Portal hypertension is one of the major complications of chronic liver disease and cirrhosis. Noninvasive duplex Doppler ultrasound can be repeated in the follow-up of hemodynamics of the portal system. Doppler sonography can identify and localize the course and dynamic of blood flow. Ultrasound contrast agents are gas-filled microbubbles that can improve the flow with poor Doppler signal quality. The main portal vein flow velocity and volume can be measured using duplex sonography. In this article, a variety of assessments for portal hemodynamics, including portal vein pulsatility index, portal vein congestion index, hepatic artery resistance index, hepatic circulatory index, splenic arterial pulsatility index, hepatic perfusion index, intrahepatic circulatory time, hepatic vein waveform and hepatic vein transit time, are discussed. Color flow images of esophageal varices and para-esophageal veins can be generated by the newly developed electronic radial endoscopic Doppler ultrasonography. Pulse-inversion ultrasonography improves the detection of liver metastasis and reveals more lesions of smaller size than conventional ultrasonography.

**KEY WORDS** — cirrhosis, duplex Doppler, portal hypertension, vein flow velocity

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Portal hypertension is one of the major complications of chronic liver disease and cirrhosis. The presence of portal hypertension is related to the formation of esophageal varices, ascites and fluid and electrolyte redistribution, hepatorenal syndrome, and hepatic encephalopathy [1–3].

Development of imaging system has improved clinical diagnosis of cirrhosis. The images help identify structural changes of the liver, portal hypertension and collaterals, ascites, and vascular change. Clinical evaluation of portal hypertension may predict the development of esophageal and gastric varices, prevent variceal hemorrhage, predict survival rate, and assess the efficacy of treatment of portal hypertension [4]. Sonography has been widely used for the follow-up of chronic liver diseases in identifying cirrhosis and hepatocellular carcinoma. The sonographic findings of increasing diameter and irregular shape of portal vein (PV), increasing collaterals and the presence of ascites and splenomegaly may suggest portal hypertension and cirrhosis. The hemodynamic change of the portal system has been assessed by using duplex Doppler ultrasound, hepatic vein (HV) catheterization [5], computed tomography [6], magnetic resonance imaging [7], and portal scintigraphy [8].

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Noninvasive duplex Doppler ultrasound can be utilized repeatedly in assessing the hemodynamics of the portal system [9]. The velocity and direction of blood flow are assessed by measuring the Doppler shift. The direction of blood flow is shown in red and blue color, and the color intensity represents the flow velocity. Color Doppler sonography can identify and localize the course and dynamic of blood flow. In power Doppler sonography, the Doppler signal amplitude is squared to show the small blood vessels with low blood flow velocities. Tissue harmonic imaging can enhance the spatial and contrast resolution of B-mode images. Ultrasound contrast agents are gas-filled microbubbles with diameters in the micrometer range to improve the flow with poor Doppler signal quality [10–12].

Portal hypertension occurs in various etiologies. The color Doppler sonography is useful in the identification and localization of collaterals developed in portal hypertension, including umbilical vein, coronary gastric veins, esophageal collaterals, and collaterals in splenic hilum [13]. Invasive modalities for studying portal circulation are often not appropriate in patients having severe acute hepatitis or cirrhosis with hepatic failure, poor blood coagulation and ascites. Ultrasonic Doppler is safe and noninvasive in clinical evaluation of the portal blood flow, even in the case of patients with liver failure.

Doppler sonography is helpful in the follow-up of surgical intervention such as portacaval shunt, splenorenal shunt, transjugular intrahepatic shunt, hepatectomy, and liver transplantation [14–20]. PV thrombosis is shown by tumor thrombus and undetectable flow inside the PV [21,22]. Contrast-enhanced sonography is superior to Doppler sonography for the detection of portal and HV thrombosis complicating malignancies [23,24]. Doppler sonography can assess intrahepatic hemodynamics and shunt patency before and after transjugular intrahepatic portosystemic shunt [25–28]. Findings of increased hepatic artery (HA) blood flow velocity and decreased portal blood flow velocity suggest liver regeneration after liver transplantation [29]. Doppler sonography may detect HA stenosis or thrombosis in patients who have undergone liver transplantation [30,31]. Hepatic arterial stenosis or thrombosis is observed as a change in the spectral waveform to a tardus-parvus pattern [32].

Variability

The measurement of portal blood flow often has high variability. The interobserver and intraobserver variability should be measured to ensure the data reliability [33,34]. Portal blood flow is better measured by the same technician or physician to avoid interobserver variation. The portal blood flow from the controls are measured twice at 30 minutes apart in the same day (A vs. B) and at the same time on consecutive days (A vs. C). The percent difference of intraobserver variability is calculated by the equation: absolute values of (A−B)/A × 100 or (A−C)/A × 100. Coefficient of variation is calculated by the equation: (standard deviation/mean) × 100. Ultrasonic contrast agent has been reported to be able to reduce interobserver and inter-equipment variability [35].

