Diagn Interv Radiol 2020; 26:541–545

ABDOMINAL IMAGING

PICTORIAL ESSAY

Congestive hepatopathy: the role of the radiologist in the diagnosis

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ABSTRACT

The liver has a complex vascularization and is subjected to a high metabolic demand, making it vulnerable to hemodynamic changes. As a result, several pathologies can develop, one of which is congestive hepatopathy. This disease occurs secondary to various cardiovascular conditions that generate a persistent passive venous congestion in the liver, which in the long term can culminate in fibrosis and cirrhosis, which in turn increases the risk of developing hepatocellular carcinoma. In order to avoid this outcome, early diagnosis is crucial; however, both the clinical presentation and laboratory tests are unspecific, and they are only altered in advanced stages of the disease. One form of early detection is through imaging findings, there being various useful modalities such as Doppler ultrasonography (US), computed tomography, and magnetic resonance imaging. The purpose of this article is to detail the imaging findings of congestive hepatopathy in the different available modalities, with special emphasis on Doppler US, highlighting the role of the radiologist in the suspicion of this disease. We summarize the pathophysiologic mechanisms of congestive hepatopathy, clinical findings, and provide description of its main differential diagnoses.

Congestive hepatopathy refers to liver damage caused by a variety of cardiovascular conditions that trigger passive venous congestion in the liver (1). There are several types of pathologies that can obstruct venous outflow, resulting in congestion of the hepatic parenchyma. The literature refers to congestive hepatopathy as the result of heart disease; however, it can also be the product of anomalies of the inferior vena cava (IVC) or the hepatic veins (2). Any cause of right heart failure can cause an increase in central and hepatic venous pressure. Some common causes include valvulopathies (e.g., mitral stenosis, tricuspid regurgitation), congestive heart failure, right ventricular myocardial infarction, constrictive pericarditis, cardiomyopathy, severe pulmonary hypertension and *cor pulmonale*. It is also common in patients with congenital heart disease who have undergone the Fontan procedure, which redirects the systemic venous return to the pulmonary arteries, without passing through the right ventricle (3).

All these pathologies cause an increased venous pressure in the IVC and hepatic veins, and then to the hepatic sinusoids. Persistent congestion in the perisinusoidal space (of Disse) produces accumulation of exudate and edema, which hinders the diffusion of oxygen and nutrients, with the resulting atrophy of the hepatocytes in zone 3 of the hepatic lobule, around the central veins (Fig. 1) (4). In more advanced stages, perivenous and perisinusoidal fibrosis develops, as well as the development of fibrous walls between the hepatic veins. This fibrosis pattern observed in hepatic congestion is known as "inverse lobulation". The macroscopic examination reveals a liver with reddened areas that represent congestion around the central veins (zone 3) surrounded by healthy or fatty parenchyma (zone 1). This appearance has been called "nutmeg liver" (2).

Clinical presentation and laboratory exams

Patients with congestive hepatopathy generally maintain a preserved liver function and are usually asymptomatic with respect to their hepatic disease in the initial stages (2); nevertheless, sometimes they can experience a slight dull pain in the upper right quadrant. Moreover, they can present with jaundice, which in severe cases can be confused with

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Received 25 December 2019; revision requested 17 January 2020; last revision received 31 January 2020; accepted 10 February 2020.

Published online 9 October 2020.

DOI 10.5152/dir.2020.19673

You may cite this article as: Morales A, Hirsch M, Schneider D, González D. Congestive hepatopathy: the role of the radiologist in the diagnosis. Diagn Interv Radiol 2020; 26:541–545

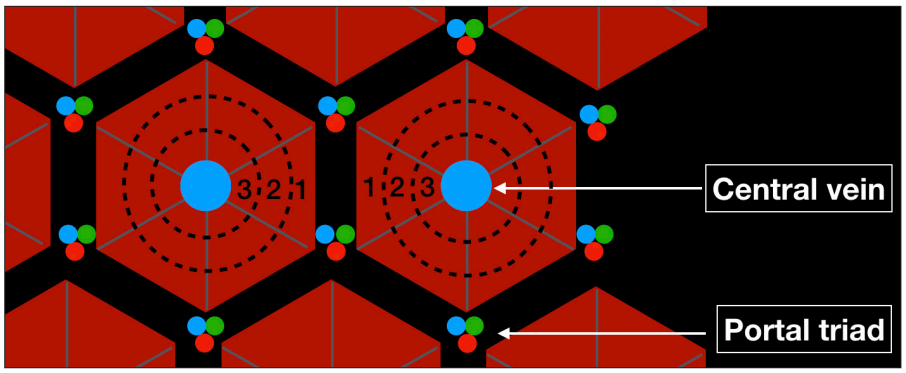

Figure 1. Diagram that shows hepatic microcirculation: Zone 1, area closest to the portal triad (best oxygenated); Zone 2, between zones 1 and 3; Zone 3, perivenular area (worst oxygenated), most susceptible to damage by vascular alterations.

