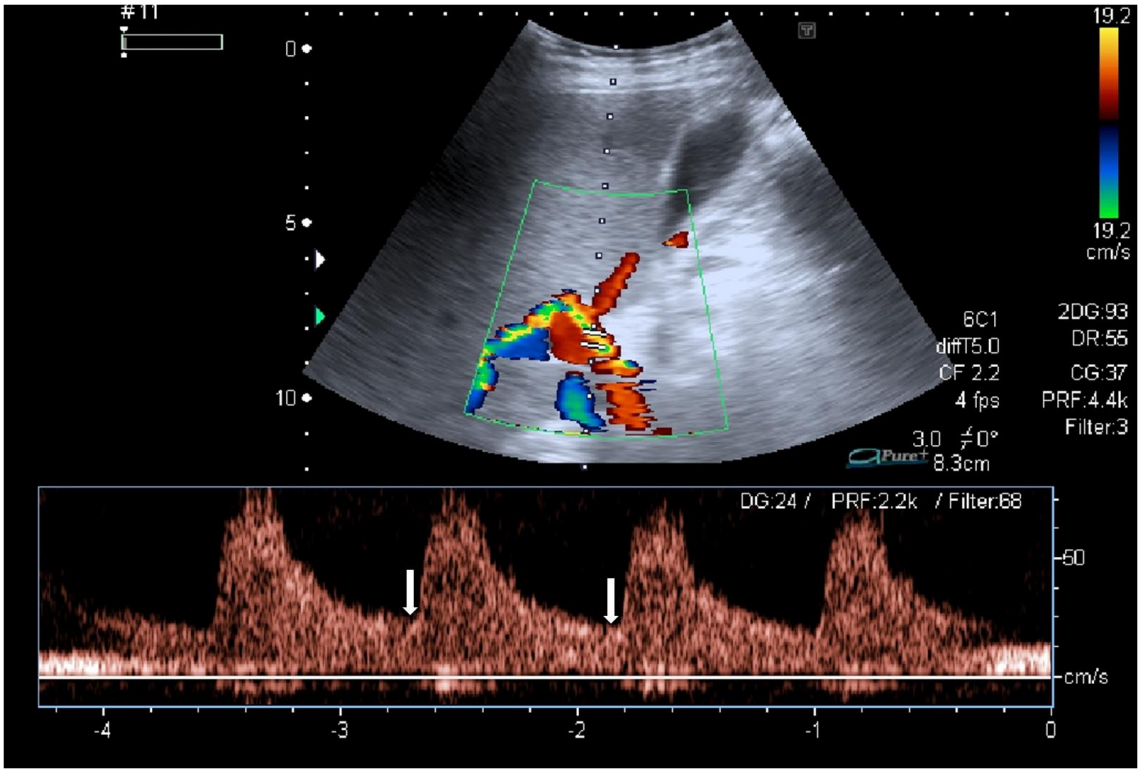

Fig. 2
Annotation:
Hepatic artery: antegrade flow (displayed above the baseline) toward the liver, typical spectral Doppler waveform (pulsatile flow, normally seen in arteries). It's a low-resistance artery, the trough of the waveform at end diastole is high (arrows).

