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Abstract

Background: We retrospectively evaluate the potential protective influence of patent paraumblical vein (PUV) collaterals against portal vein (PV) thrombosis and reduced PV diameter in candidates for orthotopic liver transplant (OLT)

Methods: Dynamic 3D contrast-enhanced MRI at 1.5T was obtained in 309 patients with cirrhosis without evidence of malignancy. All MR studies were reviewed by one reader for PUV collaterals, PV thrombosis and PV diameter. Statistical analysis was performed by Fisher exact tests; 50 selected studies were reviewed independently by two additional readers to determine interobserver agreement via intraclass correlation coefficient (ICC).

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Results: Patent PUV was noted in 119 of 309 patients (38.5%). Mean PV diameter was 13.4 ± 3.0 mm in patients with PUV compared with 11.3 ± 3.6 mm without PUV ($P < 0.01$). Main PV thrombosis was present in 13 of 309 patients (4.2%) and significantly more frequent in those without PUV than with PUV (6.3% vs. 0.8%, $P < 0.05$). ICC indicated almost perfect agreement among three readers for presence of PUV collaterals (ICC = 0.91) and PV thrombosis (ICC = 0.96).

Conclusion: Our results suggest that patients with patent PUV appear less likely to develop main PV thrombosis or small PV diameter, suggesting a protective effect of PUV on PV patency.

Key words: Paraumbilical vein—Cirrhosis—Portal vein thrombosis—MRI-liver transplantation

Cirrhosis of the liver is characterized by progressive fibrosis and architectural disarrangement, leading to an increased intrahepatic resistance to portal venous inflow and the development of portal hypertension. This is exacerbated by an increase in portal blood flow, promoted by splanchnic arteriolar vasodilatation and splenomegaly [1]. The pathological increase in portal pressure leads to the development of a collateral portal-systemic circulation, which forms in an attempt to decompress the portal system by diverting blood flow to systemic veins [2]. This in turn decreases portal vein flow volume and velocity, which is probably a factor in the

high incidence of portal vein (PV) thrombosis among patients with portal hypertension [3–5].

Liver transplantation is sometimes needed to treat hepatic failure or early stage hepatocellular carcinoma. Adequate portal venous inflow to the hepatic allograft is mandatory for successful liver transplantation [6]. PV thrombosis or small PV caliber less than 1 cm while not an absolute contraindication to orthotopic liver transplantation (OLT), has a substantial effect on surgical complexity and perioperative morbidity and mortality rates [7]. To properly plan for OLT and to minimize operative time, preoperative detection of PV thrombosis is of paramount importance. Color flow Doppler sonography and conventional angiography are commonly used to study the portal venous system, but each has its limitations. For this reason, regular short-interval serial MRI is recommended for candidates for hepatic transplantation, to direct intraopertive visualization [8– 10].

A subset of patients with portal hypertension shunt their portosystemic flow primarily through paraumbilical vein (PUV) collaterals, which drain blood from the left PV toward veins of the ventral abdominal wall [11]. This portosystemic shunt is different from others, in that it involves shunting through the main PV, rather than away from it. We are not aware of any data to support the theoretical protective effect of patent PUV against PV thrombosis and reduced PV size. Thus we retrospectively evaluate the potential protective influence of PUV collaterals against PV thrombosis and reduced PV diameter in candidates for OLT.