biliary obstruction (5). On physical examination, hepatomegaly is usually the most striking feature and there can be ascites in 25% of the cases, characteristically without splenomegaly (4). Hepatojugular reflux is usually present and it is useful to differentiate hepatic congestion from other hepatic diseases. The clinical presentation is usually dominated by symptoms related to congestive heart failure, and in most cases the patient's prognosis is determined by the presence of the underlying cardiovascular disease, which is why its rapid detection is essential to a favorable evolution, with the radiologist being a fundamental pillar in early diagnosis.

The laboratory examinations are of little use for the diagnosis of this hepatopathy, as they remain in ranges near normal until advanced stages of the disease and are not correlated with the degree of fibrosis (6). There may be hyperbilirubinemia, although total bilirubin rarely exceeds 3 mg/dL. The serum levels of alkaline phosphatase (AP) are usually normal or slightly high in acute heart failure, even in the presence of jaundice, which helps to distinguish jaundice due to venous congestion from that caused by biliary obstruction. However, the AP level can be high in the context of serious chronic heart failure (4). The hepatic enzymes may also reach up to 2-3 times the normal value, there being a correlation between their elevation and hepatocyte necrosis in zone 3 of the hepatic lobule. Hypoalbuminemia may be slight (never below 2.5 g/dL), which is not correlated with liver damage, but rather with malnutrition. Atrial natriuretic peptide levels also tend to be high (2).

Main points

- Patients with congestive hepatopathy are usually asymptomatic at first, with preserved liver function.
- Doppler US is an excellent tool to study the morphology and vascular behavior of the liver.
- The morphology of the spectral wave of the hepatic veins makes it possible to detect some causes of congestive hepatopathy, such as right heart failure or tricuspid insufficiency.
- In both CT and MRI, the presence of heterogeneous enhancement, especially at the periphery of the liver, is a sign of the decelerated flow resulting from parenchymal congestion.

Imaging findings in congestive hepatopathy

Images fulfill a fundamental role in the detection of congestive hepatopathy. As noted previously, the clinical presentation and laboratory examinations only appear altered in advanced stages of the disease. The different imaging modalities available are discussed next, with an emphasis on the distinguishing elements. The common findings for other causes of chronic liver disease are beyond the scope of this article.

Ultrasonography

The liver has a complex vascularization and is subject to a high metabolic demand (7). It can receive up to 25% of cardiac output (75% of the portal vein and 25% of the hepatic artery). Ultrasonography (US) is a very useful modality for the morphological study of the liver and its hemodynamic properties. The Doppler US can evaluate the direction of the blood flow and the speed of hepatic vascularization. The blood vessels of the liver, which include the hepatic artery, hepatic veins and the portal vein, present diameters, spectral curves and characteristic speeds that may be altered under certain stimuli.

According to a recent study (8), the average diameter of the intrahepatic IVC at rest is 19.4±4.0 mm, and 5.6±6.6 mm with the Valsalva maneuver. For the middle hepatic vein, these values are 6.0±1.5 mm and 3.8±2.0 mm at rest and with the Valsalva maneuver, respectively. Another study refers to the normal diameter of the right hepatic vein being 5.6–6.2 mm at rest (9), increasing to 8.8 mm in the presence of heart failure and reaching 13.3 mm in the presence of heart failure with pleural effusion. As previously mentioned, under normal conditions, the IVC and hepatic veins narrow during inspiration or with the Valsalva maneuver. In the presence of right heart failure, the normal variation of the venous diameter with respiratory movements can be reduced or absent (2). In physiological conditions, the hepatic veins present a predominantly anterograde flow (below the baseline) studied with a spectral Doppler US, with a triphasic wave pattern, in which four waves can be identified: "a", "S", "v" and "D", each corresponding to a different phase of the cardiac cycle (Fig. 2).