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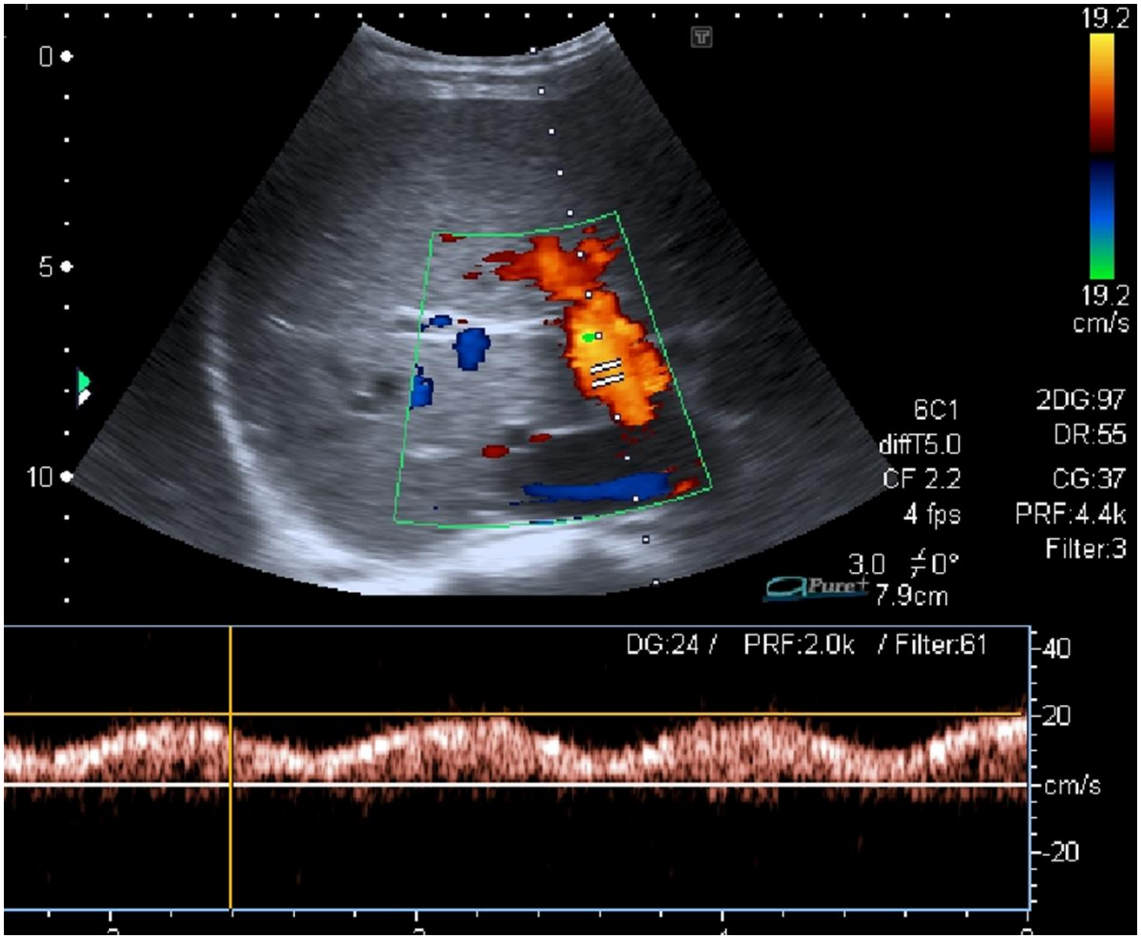

Fig. 3
Annotation:
Normal portal venous flow direction and waveform: the direction of flow is antegrade or hepatopetal (waveform above the baseline) and there is a normal phasicity.