Material and methods

Patients

This HIPAA compliant retrospective study was approved by our institutional review board. Informed consent was not required. Liver MR records and clinical MR requests from January 1999 through January 2005 at our institution were searched and then cross-referenced to the histopathology records to identify patients with histopathologically proved cirrhosis. Patients were excluded from the study (a) if they had any evidence of solid malignant tumors [primary HCC (n = 68), primary pancreaticobiliary cancer (n = 12), secondary malignancy (metastasis from pancreatic cancer n = 9, colorectal carcinoma n = 3, renal carcinoma n = 3] (b) cirrhosis without histopathological confirmation (n = 17), transjugular intrahepatic portosystemic shunt (TIPS) (n = 2). Consequently the study population included 309 consecutive patients (191 men and 118 women) aged 18–86 years (mean age 54 years). These patients had been referred for MR imaging to evaluate the severity of cirrhosis and portal hypertension and to acquire preoperative studies before liver transplantation. All patients had biopsy-proven cirrhosis and documented portal hypertension proven by endoscopic visualization of varices or established imaging criteria [12]. The cirrhosis was attributed to viral hepatitis alone in 232 patients (HCV n = 170, HBV n = 52, both n = 10), alcohol in 48, cryptogenic in 12, primary sclerosing cholangitis in seven, autoimmune in three, primary biliary cirrhosis in three, non alcoholic steatohepatitis in two, congenital biliary atresia in remaining two patients.

Cirrhosis was histopathologically confirmed by means of percutaneous liver

biopsy in 280 patients and transjugular liver biopsy in 29. According to Child-Pugh classification, of the 309 patients, 93 were in class A, 147 in class B and 69 in class C.

MR Imaging

All MR examinations were performed during suspended respiration with a 1.5-T system and phased array coil (Signa: General Electric Medical Systems, Milwaukee, WI; or Achieva: Philips, Best, Netherlands). The sequences included two-dimensional (2D) coronal and axial single-shot fast spin-echo T2-weighted MR images (TR infinite; TE 180–200 ms); axial fat suppressed fast spin-echo T2-weighted MR images (TR 2500–4000 ms, TE 70–90 ms); spoiled dual gradient echo (SGRE) T1weighted in-and out-of-phase MR images (TR 120– 200 ms, TE = 2.3 and 4.6 ms, flip angle 90 º). Parameters for 2D images included section thickness 5–8 mm with intersection gap 0–1 mm; matrix, 256 ·160–192; field of view 32 cm transverse and 24 cm anteroposterior; NSA = 1 or less. Three-dimensional (3D) dynamic enhanced transverse SGRE fat suppressed MR images were obtained with 5 mm thick resolution in the slice axis, at 2.2–2.5 mm increments using zero-fill interpolation, TR/ TE/flip angle = 3–6.1/0.9–2.1/12–20º, and parameters otherwise similar to those of the 2D images. Acquisition time per 3D volume was 21 s, obtained during suspended respiration; 20 cc of contrast material (Magnevist; Berlex Laboratories, Wayne, NJ, USA) was administered intravenously via power-injector (OptistarTM LE; Mallinckrodt; Hazelwood, MO, USA) at 2 mL/s followed by a 20-mL saline solution flush. First-pass arterial enhancement was optimized using a timing

bolus sequence, or by observing enhancement on images reconstructed real-time. Dynamic imaging was performed during breath-hold before the injection (unenhanced), immediately after injection (hepatic arterial phase) and 30 s afterward (portal venous phase). Additional delayed phase images of the entire liver were acquired using a 2D single-section SGRE technique with TR/TE of 19–20/ 1.5–2.1 and flip angle, 40 º; in some patients, an additional delayed phase 3D SGRE series was acquired.

Review of records and images

MR images were reviewed independently on a picture archiving and communication system (PACS) workstation (Canon Medical Systems, Irvine, CA, USA) initially by one radiologist (S.K.V with 3 years of body MR experience), without access to the prospective reports or any clinical information.

All patients were screened for the presence of portosystemic shunts and varices such as splenorenal shunt, esophageal varices with particular attention to PUV that runs along the ligamentum teres in the falciform ligament. Based on the retrospective image review, two groups from within the study population, one with and one without patent PUV were identified. Patent PUV was defined as a clearly visible vessel connecting the left PV to collateral veins at the anterior abdominal wall. Maximum transverse diameters of PUV at its widest part were recorded on 3D gadolinium-enhanced GRE fat suppressed sequence during portal venous phase.