In pathological conditions, this triphasic pattern can be altered. In both tricuspid insufficiency and right heart failure, there is an increase in pulsatility that is reflected as a relative increase in the anterograde and retrograde speeds. In tricuspid insufficiency high "a" and "v" waves are observed, with a reduced "S" wave, being smaller than the "D" wave (Fig. 3), and in severe cases this can become retrograde, forming an a-S-v complex (Fig. 4). In right heart failure, high "a" and "v" waves are also observed, but an adequate ratio between S and D waves is maintained (10) (Fig. 5).

Regardless of the pathological condition, the "a" wave is almost always the highest above the baseline, which makes it possible to identify it and thus know what the remaining waves correspond to.

The portal vein always has an anterograde (hepatopetal) flow and variability resulting from cardiac activity (smoothly undulating wave), with systolic speeds between 16–40 cm/s and pulsatility indices (PI) > 0.5 (lower PI = greater pulsatility). Both tricuspid insufficiency and right heart failure generate an increase in pulsatility (PI < 0.5) in the spectral curves (Fig. 6).

The hepatic artery is characterized as having an anterograde flow, pulsating with low-resistance spectral waves (RI, 0.55–0.7) and maximum systolic speeds of up to 30–60 cm/s. In cases of hepatic venous congestion, there is an increase in arterial resis-

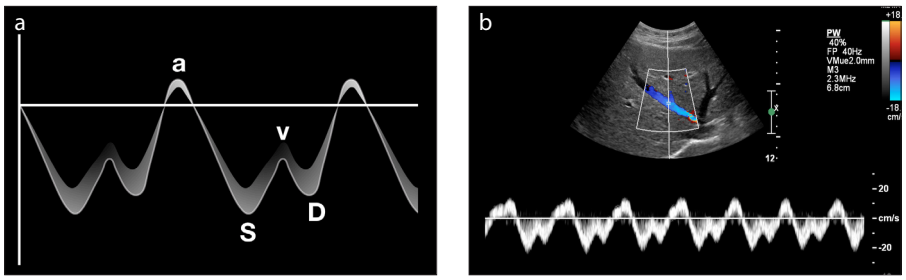

Figure 2. a, b. Diagram **(a)** and spectral Doppler US **(b)** show the normal triphasic pattern of the hepatic veins. The "a" wave is correlated with the atrial contraction, at the end of the diastole, and is where the maximum retrograde flow occurs. The "S" wave corresponds to the anterograde flow during ventricular systole, and is the lowest point (or fastest) in the cycle. The ascending peak of the "v" wave is correlated with the opening of the tricuspid valve, which marks the transition between systole and diastole. This peak can be above the baseline (retrograde) or below it (anterograde) in normal conditions, but below the "a" wave. The "D" wave is anterograde and is correlated with ventricular filling in the early diastole.

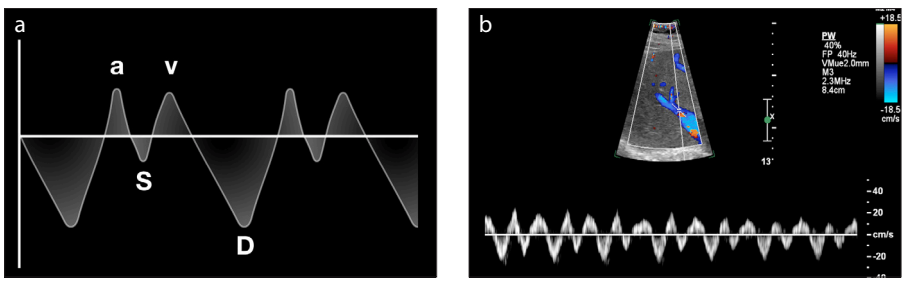

Figure 3. a, b. Slight to moderate tricuspid insufficiency. Diagram **(a)** and spectral Doppler US **(b)** of the hepatic veins in case of slight to moderate tricuspid insufficiency, where greater pulsatility and a reduction in amplitude of the "S" wave approaching the baseline is observed. The morphology of the wave can be confused with the pattern of right heart failure; however, the "S" wave is less deep than the "D" wave, which makes it possible to distinguish it.