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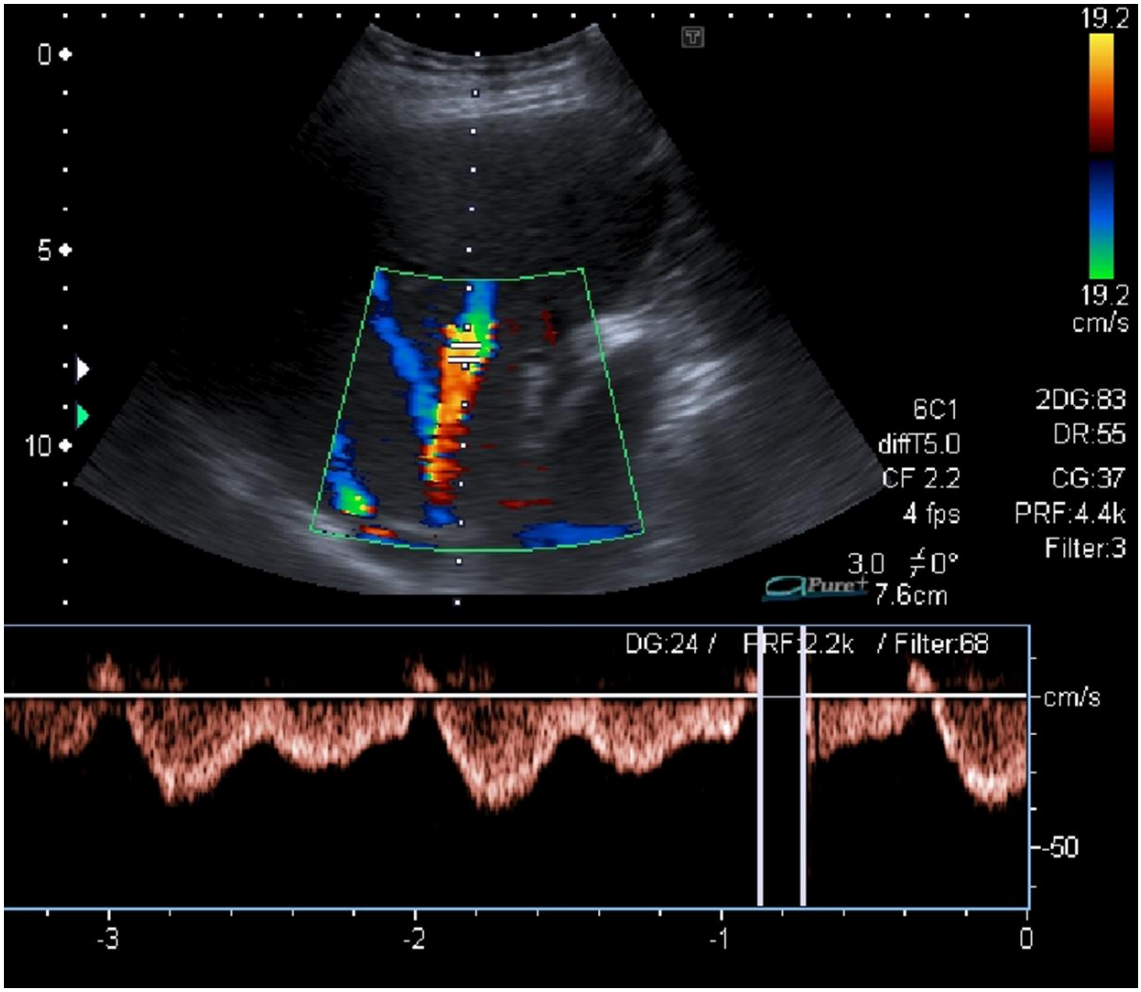

Fig. 4

Annotation:

Hepatic veins: predominantly antegrade flow to the heart, typical spectral Doppler waveform (bidirectional waveform, triphasic). Note that antegrade flow in the hepatic veins is displayed below the baseline.

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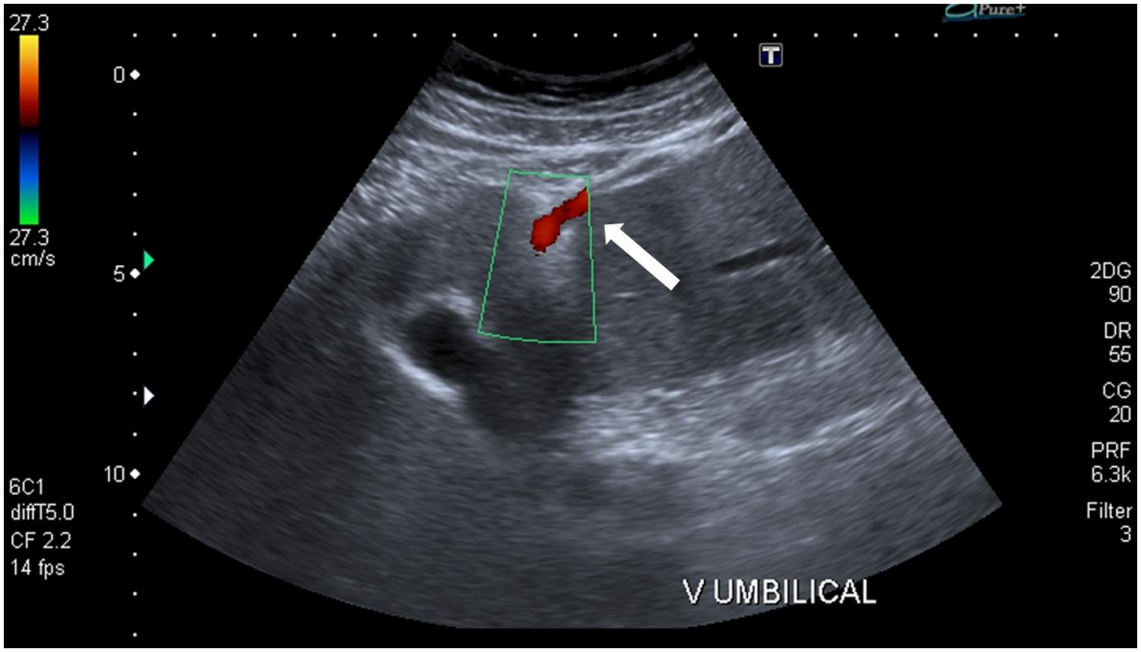

Fig. 5
Annotation:
Recanalized umbilical vein in portal hypertension: longitudinal color Doppler sonogram demonstrates that flow in this vessel (arrow) is hepatopedal (away from the liver), indicating that the umbilical vein is functioning as a portosystemic collateral.

