Can Sonography Replace Splenoportovenography in Evaluation of Patients with Portal Hypertension?

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Abstract
Real-time sonography and splenoportovenography were compared in 17 patients with portal hypertension for their relative efficacy and limitations with respect to diameters of portal vessels, visualization of collaterals and demonstration of portal vein occlusion. Sonography was able to diagnose portal vein thrombosis and to differentiate an occluded portal vessel from a patent portal vessel not-visualized due to hepatofugal blood flow in the presence of infrahepatic obstruction. However, sonography had limitations in demonstrating venous structures in the presence of excessive bowel gas or fat, and did not provide the flow patterns and the complete picture of the portal vasculature in a single setting. We conclude that the two procedures are complementary to each other, and if combined in patients with portal hypertension, the portal venous system can be evaluated more thoroughly for surgical treatment.

Key words: Hypertension-portal vein, ultrasonics.

Introduction
Splenoportovenography (SPV) has been considered as a simple, effective and safe invasive procedure in comparison to other imaging techniques for evaluating patients with portal hypertension (PH), and is the procedure of choice at many health institutions including ours. However, this procedure is not free of risk and radiation hazards. Recent studies have emphasized the usefulness of ultrasonography (US) in diagnosing PH and its cause, by demonstrating dilated portal vessels, by visualizing various portosystemic collaterals, and by demonstrating patency of portal vessels or the presence of echogenic thrombotic material in their lumen.

Webb et al compared US and SPV findings in patients with PH, but their basic pretext that portal vessel diameters are maximum during expiration is contradicted by the pathophysiological as well as ultrasonic studies. Furthermore, they did not correlate the procedures in detecting collaterals. We, therefore, compared the two procedures in the present study.

Material and Methods
The study group comprised of 17 patients with PH (13 males, 4 females; mean age 35 years, range 16-53) with endoscopically proven esophageal varices; SPV was performed within 24 hours and before any plasma fraction infusions so as to minimize hemodynamic alterations. The sonologist was not aware of clinical and endoscopic observations. The control group consisted of 100 normal healthy subjects (78 males, 22 females; mean age 32 years, range 17-50) who were examined by US only.

The patients and the control subjects were examined with real time, linear-array ultrasonic scanner (FUJIFILM 24F) using a 5-0 MHz transducer. To avoid the effects of meal, posture and respiration, the subjects fasted overnight, and all measurements were made in the supine position and during held inspiration. The portal vein (PV) was best displayed with patients in the right anterior-oblique position in the sagittal scan of the inferior vena cava, which it crosses anteriorly. The splenic (SV) and superior mesenteric (SMV) veins were seen on transverse and sagittal scans respectively, when they joined to form the portal vein. We tried to trace the complete course of the portal vessels as far as possible and looked for any echogenic mass or irregularity in their lumen. The diameters were measured at their broadest point. The subjects were scanned to demonstrate any aberrant tubular and/or tortuous anechoic venous structures in the upper abdomen corresponding to the anatomical site of various portosystemic collaterals.

SPV was performed by introducing a fine bore (21 gauge) needle and a flexible catheter over the needle under image-amplified guidance and then injecting 20 ml of meglumine iothalamate-280 into the splenic pulp in 8-12 seconds. Roentgen films were exposed at 5, 15 and 25 seconds from the start of the injection. Splenic pulp pressure was also measured during SPV examinations in all patients.

We also carried out liver biopsy using a Vim-Silverman needle, and subsequent histopathological examinations in all cases. In two cases, net wedged hepatic vein pressure over inferior vena caval pressure was estimated by right heart catheterization, recording the pressures on SAN-EI polygraph-146 machine.

All results are expressed as mean ± standard deviation (M ± SD); and the Student’s t test was used for statistical analysis.

Results
The etiology of PH in the 17 patients included cirrhosis (4 cases), non-cirrhotic portal fibrosis (6), portal vein thrombosis (3), cirrhosis with portal vein thrombosis (1) and non-cirrhotic portal fibrosis with portal vein thrombosis (3).
PV, SV and SMV were visualized in 100, 98 and 96 of the 100 normal subjects by US; in patients, these were seen by US in 17, 15 and 14 cases, and by SPV in 15, 15 and 6 cases. In control subjects, sonographically measured diameters of PV, SV and SMV were 10.4 ± 1.6 mm (8–16), 8.4 ± 1.3 mm (6–12) and 8.1 ± 1.2 mm (6–11) respectively. In patients, US measured the respective values to be 23.2 ± 4.4 mm (18–32), 17.5 ± 2.3 mm (13–21) and 16.6 ± 1.0 mm (14–18), while SPV measurements were 22.9 ± 3.6 mm (18–30), 17.4 ± 2.2 mm (14–20) and 16.2 ± 1.4 mm (14–18). The diameters were significantly wider in the patients (by both the techniques) as compared to those in the normal subjects (P < 0.001); whereas, on comparing the values obtained by the two procedures in patients, the differences were insignificant (P > 0.1).