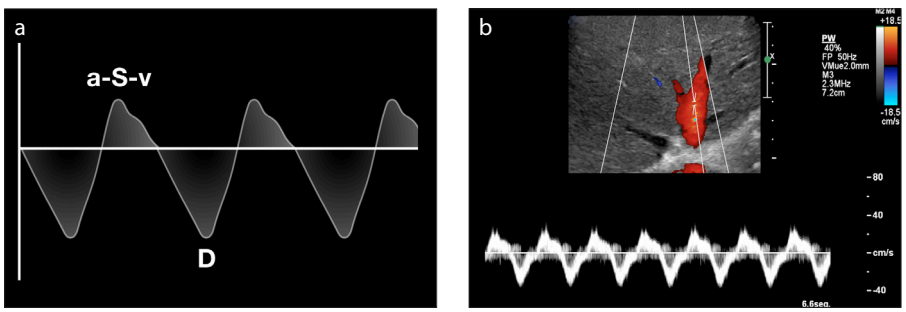

Figure 4. a, b. Severe tricuspid insufficiency. Diagram **(a)** and color spectral Doppler US **(b)** of the middle hepatic vein in a patient with severe tricuspid insufficiency. The dilated hepatic vein is observed with reverse flow on the color Doppler and a spectral wave that presents an increase in pulsatility and a retrograde "S" wave, which forms an a-S-v complex.

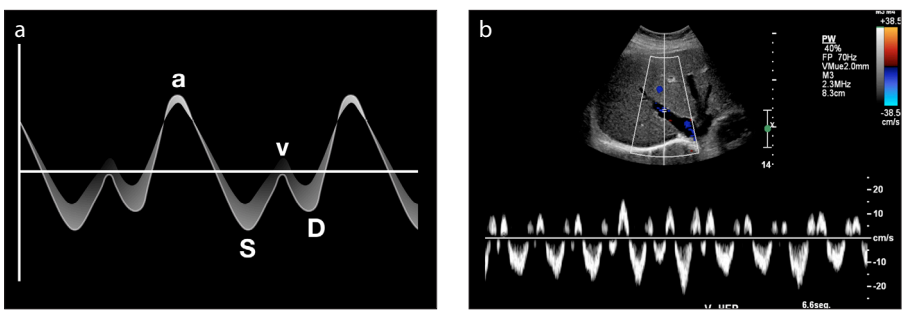

Figure 5. a, b. Right heart failure. Diagram **(a)** and color spectral Doppler US **(b)** of the right hepatic vein in a patient with right heart failure. An increased diameter of the right hepatic vein is recognized, which presents flow with increased pulsatility on the spectral Doppler. The "S" wave maintains a greater depth than the D wave, which helps to differentiate it from tricuspid insufficiency.

tance (RI > 0.7), in both acute cases, as a result of diffuse vasoconstriction, and chronic cases, as a result of parenchymal fibrosis.

Although there are changes in the arterial and portal flow, as previously described, these variations are common to every disease that ultimately determines cirrhosis (6), with these variations being unspecific for congestive hepatopathy in isolation.

Computed tomography and magnetic resonance imaging

With both computed tomography (CT) and magnetic resonance imaging (MRI), a range of morphological abnormalities can be observed that depend on the stage of the disease. In early stages, venous congestion can manifest with hepatomegaly; however, in patients in more advanced stages, signs of cirrhosis are noted such as an atrophic and nodular liver. As seen on the US, the IVC and hepatic veins are dilated, and in severe cases, venous shunts can be observed between the hepatic veins. In addition, the vascular behavior, shown by contrast enhancement patterns, is also altered. In the arterial phase, there is early enhancement of the IVC and the hepatic veins as a result of the reflux of the contrast from the atrium into the IVC (Fig. 7). In the parenchymal phase a speckled enhancement pattern, heterogeneous, with linear and curvilinear areas of lower enhancement, is observed due to delayed opacification of the small and medium hepatic veins (11). The abnormal enhancement pattern is typically observed at the periphery of the liver and is best evaluated in the portovenous phase (Fig. 8). In later phases, contrast retention by the liver causes an equilibrium state to be reached, observing a more homogeneous enhancement. Additionally, fine bands of low attenuation surrounding the central portal veins can be identified, which represent periportal lymphedema and which can enhance in later phases, possibly being confused with portal thrombosis.