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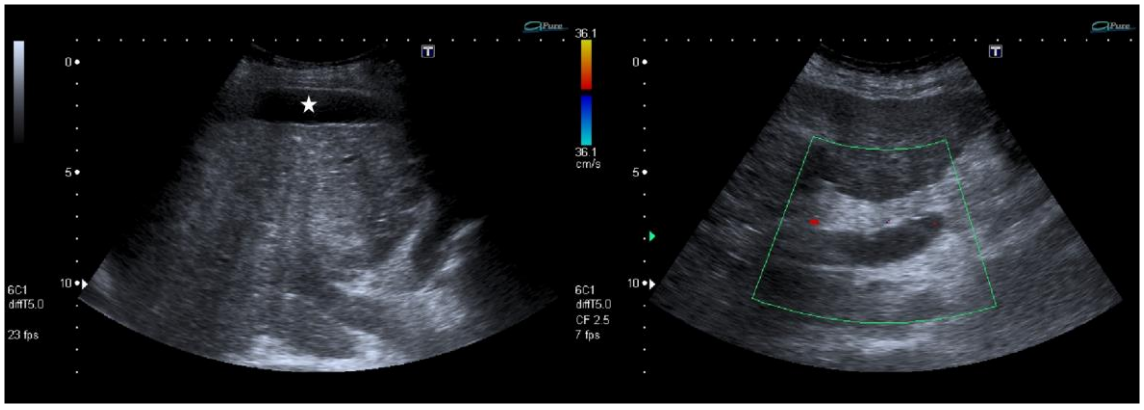

Fig. 6
Annotation:
Portal vein thrombosis: echogenic material within the portal vein lumen (slightly dilated) and absence of portal flow on color Doppler examination. Note that the liver has a cirrhotic appearance and there is ascites (star).

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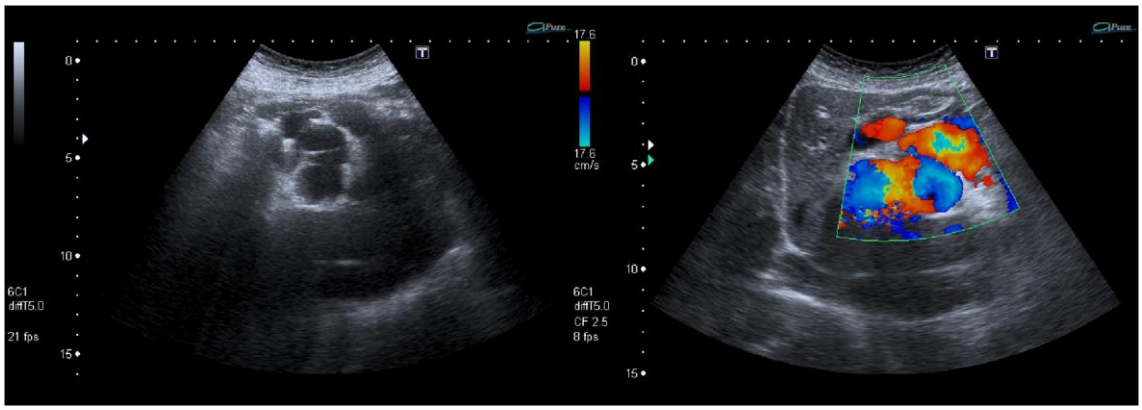

Fig. 7
Annotation:
Cavernous transformation of the portal vein: an irregular tangle of collateral vessels is seen at the porta hepatis.