Thrombosis was classified as occlusive or nonocclusive. The maximum transverse diameter of the main PV was measured with calipers from wall to wall

midway between the splenoportal confluence and the portal vein bifurcation in the porta hepatis on 3D gadolinium-enhanced GRE fat suppressed sequence during portal venous phase. The splenic vein was evaluated from the confluence to splenic hilum; and the superior mesenteric vein, from the confluence to the right colic branch. The vessels were defined as patent if the entire lumen was filled with contrast material—enhanced blood on enhanced images. An occlusive thrombus was defined as a nonenhancing filling defect within the lumen of the vessel seen on contrast enhanced images. Nonocclusive thrombi were identified when there was contrast-opacified blood adjacent to the existing thrombus, and these thrombi were further divided into those occupying more than 50% of the lumen or less than 50% of the lumen. A splenorenal shunt was defined as a spontaneous anastomosis of the splenic vein or a perisplenic varix to an enlarged left renal vein. Presence or absence of ascites was noted.

To evaluate interobserver agreement, a subset of 50 examinations were reviewed independently by two additional readers (D.G.M., Y.L with 21. 2 years of body MRI experience) for assessment of PV thrombosis and PUV, who were blinded to the initial evaluation by the first reader. These 50 examinations included all patients with main or branch PV thrombus identified by the first reader (n = 17), and 33 arbitrarily selected additional examinations from both PUV and non-PUV groups. Data were entered into a spreadsheet (Excel; Microsoft, Redmond, WA, USA). When the readers disagreed about the presence of PV thrombosis, a majority opinion was used as final decision for data analyses.

Statistical evaluation

Statistical analysis was performed with SAS software (SAS Institute, Cary, NC, USA). Descriptive statistics (mean, SD) were provided where appropriate. A non-paired Student t test was used to assess the statistical significance of diameter of main PV between patients with vs. without patent PUV. We used a Fisher exact test to determine the statistical significance of differences among the patients with and without PUV in incidence of main PV thrombosis, occluded main PV thrombosis and main PV size less than 1 cm. Reliability for the presence PV thrombosis and PUV collaterals for the three readers was assessed by computing the intraclass correlation coefficient (ICC). ICC for inter-observer reliability was performed using Shrout–Fleiss methodology [13].

Results

Results are summarized in Table 1. Main PV thrombosis was present in 13 of 309 (4.2%) patients; main PV thrombosis was occlusive in six and nonocclusive in seven. For all 13 patients with main PV thrombus, the blinded retrospective detection of presence and extent of thrombus agreed with the findings in the prospective report. Multireader discrepancy in interpreting the presence of thrombus in main PV occurred in only 1 of 13 patients with partial PVT, which was resolved in consensus as presence of PV thrombosis. Interobserver ICC for main PV thrombosis was 0.96. Main PV thrombus extended to the confluence of the splenic and superior mesenteric veins in two patients. No patients had thrombosis of the

splenic vein, superior mesenteric vein or confluence without involvement of the main PV. Four patients without main PV thrombus had branch thrombi identified. These four examinations were included among the 50 reviewed by additional readers, but thrombi that did not involve the main PV were not considered further.

Splenorenal shunt was recognized in 24 (8%) of 309 patients including 5 of 119 (4.2%) with PUV and 19 of 190 (10%) patients without PUV ($P < 0.05$).

Patent PUV was noted in 119 of 309 patients (38.5%) (group 1), with a mean diameter of 5.2 ± 3.3 mm (range 2–19 mm). The prevalence of PUV were more often present in patients with Child-Pugh class C (52%)or class B clinical status (41%) than in those with class A status (24%)($P < 0.05$).

For the subset of 50 patients viewed by three readers, interobserver ICC for PUV collaterals was 0.91. PUV was not present in any of the six patients who had occlusive main PV thrombosis ($P = 0.08$) (Fig. 1). Overall, main PV thrombosis which was nonocclusive seen only in 1 of 119 (0.8%) patent PUV (Fig. 2), compared with 12 of the 190 (6.3%) without PUV ($P = 0.02$). Mean PV diameter was 13.4 ± 3.0 mm among the 119 patients with PUV (Fig. 3) compared with 11.3 ± 3.6 mm among the 190 patients without PUV (group 2) ($P < 0.01$). PV diameter size less than 1 cm was seen in 10 of 190 (5.1%) patients without PUV, compared with 3 of 119 (2.5%) patients with PUV ($P = 0.19$). Three of these patients with PUV who had PV less than 1 cm had splenorenal shunt (diameters 8, 10 and 20 mm; mean 12.6 ± 6.4 mm) (Fig. 4), none had ascites.