The edema and hepatic fibrosis that predominates adjacent to the hepatic veins are observed as low-density reticular areas on the unenhanced CT. On MRI, these regions of edema/perivenous fibrosis are observed with a high signal intensity in T2-weighted sequences and with a low signal intensity in T1-weighted sequences (Fig. 9). These findings become more conspicuous in the portovenous phase. If hepato-specific contrast medium is available, these anomalies in the hepatobiliary phase can be seen as peripheral

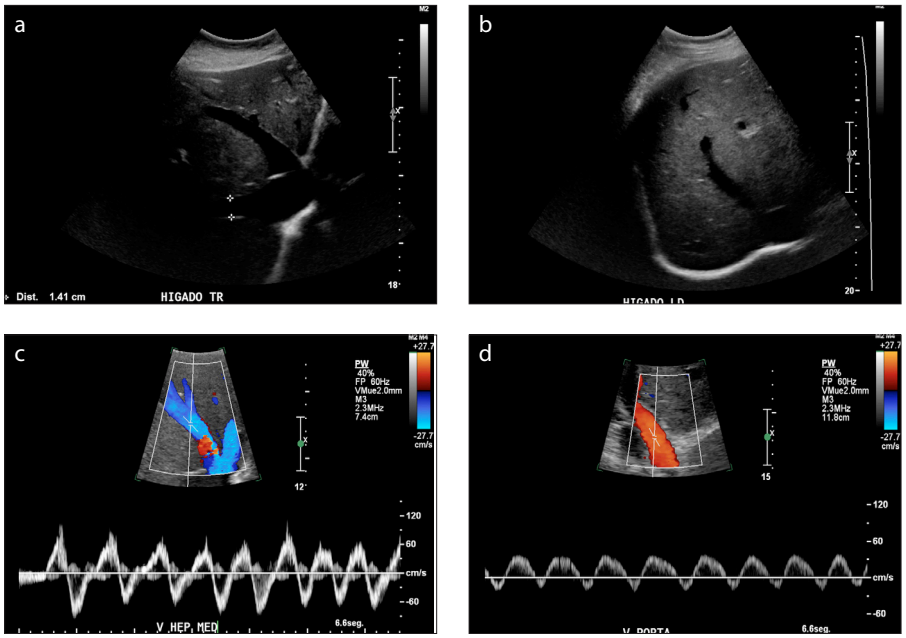

Figure 6. a–d. Findings on the US in B mode, color spectral Doppler in a patient with severe tricuspid insufficiency. US images **(a, b)** show the hepatic parenchyma of heterogeneous echostructure, with dilation of the right hepatic vein. Inversion of the S wave is observed on the spectral Doppler **(c)**, identifying the a-S-v complex. The portal vein also shows an increased pulsatility on the spectral Doppler **(d)**, presenting reverse flow in cyclical form, which is always considered pathological.

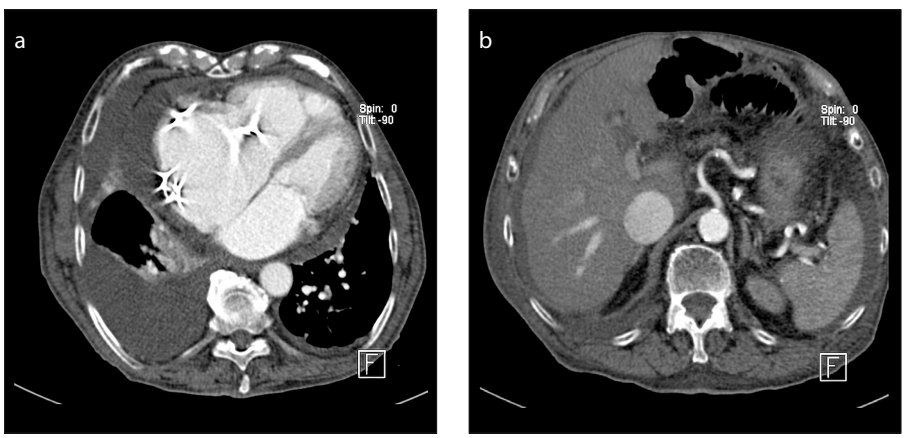

Figure 7. a, b. Arterial phase axial CT images **(a, b)** in a patient with congestive hepatopathy. Cardiomegaly can be observed with hypertrophy of the right chambers, bilateral pleural effusion with right predominance and ascites. There is enhancement of the IVC and hepatic veins with greater density than the portal vein, which results in reflux of the endovenous contrast from the right atrium.

reticular regions with faint enhancement (2).