Coronary-gastroesophageal veins could be visualized by SPV in the same patients (14 cases) in whom they were seen on sonography. Patent umbilical vein was documented by both the techniques in six cases, whereas in another five patients, in whom the vein was well displayed on oblique/longitudinal scans, SPV could not opacify it. Splenorenal shunts were seen in only three patients by US, while they were opacified in seven cases by SPV.

In seven patients with unsuspected extra-hepatic portal obstruction, portal vein thrombosis was first detected on sonographic screening; the diagnosis in all of them was similar on subsequent SPVs, except in one case, where the vein was seen completely blocked on sonography while the venography demonstrated a partial obstruction. US could display the whole course of PV and extension of the thrombus; in three cases, the whole of the vein and in four cases, the distal one third of the vein.

In eight patients, both techniques revealed no portal vascular obstruction and suggested an intrahepatic cause of PH, being confirmed by subsequent histopathologic studies, viz. cirrhosis in two cases and non-cirrhotic portal fibrosis in six cases.

In two patients, US showed dilated and non-obstructed portal vessels along with patent umbilical veins and splenorenal shunts, whereas SPV demonstrated no portal vasculature except the splenorenal shunts. Subsequent liver histopathology revealed cirrhosis, and the higher wedged hepatic vein pressures (18.0 mm Hg and 18.5 mm Hg) excluded extrahepatic obstruction in both cases.

Discussion
In the present study, US appeared to be more efficient in displaying the portal vessels, especially the mesenteric vein, as the SMV could be visualized in 14 patients with US and in only six cases on SPV. The sonographically measured portal diameters were significantly higher than the upper limit in the normal subjects. Diameters of PV, SV and SMV more than 15, 12 and 11 mm respectively could be considered diagnostic of PH; our findings substantiate those of Kurol et al.5

We observed no difference in measurements of the portal vascular diameters on US and SPV. In contrast, Webb et al.6 found the portal vein diameters on SPV (measured during expiration) 1.5–2 times greater than that by sonography (measured during inspiration) and explained that the diameters were maximum during expiration; their explanation, however, is contradicted by pathophysiology as well as ultrasonic studies,7 which have documented the largest diameters during inspiration.

Coronary-gastroesophageal collaterals were detected with equal sensitivity (14 patients) by the two techniques. However, SPV could opacify an umbilical vein in only six of 11 patients with sonographically seen patent umbilical vein, probably because of a marked hepatofugal blood flow in the rest of the cases. SPV was superior to US in demonstrating splenorenal shunts, since the presence of gas in the gastrointestinal loops and the small size of these collaterals limit the sonoographic sensitivity.

The two procedures were almost equally efficient in detecting portal vein thrombosis, found in seven patients. The efficiency of US in evaluating the whole course of the portal vessels for their patency and extension of the thrombus has an extra benefit while deciding the type of surgery.

We could not differentiate by SPV between an extra-hepatic blockade and a non-visualized patent portal vein in two cases. The inability of venography to detect the reversal of blood flow in the portal vein in the presence of a high grade intrahepatic portal obstruction has been previously shown.8 Ultrasound examination, on the other hand, could demonstrate the complete course of the vein.

Ultrasound also has its limitations. The presence of excessive gastrointestinal gas and obesity interfere with ultrasonic visualization of the portal venous system.4 US does not provide any clue regarding the location of portal blood flow unless combined with pulsed Doppler duplex devices, which are, however, quite costly, and their operation and interpretations are rather difficult and time-consuming.15 Furthermore, sonography, unlike venography, cannot provide a complete picture of the portal system in one setting, and provides different views of the portal vessels (not in usual anatomical planes), which make their interpretation difficult for the operating surgeons.

Sonography appears to be a reliable screening device and assists in the diagnostic efficacy in certain situations, but cannot replace the venographic procedure, particularly in the surgical management of patients with portal hypertension.

References
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