Esophageal varices were seen in 57 of 309 (18%) patients, including 26 of 119 (22%) with PUV and 31 of 190 (16%) patients without PUV (P = NS).

Discussion

The presence and hemodynamic effects of patent PUV in patients with portal hypertension have been examined with Doppler sonography and MRI [8–10, 14], with reported frequency of PUV ranging from 11 to 97% [11, 15–17]. In our study, we detected PUV in 38% of MRI examinations in the patients with portal hypertension. The increasing prevalence of PUV collaterals with progression of liver disease was evident in the present study, in which shunts were found significantly more frequently in patients with grade C or B cirrhosis than in those with grade A cirrhosis [18].

The PV diameter, averaging 1 cm in healthy adults [19], increases during the initial stages of portal hypertension [12, 20], with a PV larger than 13 mm reported as characteristic of portal hypertension in the appropriate clinical setting [21]. However, massive shunting away from the liver in later stages is associated with reduced PV caliber or with portal thrombosis, both of which can complicate OLT, particularly if not detected preoperatively [14, 22]. The incidence of PV thrombosis varies according to the characteristics of the patients evaluated, ranging from 0.6 to 16.6% [23–26]. Therefore, in our clinical practice, we routinely evaluate the patency of the PV preoperatively, using frequent sonography and annual MRI, to determine if an alternative surgical method is necessary to assure portal inflow.

Ability to detect PUV collaterals is important in diagnosing and managing patients with chronic liver disease and portal hypertension. It is necessary for transplant surgeon to know the extend and patency of PUV collaterals as it may increase the

risk of bleeding if it is occluded due to elevated portal pressure. Although portal hypertension decreases flow to hepatic parenchyma regardless of the character of portosystemic shunting, a PUV allows this shunting to occur through the main PV, rather than away from it, thereby maintaining the volume and velocity of main PV flow, potentially preventing reduced main PV size and reducing the likelihood of main PV thrombosis [27]. In contrast, in the presence of a large splenorenal shunt, a portion of splenic venous blood is diverted into the shunt and subsequently causes a significant reduction of portal venous velocity and flow volume [28–30]. Our observations have confirmed the expected protective effect of PUV against complete PV thrombosis consistent with previous observations [31] of higher PV mean velocity in the presence of a patent PUV. However the magnitude of this protective effect may be reduced if the PUV is small, or if massive extrahepatic (e.g. splenorenal) shunt develops, as in all 3 of 119 patients with PUV who had PV size less than 1 cm or if ascites is present [32], as in 1 of 119 patient with PUV who had non occlusive thrombus in main PV.

The potential protective effect of PUV on maintaining PV diameter is supported by our finding of larger PV size in patients with PUV compared to those without PUV. However, the incidence of portal vein smaller than 1 cm was not significantly different between these two groups, possibly because of variable contribution towards portosystemic shunting of splenorenal and other shunts. Another potential protective effect of PUV, against bleeding from esophageal varices, cannot be directly addressed from our study.

Our study has several limitations. One limitation was our inability to confirm our

imaging findings by conventional direct or indirect portographic methods. Conventional portography is invasive and therefore was not performed in most patients. Many investigators have reported the use of Doppler sonography [27, 33]to determine the direction of portal flow. In our experience, we have noted some problems, including body habitus, operator dependence, failure to depict partial thrombosis or shunts, and the false positive diagnosis of PV thrombosis due to stagnant or hepatofugal flow. Any of a number of technical factors (such as inappropriate color gain, output, sensitivity, and gray-scale vs. color write priority settings) could have resulted in an artifactual lack of flow.

