Pulsatile Portal Vein Sign- An Indication Of Underlying Tricuspid Regurgitation With Congestive Failure
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Citation

Abstract
Pulsatile portal vein sign is an uncommon finding which usually results when the elevated systemic venous pressure is transmitted through the hepatic sinusoids to the portal vein. It is most commonly seen with tricuspid regurgitation and is suggestive of underlying congestive heart failure.

CASE REPORT
A 34 year old female presented to the medical out patient clinic with complaints of jaundice for 2 weeks duration along with mild distension of abdomen. Past history was significant for a rheumatic valvular heart disease for past 16 years. There was associated history of dyspnoea on exertion and orthopnoea. On examination, the patient had mild hepatomegaly with ascites. The tip of spleen was palpable just below the costal margin. The patient also had a loud pan systolic murmur best heard over the tricuspid area.

CBC was unremarkable. The hepatic enzymes and bilirubin were mildly elevated. Viral serologies were negative. Renal functions were normal. The patient was referred for an ultrasound scan to ascertain the cause of deranged hepatic function.

Ultrasound scan through the liver revealed coarsened echo texture of liver with surface nodularity suggestive of cirrhosis (Figure-1). Spleen was mildly enlarged. Small amount of ascites was also seen.

Doppler imaging of the portal vein revealed presence of pulsatile flow in the portal vein, which dropped below the baseline with each systole (Figure-2). Associated marked enlargement of IVC and hepatic veins was seen (Figure-3) with dilatation of cardiac chambers (right more than left). (Figure-4)

Subsequent cardiac echo confirmed presence of multiple valvular diseases with predominant TR.

Figure 1
Figure 1: Ultrasound scan through liver showing coarsened echo texture with surface nodularity. There is associated ascites.
Figure 2
Figure 2: Spectral Doppler trace of the portal vein showing pulsatile waveform

Figure 3
Figure 3: Scan at the level of hepatic veins showing marked enlargement of the same with grossly dilated IVC.

DISCUSSION
Normal subjects typically demonstrate minimal variation in portal vein velocity on spectral Doppler analysis during breath holding. The mildly phasic pattern is most likely a reflection of changes in transmitted right atrial pressures, with significant dampening by resistance in venules, sinusoids and small portal vein branches.

Pulsatile portal venous flow is said to be present when the minimal portal vein flow velocity drops to or below the baseline.

Pulsatile portal venous flow is commonly seen with tricuspid regurgitation. In their study of 17 patients, Abu Yousef et al found tricuspid regurgitation in 15 of the 17 patients that were evaluated.

Most common cause of tricuspid regurgitation is dilatation of right ventricle due to left ventricular failure, mitral stenosis, portal hypertension, pulmonary stenosis or atrial septal defect.

In patients with severe tricuspid regurgitation (grade 3 or more), the valvular leak increases distal vascular impedence of portal circulation, which is maximum towards late ventricular systole. This in turn may impede antegrade blood flow in the portal vein, resulting in either return to base line or even flow reversal during ventricular systole. The later flow pattern occurs in more severe cases of hepatic sinusoidal congestion. These portal flow patterns may be characteristic of TR and are suggestive of associated congestive heart failure.
Other causes that may result in a pulsatile portal venous flow include aortic-right atrial fistula or a fistula between portal and hepatic veins.

Since both these causes are rare, the findings of a pulsatile portal venous flow should lead one to consider the possibility of TR with associated CHF.

Other ancillary findings in such cases include dilated IVC (diameter more than 2.5 cm) and hepatic veins with abnormal spectral waveform.

Chronic sinusoidal congestion may induce cirrhotic changes in liver, as in this case with associated secondary manifestations of cirrhosis resulting in coarsened live echo texture with surface nodularity, spleenomegaly and ascites.

Gray scale imaging usually also reveals markedly dilated RV and RA secondary to TR.

**LEARNING POINTS**

- The radiologist should be aware to the possibility of hepatic dysfunction secondary to underlying cardiac pathology.
- In a patient with pulsatile portal venous flow, the most likely cause is underlying TR with associated CHF.

**References**

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