Portal Vein Pulsatility Index (PVPI)

PVPI is calculated by the equation: (maximum−minimum)/maximum frequency shift [36]. PVPI is significantly lower in patients with chronic liver disease and cirrhosis [37]. Color duplex sonography can be used to differentiate the hepatofugal, stagnant and hepatopetal direction of flow. Stagnant portal flow is presented as no detectable flow, which may occur during a right heart failure [36]. Hepatofugal portal flow can be identified by observation of reversed portal vein flow (PVF) in cirrhotics [38]. The markedly increased PVPI can predict a right heart failure [36,39]. Transient stagnant and hepatofugal portal flow may occur in the case of severe right heart failure, mainly during the ventricular systole [36].
**PVF**

The main PVF velocity and volume can be measured using duplex sonography. Mean portal blood velocity is calculated using the equation: mean portal blood velocity (cm/s) = 0.57 × mean maximum portal blood velocity (cm/s) [40]. The cross-sectional area can be recorded at the main PV site where the portal blood velocity is measured. Estimated PVF is calculated using the equation: portal blood flow volume (mL/min) = cross-sectional area (cm²) × mean portal blood velocity (cm/s) × 60 seconds [40].

The PVF is reduced by 80% after 60 minutes of exercise [41]. In healthy pregnant women, the portal venous blood flow significantly increases after 28 weeks of gestation and the hepatic arterial blood flow remains unchanged [42].

In severe acute hepatitis, PVF decreases in patients having ascites [43]. Patients with severe acute hepatitis may have decreased PVF because of the increased sinusoidal pressure without well-developed portosystemic collaterals. Higher serum-ascites albumin gradient positively correlates with higher PVF in severe acute hepatitis patients with ascites [44]. Lower PVF is also identified in patients with fulminant hepatic failure [45] and hepatic encephalopathy [46,47].

In chronic liver diseases, portosystemic collaterals contribute as an important factor in the hyperdynamic state [48]. Patients with chronic hepatitis and cirrhosis have reduced portal blood flow velocity [49,50]. Patients with chronic hepatitis C that have responded to interferon therapy have significant increases in portal blood flow velocity at the end of interferon treatment [51]. In patients with alcoholic cirrhosis, PV blood velocity and flow are correlated to the severity of portal hypertension and to the severity of liver failure [52].

Cirrhotics with the lowest portal velocity have esophageal and gastric varices with a red color sign [49]. The presence of a non-hepatopetal flow pattern implicates an increased risk of esophageal varices bleeding [53]. Decreased right portal venous peak velocity is a risk factor for postoperative hyperbilirubinemia [54].

Doppler sonography is also helpful in the follow-up of the changes of portal hemodynamics under medical treatment such as somatostatin, terlipressin, nitroglycerin, vardenafil, propranolol and nadolol [55–60]. Large-volume paracentesis does not change the portal hemodynamics 24 hours after paracentesis [61].

**Portal Vein Congestion Index (PVCI)**

PVCI has been used to assess the pathophysiologic hemodynamics of portal venous system in different forms of liver diseases [62–64]. PVCI is calculated by the equation: (area/mean portal blood velocity) × 100 [62]. PVCI has a significant increase in patients with Child’s C cirrhosis [65]. Patients with esophageal variceal bleeding have significantly greater splenic blood flow volume and splenic vein congestion index [66]. A study confirms that portal hypertension is associated with patients with high PVCI [67]. Patients with variceal red signs have significantly higher PVF and PVCI than patients without the red signs, and their perfusion pressure gradients are lower [68]. However, subsequent studies show that portal hemodynamics is unrelated to the degree of endoscopic abnormalities in patients with liver cirrhosis [69].

Parasympathetic hypofunction, sympathetic hypofunction and portal hemodynamics are closely related to gastric motility in cirrhotic patients [70]. The postprandial PVF increments are low in patients with esophageal varices of any degree and class B and C cirrhosis. There is no postprandial decrease in PVCI in patients with severe cirrhosis [71].

**Hepatic Artery Resistance Index (HARI)**

HARI is calculated by the equation: (peak systolic value – end diastolic value)/peak systolic value [72]. In PV thrombosis, reduced HARI is helpful for determining venous abnormality [72]. HARI is significantly higher in patients with cirrhosis and is directly correlated with the hepatic venous pressure gradient [73].
Ethanol-related hepatic arterial vasodilation occurs in alcoholic hepatitis and alcoholic patients without liver damage; HA resistance significantly increases in alcoholic and viral-related cirrhosis [74,75]. HARI may detect an increase in vascular resistance in acute liver transplant rejection [76]. Pre-transplant HARI is not correlated with clinical outcome and hospital stay [77]. Patients with fatty liver have higher HARI due to an increase in intrahepatic resistance, which can be reversed by metformin treatment [78,79].