Our retrospective study did not provide adequate data to determine which patients had hepatofugal flow. Since many patients with PUV also had other varices which may have contributed as a risk for PV thrombosis, we could not determine the relative contribution of PUV towards the overall volume of portosystemic shunting. A prospective study of the effects of PUV, including measurement of blood flow velocity of the intrahepatic right and left PV, main PV and patent PUV as well as portohepatic gradient, would provide deeper understanding. Due to retrospective design, we had small numbers of patients who had occlusive main PV thrombosis and main PV size less than 1 cm in both groups to allow for a sufficiently powerful statistical analysis, however trends towards increased frequency of both of these complications were observed in patients without PUV. We did not use Doppler ultrasound in most patients, but suspect that a similar relationship between paraumbilical collateral and portal thrombosis would be seen using this or other methods for evaluating the portal venous system.

Another limitation of our study is that most examinations were only reviewed by one radiologist, although multireader review of 50 cases showed near perfect agreement regarding PV thrombosis and PUV. It is possible that one or more PUVs might have been missed, but this does not affect our observation that patients with main PV thrombosis were unlikely to have PUV.

In summary, our findings indicate that an enlarged patent PUV decreases the risk of main PV thrombosis in patients with portal hypertension who are candidates for liver transplantation.

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TABLES & FIGURES

Table 1. Summary of 309 patients with cirrhosis

Findings	Patients with PUV Group 1 (n = 119)	Patients with no PUV Group 2 (n = 190)	P value
Overall main PV thrombosis	1	12	<0.05
Occlusive main PV thrombosis	0	6	NS
Small main PV (<1 cm)	3	10	NS
Main PV diametera	13.4 ± 3.0	11.3 ± 3.6	<0.01
Splenorenal shunts	5	19	<0.05
Esophageal varices	26	31	NS

Data are the number of patients
NS, Not significant
[a] Data are mean ± SD

Figure 1. Forty-eight-year-old man with chronic portal vein (PV) thrombosis. Axial T1-weighted 3D gadolinium-enhanced gradient-echo dynamic MR images (TR/TE, 6/2.0) obtained during portal venous phase reveals (A) portal venous occlusion with cavernous transformation of the PV (arrow) at the porta hepatis (B) no evidence of patent paraumblical vein in the falciform ligament (arrow). Signs of portal hypertension splenomegaly and portosystemic collateral vessels are evident.

