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Ultrasound examination of hemodynamic changes in patients with hepatitis

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Abstract

This research was aimed to evaluate portalsystemic hemodynamic changes in chronic severe hepatitis. Hemodynamic parameters included portal vein diameter, portal vein peak velocity, portal vein volume, spleen length, spleen vein diameter, spleen vein volume and umbilical vein recanalization. They were measured by color doppler ultrasonography in 30 patients with chronic severe hepatitis, compared with 40 normal controls, 25 patients with chronic hepatitis B, 30 patients with compensable cirrhosis, and 25 patients with decomposable cirrhosis.

Keywords: *Ultrasound, hepatolienal syndrome, hepatic vein, doppler, cirrhotic process.*

Introduction

Ultrasound examination (ultrasound) is widely used in the diagnosis of liver diseases due to its availability, non-invasiveness, and the absence of radiation exposure. However, the question of the diagnostic value of this method in chronic hepatitis remains open. Ultrasound for suspected chronic hepatitis is performed to determine the size, assess the state of the liver parenchyma and the presence of signs of portal hypertension, it is additionally recommended to examine the spleen, determine the caliber of the portal vein and the speed of portal blood flow. There are a number of reports in the literature, sometimes contradictory, about the echographic manifestations of chronic hepatitis. It is reported that in chronic hepatitis, echograms can visualize both a structurally homogeneous liver, and with inclusions of single structures, more often in the projection of the left lobe of the liver. Chronic hepatitis is characterized by the presence of hepatolienal syndrome, uneven tissue density, areas of increased echogenicity, increased echogenicity of the liver and spleen, graininess of their structure, sometimes there may be small-focal heterogeneity with signs of distal ultrasound fading, detection of enlarged lymph nodes in the gates of the liver, changes in the vessels of the liver and spleen. The proper vessels of the liver are represented by the proper hepatic artery, which is a branch of the common hepatic artery, the portal vein, and the hepatic veins. The common hepatic artery arises from the celiac trunk and goes to the hilum of the liver. In its course, it gives off the right gastric artery and divides into two branches, the proper artery of the liver and the gastroduodenal artery. The proper hepatic artery passes between the sheets of the hepatoduodenal ligament in front of the portal vein and to the left of the bile duct. The hepatic vein (HV) is the only draining vessel in the liver, which has two supplying vessels, from the liver sinus to the inferior vena cava (IVC). The thin-walled veins are anechoic under ultrasonography, do not have valves, and can be distinguished from the portal vein. The spectrum of HVs can reflect changes in blood flow through the tricuspid valve during the cardiac cycle, leading to pulsatile changes in the spectrum during ultrasonography. The normal waveform of HVs is triphasic with two hepatofugal phases related to atrial and ventricular diastole, and a short phase of retrograde (hepatopetal) flow caused by the pressure increase in the right atrium at atrial systole. With increased stiffness in the liver parenchyma (especially around the HVs), the hepatic waveform becomes less pulsatile with no retrograde flow, and can eventually lead to a flat waveform. The ability to detect a hepatic parenchymal abnormality by hepatic venous Doppler studies has been discussed by several authors: their opinions vary considerably. Hence, a semi-quantitative parameter DI was introduced and data showed that evaluation of the HV DI might be a useful supplemental method for assessing the therapeutic response to portal hypertensive drugs when portal pressure examination is not available.The exact cause of abnormalities in HVs is controversial. One study suggested that terlipressin-induced improvement in the waveforms is evidence that a hemodynamic effect of high portal pressure rather than a fixed structural abnormality is the pathogenic mechanism responsible. In the study, we evaluated the relationship between changes in intrahepatic blood flow and portal pressure (PP) measured intraoperatively in patients with portal