### Hepatic Circulatory Index (HCl)

HCl (cm²/s²) is calculated by using the formula: right portal venous peak velocity (cm/s) × right hepatic arterial peak systolic velocity (cm/s)/splenic arterial pulsatility index (SAPI); patients with hepatitis or cirrhosis have lower HCl [80].

### SAPI

High SAPI predicts prolonged postoperative ascites [54]. An increase in SAPI can predict acute rejection after a liver transplantation [75]. A decrease in SAPI also occurs in small-for-gestational-age fetuses [81,82]. The pulsatility index in the splanchnic area, including SAPI, rises steeply in old patients, probably as a result to compensate decreased cardiac output [83]. Intrarenal arterial pulsatility index and resistive index are higher in cases of portal hypertension with ascites [84,85]. Renal vasoconstriction causes an increase in renal vascular resistance, which can be detected by Doppler ultrasonography [86].

### Hepatic Perfusion Index (HPI)

HPI is measured by the ratio of hepatic arterial to total liver blood flow (sum of hepatic arterial and portal venous blood flow); a value between 0.3 and 0.8 (normal <0.26) is sensitive enough to detect occult colorectal liver metastases [87,88]. HPI is higher in colorectal liver metastases with reduction of portal inflow and subsequently increased hepatic arterial blood flow [89,90]. Other studies have not confirmed the role of HPI in predicting occult hepatic colorectal metastases [91]. Obese patients have significantly higher HPI values [92]. Fatty liver index is measured by the equation: 1.03 × aspartate aminotransferase (IU/L) + 0.152 × triglyceride (mg/dL) − 49.75 × HPI [92].

### Intrahepatic Circulatory Time

Intrahepatic circulatory time is calculated as the difference between the HV and HA arrival times (HV−HA interval time) or the HV and PV arrival times (HV−PV interval time). Intrahepatic circulatory time has been used as a diagnostic test for compensated cirrhosis [93].

### Hepatic Vein Waveforms (HVWs)

Poorer grade of cirrhosis has higher mean HV velocity; cirrhotics with high HV velocity of ≥20 cm/s is presented clinically with moderate to massive ascites [94]. HVW is useful in the noninvasive evaluation of severity of portal hypertension; biphasic and monophasic HVW are associated with severe portal hypertension [95–97]. Non-triphasic HVW may assess the progression of chronic viral hepatitis and grading of cirrhosis [98]. Monophasic or biphasic HVW occurs in children with acute viral hepatitis, especially when accompanied by change in hepatic echogenicity [99]. Patients with fatty liver may also develop abnormal biphasic or monophasic HVW patterns [100]. Abnormal hepatic venous flow velocities are signs of abnormal right ventricular filling pattern before a cardiac surgery, which are associated with increasing need for vasoactive support after a cardiopulmonary bypass [101]. However, recent studies suggest that monophasic HVW may occur in apparently healthy patients [102]. HVW pattern has a limited clinical role in the assessment of hepatic fibrosis or inflammation in patients with chronic hepatitis C [103].
Hepatic Vein Transit Time (HVTT)

Hepatic cirrhosis is accompanied by arterialization of the liver, intrahepatic shunts, pulmonary arteriovenous shunts and hyperdynamic circulatory state. The hepatic first pass of a bolus of an ultrasound contrast agent injected into a peripheral vein is accelerated in patients with cirrhosis \[104\]. HVTT is measured as the time after Levovist injection at which a sustained signal increase of > 10% of baseline was seen. An early arrival time of a microbubble in a HV of less than 24 seconds is a sensitive indicator of cirrhosis \[105,106\].

Recent progress of sonography improves imaging diagnosis. Color flow images of esophageal varices and para-esophageal veins can be generated by the newly developed electronic radial endoscopic Doppler ultrasonography and contrast agents; pulsatile waves in esophageal varices could also be detected, suggesting that arterial flow is involved in the formation of esophageal varices \[107,108\]. When a liver is scanned in the pulse-inversion mode for at least 2 minutes after a Levovist injection, increased intensity is shown in liver parenchyma; microbubbles of Levovist accumulating in liver parenchyma have been reported occurring in late liver-specific parenchymal phase \[109\]. Pulse-inversion ultrasonography is used to detect the liver parenchymal phase of Levovist. The increased vascular resistance in cirrhosis reduces portal perfusion and Levovist accumulation. Microbubble disruption of the liver parenchyma in the late phase of enhancement with Levovist is considered to be lower in cases with cirrhosis \[109\]. Scanning in pulse-inversion mode after a Levovist injection improves the detection of liver metastasis and reveals much smaller size lesions than conventional ultrasonography and computed tomography \[110\].

References


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