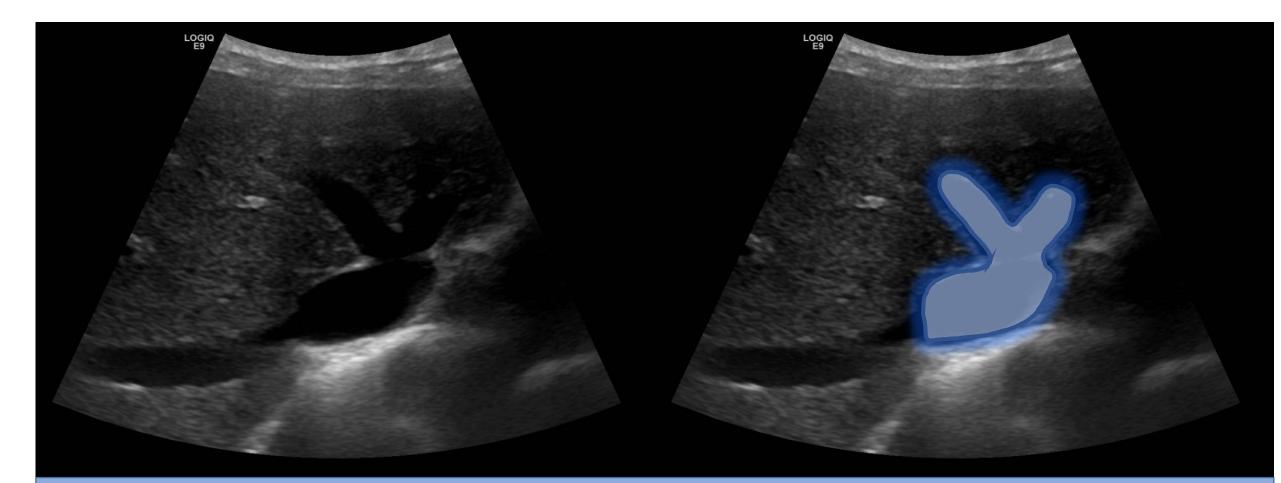
# Passive Hepatic Congestion – A forgotten cause of abnormal liver function tests? Jamie Wild



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#### Introduction

Ultrasound practitioners are often faced with requests where the clinician is trying to determine the cause of abnormal liver function tests (LFTs). The most common pathology encountered in practice which can result in abnormal LFTs is that of fatty liver disease relating to either alcohol use or non-alcoholic fatty liver disease (NAFLD) (Malakouti et al, 2017). Given the prevalence of fatty liver disease in the population and ultrasound's ability to diagnose fatty infiltration relatively easily, it is vital that ultrasound practitioners do not become complacent, and that other pathological processes are considered, especially when no clear cause for the abnormal LFTs is initially identified during the ultrasound examination. While there are a host of other causes of abnormal LFTs, passive hepatic congestion (PHC) is one cause of abnormal LFTs which can be overlooked in the pursuit of the more obvious and common fatty liver. PHC may be asymptomatic for a prolonged period of time with the only clue to suspect its presence being abnormalities in the LFTs (Fortea et al, 2020). If not recognised, chronic congestion leads to hepatic injury eventually resulting in fibrosis and cirrhosis (Hilscher and Sanchez, 2016). As with any pathology, proper understanding and recognition of the clinical presentation and imaging features are vital if a confident and accurate diagnosis is to be achieved.



### PHC Aetiology

Passive hepatic congestion (PHC) is due to stasis of blood within the liver parenchyma, secondary to decreased venous drainage. Various cardiopulmonary diseases including congestive heart failure, constrictive pericarditis, cardiomyopathy and tricuspid regurgitation may lead to an increase in central venous pressure which is then transmitted to the liver by the inferior vena cava (IVC) and

Fig 1. "Bunny sign" Ultrasound demonstrating the dilatated IVC (head), mid hepatic vein (right ear) and left hepatic vein (left ear).

### Ultrasound Features

- Dilated IVC (see Fig 1)
- Dilated hepatic veins (see Fig 1) >8mm
- Hepatomegaly
- Ascites
- Thick walled gallbladder
- Stasis of blood within hepatic veins

#### LFT values

- ALT Elevated
- ALP Elevated
- GGT Elevated
- Bilirubin Elevated
- Albumin -Decreased

hepatic veins, resulting in hepatic congestion (Ramanathan and Sheikh, 2019). If not recognised, congestion may lead to hepatic injury through three main mechanisms (1) decreased hepatic blood flow, (2) decreased arterial oxygen saturation and (3) increased hepatic venous pressures (Hilscher and Sanchez, 2016). Passive hepatic congestion can cause elavations of alanine transaminase (ALT) alkaline phosphastase (ALP), glutamyltransferase (GGT) and direct and indirect serum bilirubin, as well as a decrease in albumin (Mauriello, 2022).