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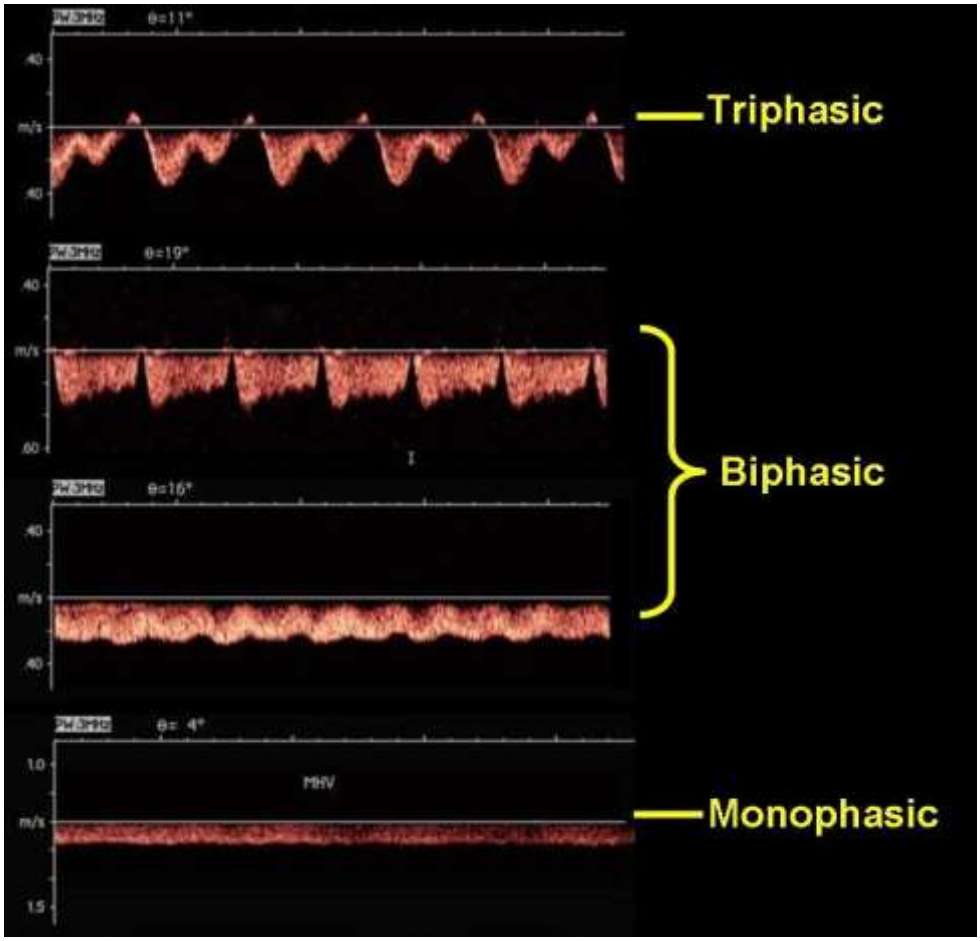
hypertension (PHT), aiming to discuss the indicative value of HV waveform and its quantitative index DI firstly, then the cause of changes in HV waveforms both from the histological and hemodynamic angles.

Material & Methods

Sixty patients who had been diagnosed with PHT and accepted surgical therapy of portosystemic shunts were investigated. PP was measured intraoperatively. Thirty healthy volunteers with no history of chronic liver disease were enrolled as the control group. HV waveforms were categorized as triphasic, biphasic or monophasic. DI was compared as the quantitative indicator of abnormal HV waveforms. Another two Doppler parameters, PVVel and HAPI were also measured. These Doppler features were compared with PP, Child-Pugh scores and histological changes assessed by liver biopsy.



HV waveform patterns in patients with PHT. Triphasic pattern (two antegrade waves below the baseline and one presystolic retrograde wave above the baseline). Biphasic pattern (with absent reversed pre-systolic wave). Monophasic pattern (with a flat pattern).

Results

In the patient group, the Doppler flow waveforms in the middle HV were triphasic in 31.6%, biphasic in 46.7%, and monophasic in 21.6% of subjects. These figures were 86.7%, 10.0%, and 3.3%, respectively, in healthy subjects. With the flattening of HV waveforms, the HAPI increased significantly (r = 00.438, p < 0.0001), whereas PVVel decreased significantly (r = -0.44, p < 0.0001). Blood flow parameters, HAPI, PVVel and HV-waveform changes showed no significant correlations with Child-Pugh scores. The latter showed a significant correlation with PP (r = 0.589, p = 0.044). Changes of HV waveform and DI significantly correlated with PP (r = 0.579, r = 0.473, p < 0.0001), and significant correlation between DI and Child-Pugh scores was observed (r = 0.411, p = 0.001). PP was significantly different with respect to nodule size (p < 0.05), but HV-waveform changes were not significantly correlated with pathological changes.

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Discussion

Various studies have been designed for evaluating the severity of liver abnormalities by Doppler ultrasound in patients with chronic liver disease. The pattern of blood flow in the HV is one of the parameters to be evaluated. Three grades of hepatic waveforms have been described by Bolondi et to indicate changes from the normal triphasic pattern to the flat pattern widely used in older studies of chronic liver disease. Nowadays, more studies are carried out to determine if analyses of HV waveforms may be useful in the assessment of PHT. In the present study, we suggest that an abnormal HV Doppler curve and quantitative index DI may be non-specific indicators of liver abnormality as well as of PP in PHT patients. Furthermore, by comparing the hemodynamic changes in the portal vein and hepatic artery and histological changes in liver parenchyma, we attempted to discuss the mechanism of abnormal HV waveform.

Even though measurement of the hepatic vein pressure gradient (HVPG) has been accepted as the 'gold standard' for assessing the degree of PHT, it is not suitable for widespread routine use because of its invasiveness. Additionally, in the presence of increased pre-sinusoidal resistance during the cirrhotic process, the portal venous pressure can be higher than the wedged hepatic venous pressure. There still was a lot of studies indicated that a good correlation between the HVPG and portal venous pressure either with a wedge catheter or a balloon catheter measured, especially in patients with alcoholic cirrhosis and hepatitis B virus (HBV) cirrhosis. Considering that limitations of HVPG, and the chance we can get the directly measured portal pressure, the present study compared HV waveforms with PP measured directly in the portal venous system for the first time.

Conclusion

In patients with PHT, a monophasic HV waveform indicates higher portal pressure. Furthermore, quantitative indicator DI can reflect both higher portal pressure and more severe liver dysfunction. Flattening of HV waveforms accompanied by an increase in the HAPI and decrease in PVVel support the hypothesis that histological changes reducing HV compliance be the cause of abnormality of Doppler HV waveforms from the hemodynamic angle.

Acknowledgements

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