In addition to diffuse parenchymal involvement, there is a greater prevalence of hypervascular nodules that behave similarly to a focal nodular hyperplasia (FNH), with homogeneous arterial enhancement and subsequent isodensity in later phases. It is important to remember that the presence of cirrhosis increases the risk of hepatocellular carcinoma in patients with congestive hepatopathy. Therefore, every hypervascular focal lesion with subsequent wash out or which increases in size must be studied.

Other findings can be observed extrahepatically that support the diagnosis of congestive hepatopathy, such as cardiomegaly, hypertrophy of the right atrium and ventricle (Figs. 6 and 7), thickening and calcification of the pericardium, pericardial effusion (Fig. 8), pleural effusion, ascites and splenomegaly. The combination of the previously described hepatic findings and cardiac anomalies is highly suggestive of congestive hepatopathy. When signs suggestive of portal hypertension are recognized, the absence of portosystemic shunts supports the congestive hepatopathy diagnosis, since in these cases there is usually no portosystemic venous pressure gradient that facilitates its development.

Differential diagnoses

There are several conditions that can simulate this pathology. Budd-Chiari syndrome is similar to congestive hepatopathy, in which there is obstruction of the hepatic venous outflow, which also results in a poor peripheral parenchymal enhancement. However, the presence of atrophic small hepatic veins (11) and thrombosis of the hepatic veins are characteristics that make differentiation possible. In addition, hypertrophy of the caudate lobe is another finding that enables differentiation. Portal vein thrombosis/embolism also produces alterations in parenchymal enhancement, being able to identify wedge-shaped peripheral perfusion defects, usually with a vein filling defect involved (1). Caroli's disease presents heterogeneous enhancement around the dilated portions of the biliary tree, findings best assessed with MRI. Hereditary hemorrhagic telangiectasia (HHT) shows heterogeneous parenchymal enhancement due to the development of vascular malformations that replace the parenchyma, especially in advanced cases, observing early enhancement in the abnormal regions, as a result of the high vascular flow.

Generally these differential diagnoses lack the particularities of congestive hepatopathy; the dilation of the IVC and hepatic veins as well as the alterations to the spectral Doppler are differential findings that must be assessed in detail in the case of a liver with signs of chronic liver damage, since the correct diagnosis directly affects patient management and prognosis.

Conclusion

Congestive hepatopathy is a clinically important entity that can go unnoticed. Both the clinical presentation and laboratory examinations are of little use in its detection; hence, the radiologist and the images play a fundamental role in early diagnosis. There are characteristic findings that can orient the diagnosis at Doppler US, CT, and MRI. As a recommendation, whenever we face a liver with diffuse involvement, we must evaluate in detail the IVC and hepatic veins to look for signs of reflux or to perform a spectral Doppler study, which will give us fundamental information to propose a diagnosis. An adequate understanding of the physiopathology of this disease is fundamental for the correct interpretation of the