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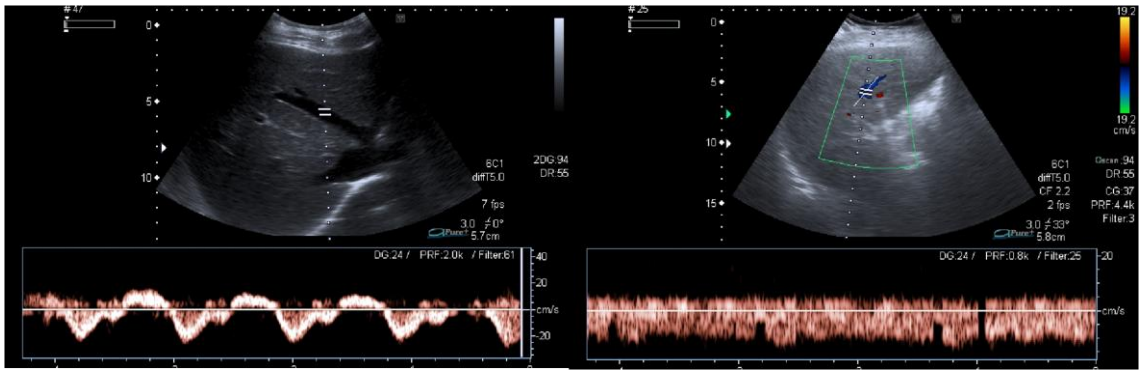

Fig. 8
Annotation:
Left: dilated hepatic veins and inferior vena cava forming a “star” appearance. The hepatic venous waveform is still triphasic (not pulsatile as in CHF). Right: compressed hepatic veins in a patient with cirrhosis, with decreased phasicity and spectral broadening.

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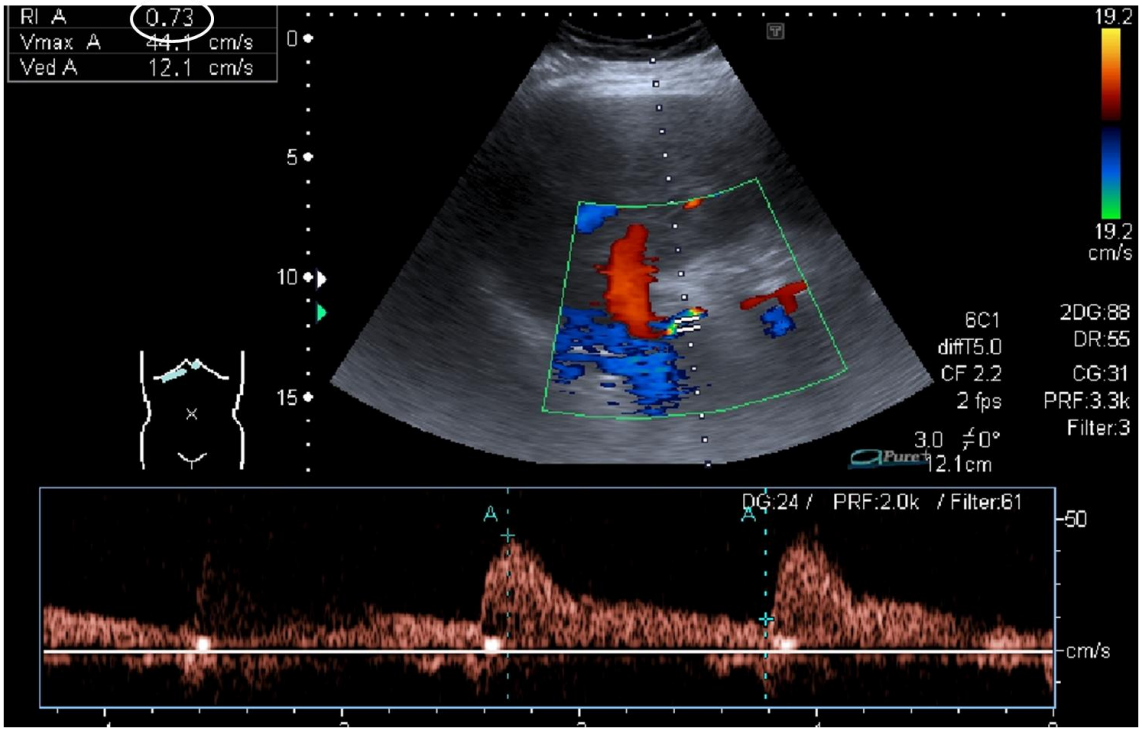

Fig. 9

Annotation:

Normal waveform and RI of hepatic artery in a patient with orthotopic liver transplantation (normal RI ranges from 0.55 to 0.8).

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