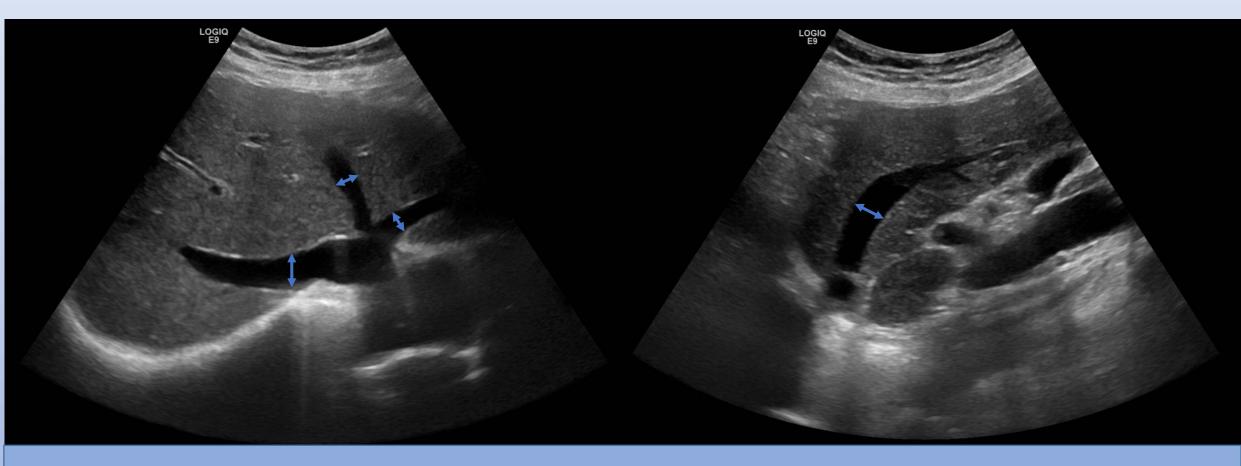


Fig 2. Ultrasound images demonstrating dilated hepatic veins. Diameter of >8mm is consistent with congestion.

#### Discussion

Ultrasound requests relating to abnormal LFTs are encountered on a daily basis within the clinical setting. While ultrasound practitioners are generally excellent at identifying a range of liver pathologies including fatty infiltration, fibrotic change, malignancy and biliary tree abnormalities, an understanding of less common and perhaps more subtle pathologies is important. There are approximately 200,000 new diagnoses of heart failure every year in the United Kingdom (UK) (Conrad et al, 2018). PHC is a common complication of heart failure and may be **a forgotten cause of abnormal LFTs** in a sonographer's practice. The recognition and understanding of PHC aetiology and ultrasound features could have an important impact on a patient's follow up. The most commonly encountered ultrasound features (and often overlooked in the author's opinion) relate to dilatation of the inferior vena cava (IVC) and hepatic veins. The IVC is compliant and reacts to both inspiration and expiration. It is considered abnormal if dilated above 21mm in a patient with cardiac or renal disease, however young healthy patients may also show a diameter of greater than 20mm (Gadi et al, 2018). If dilatation of the IVC and hepatic veins is present (>8mm), a robust diagnosis of PHC can be made, particularly in older patients. Other features to be aware of, although not as common, are hepatomegaly, ascites, thick walled/oedematous gallbladder wall and visualisation of stasis within the hepatic vessels. As mentioned, patients often present with no symptoms other than abnormal LFTs, although a vague right upper quadrant pain may be present secondary to hepatomegaly, relating to the stretching of the liver capsule. If PHC can be identified, interventions can be made and if the underlying cardiac cause can be treated, the secondary PHC should resolve. If left untreated, PHC can lead to inflammation followed by fibrosis and cirrhosis (passive hepatopathy). This further highlights the importance of alerting clinicians to the features of PHC, not only to demonstrate the cause of the abnormal LFTs but also as interventions may not only be needed in relation to the cardiac output. In cases of chronic passive hepatopathy, planning for future hepatology intervention, such as treatment options and surveillance for hepatocellular carcinoma (HCC) may also be required.





Fig 3. Chest x-ray showing cardiomegaly in keeping with heart failure (although not always present).

Check the patient's clinical records for a history of heart failure. While not always found, previous imaging may have been performed which highlights the potential for PHC and if mentioned can add weight to your report.

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