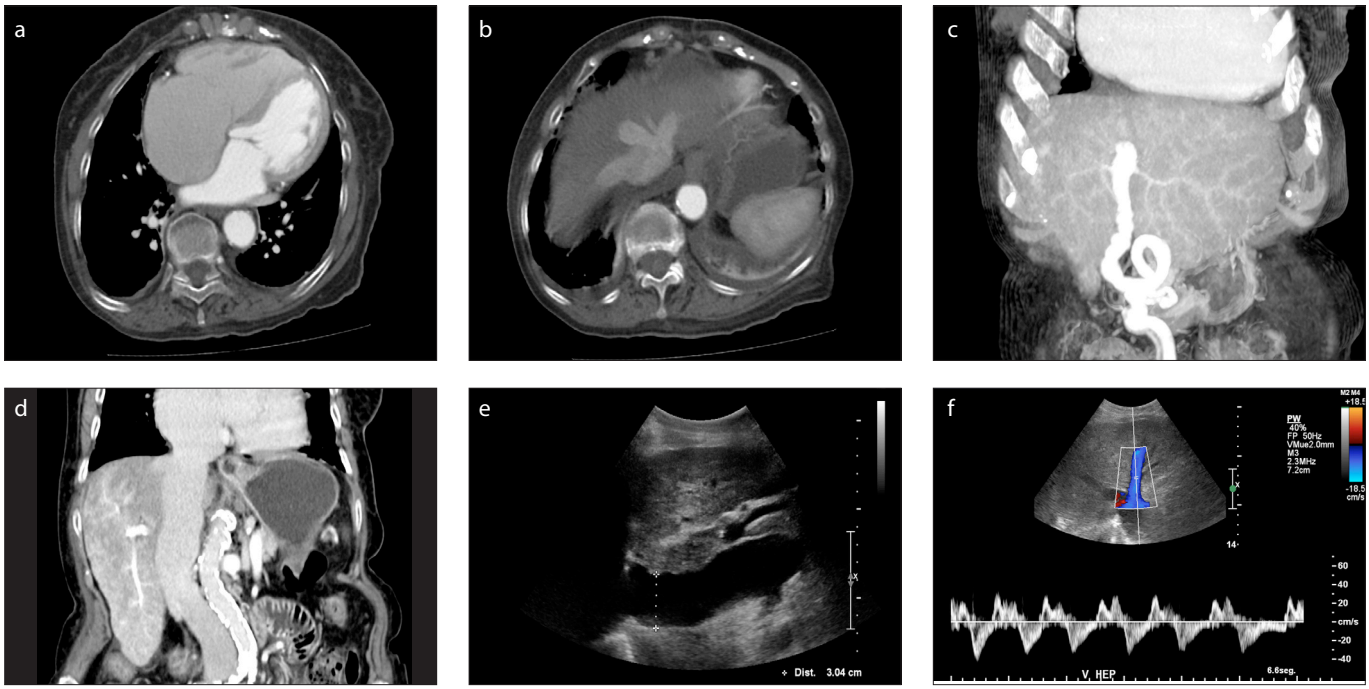

Figure 8. a–f. Congestive hepatopathy. Arterial phase axial CT **(a, b)** show cardiomegaly observed with hypertrophy of right atrium and ventricle associated with reflux of the contrast medium toward the IVC and hepatic veins. Portovenous phase coronal reconstructions **(c, d)** show a noticeable dilation of the IVC and speckled heterogeneous enhancement of the hepatic parenchyma. US images **(e, f)** show dilation of the IVC with a diameter of up to 3 cm. On the spectral Doppler of the left hepatic vein, inversion of the S wave is identified, suggesting an a-S-v complex in cyclical form.

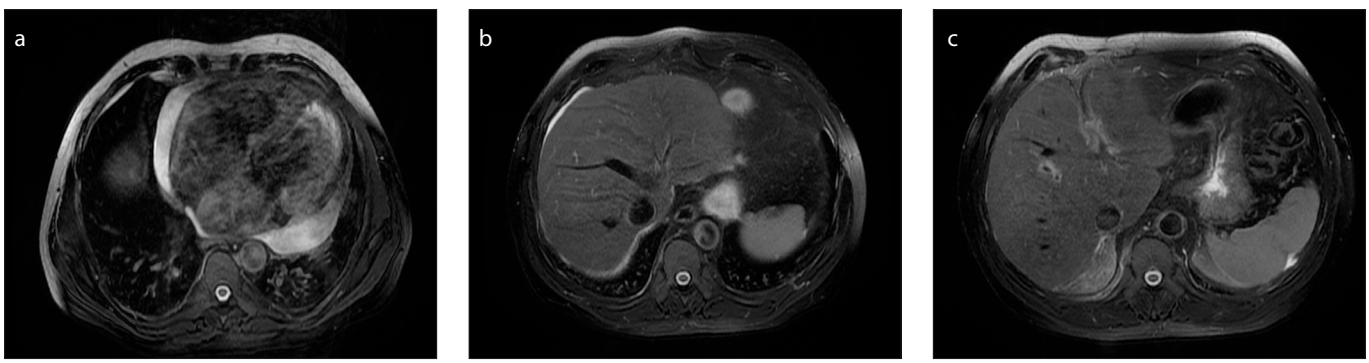

Figure 9. a–c. MRI axial fat saturated T2-weighted images **(a–c)** show signs of chronic liver disease and dilation of hepatic veins, with reticular areas showing high signal intensity around them, that represent perivenous edema/fibrosis in the context of venous congestion. Perihepatic ascites and pericardial effusion are identified extrahepatically.

images. Although there are other pathologies that also produce alterations in hepatic perfusion, the correlation of the imaging findings with the clinical history and an understanding of the physiopathology should enable a correct diagnosis to be reached.

Conflict of interest disclosure

The authors declared no conflicts of interest.

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