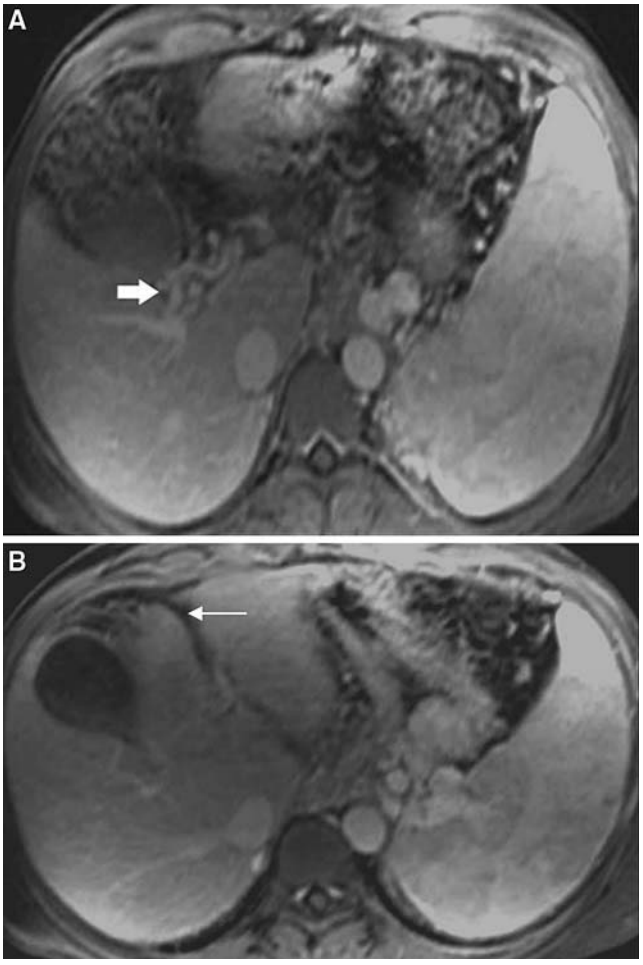

Figure 2. Patent paraumblical vein (PUV) and portal vein (PV) thrombosis in 61-year-old man with portal hypertension. Axial T1-weighted 3D gadolinium-enhanced gradient-echo dynamic MR image (TR/TE, 5/1.6) obtained during portal venous phase reveals patent PUV (white arrow) and nonocclusive thrombus in the main PV (black arrow) at the portal confluence. A moderate amount of ascites is present.

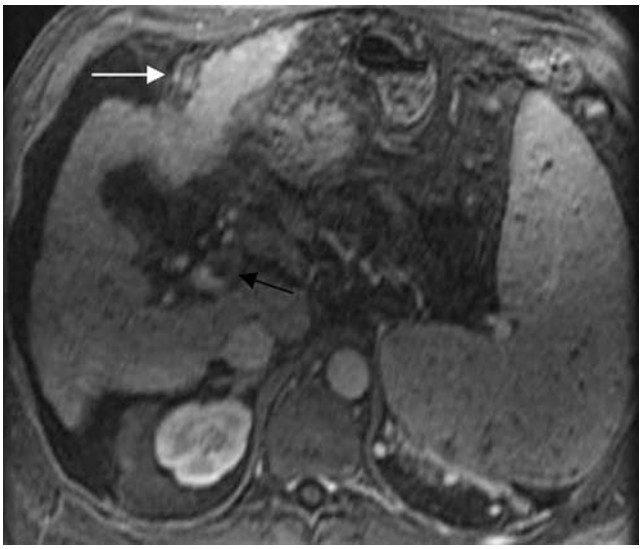

Figure 3. Patent paraumblical vein (PUV) in a 60-year-old woman with portal hypertension. Axial T1-weighted 3D gadolinium-enhanced gradient-echo dynamic MR image (TR/TE, 6/2.0) obtained during portal venous phase reveals patent intrahepatic portion of PUV with a diameter of 11 mm (white arrow) and normal intensely enhancing main portal vein (black arrow) (14 mm in diameter).

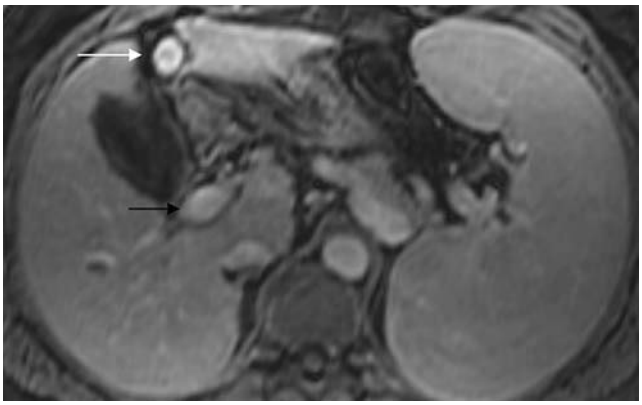

Figure 4. Patent paraumblical vein (PUV) and splenorenal portosystemic shunt in a 44-year-old man with portal hypertension. Axial T1-weighted 3D gadolinium-enhanced gradientecho dynamic MR image (TR/TE, 5/1.6) obtained during portal venous phase shows (A) small (8 mm) main portal vein (white arrow) and prominent splenorenal collateral vessel (black arrow). The decreased signal-intensity area seen within the portal vein is due to the flow artifact. (B) Axial T1weighted 3D gadolinium-enhanced gradient-echo dynamic MR image (TR/TE, 5/1.6) obtained during portal venous phase reveals patent PUV (white arrow) and prominent splenorenal collateral vessel (black arrow).