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Original Article

Portal and hepatic vein thrombosis after transjugular intrahepatic portosystemic shunt: Incidence in follow-up imaging and clinical implications

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A B S T R A C T

Background: This study investigated the incidence and clinical outcomes of portal and hepatic vein thrombosis (VT) on imaging after transjugular intrahepatic portosystemic shunt (TIPS).

Methods: A retrospective review of records at a single liver transplant center between 2010 and 2018 revealed 423 patients who underwent TIPS. Contrast-enhanced computed tomography and magnetic resonance images within 1 year post-TIPS were available for 138 patients and compared to assess the imaging findings of VT and liver infarction. The associations of VT with overall survival, patient characteristics, stent size, pre- and post-TIPS Model for End-Stage Liver Disease (MELD) scores, and post-TIPS hepatic encephalopathy at 90 days were analyzed.

Results: The prevalence of VT on imaging within 1 year was 63.0% ($n = 87$). VT within the right portal vein was more common: 41 cases were in the right portal vein, 25 in the posterior portal vein, and two in the anterior right portal vein. Ten patients had VT in the left portal vein. Four had VT in the main portal vein (MPV), and one had shunt thrombosis extending into the superior mesenteric vein. Hepatic VT was seen in the right hepatic vein in 17 patients and in the middle hepatic vein in six patients. VT was associated with liver infarction ($n = 9$, $P = 0.018$). There was no relationship between VT and sex, age, cirrhosis etiology, indication for TIPS, stent size, or hepatic encephalopathy at 90 days. VT in the MPV had poorer survival ($P < 0.001$). Older age ($P = 0.028$) and higher pre-TIPS MELD score ($P = 0.049$) were poor prognostic factors. VT was not treated.

Conclusion: Portal and hepatic VTs were common imaging findings after TIPS without worsened clinical outcomes unless VT involved the MPV. VT may cause liver infarction, but infarcts were not independently associated with poorer survival.

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Keywords: Hepatic vein thrombus; Liver infarction; Portal vein thrombus; Transjugular intrahepatic portosystemic shunt

Introduction

Transjugular intrahepatic portosystemic shunt (TIPS) is a minimally invasive procedure that has mostly replaced surgical portosystemic shunts for the treatment of many of the complications of portal hypertension in patients with liver cirrhosis, including recurrent variceal bleeding, refractory ascites, and hepatic hydrothorax.^{1–5} The technical success rate for TIPS placement is greater than 90%, with a rate of procedural complications ranging from 10% to 16%.⁶ Mortality related to TIPS is usually less than 2%; rare life-threatening complications include hemoperitoneum, hemobilia, liver ischemia, cardiac failure, and sepsis.^{5,6}

Segmental portal and hepatic vein thrombosis (VT) after TIPS has been reported in Doppler ultrasound studies.⁶ Lopera et al⁷ reported that segmental infarction was associated with thrombosis of major branches of the portal and hepatic veins, and the clinical outcomes of patients with hepatic infarction varied from self-limited transaminitis to acute liver failure with resultant mortality. To our knowledge, however, little is known about the incidence of VT in axial contrast-enhanced imaging studies. In addition, few studies have reported the long-term clinical significance of VT. Therefore, this retrospective study investigated the incidence and clinical outcomes of portal and hepatic VT in follow-up imaging after TIPS.

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Methods

After Institutional Review Board approval, data were extracted in accordance with the Health Insurance Portability and Accountability Act of 1996 from a single tertiary care liver transplant center, and 423 sequential patients who underwent TIPS between January 2010 and December 2018 were investigated (Fig. 1). Patients were included in this retrospective study if contrast-enhanced computed tomography (CT) scans or magnetic resonance imaging (MRI) scans with gadolinium were available within 1 year after TIPS. The imaging protocols included abdominopelvic CT with intravenous contrast, four-phase liver CT, and a liver MRI protocol that adhered to the American College of Radiology practice parameters. In total, 285 patients were excluded because of hepatic or portal VT prior to TIPS, prior liver transplant, prior hepatic surgery, or prior TIPS. We recorded demographic variables, pre-TIPS and post-TIPS Model for End-Stage Liver Disease (MELD) scores at 45 days post-procedure, cause of cirrhosis, indication for TIPS, shunt diameter, presence of encephalopathy at 90 days, liver infarcts on imaging, and location of the portal and hepatic VT for the 138 included patients. The majority of the included patients underwent 8- or 10-mm covered Viatorr stent graft (Gore Medical, Flagstaff, AZ, USA) placement while 6 patients underwent uncovered Wallstent (Boston Scientific, Marlborough, MA, USA) placement. Most TIPS procedures involved an access route from the right hepatic vein (RHV) to the right portal vein using fluoroscopic guidance.

The primary objectives of the analysis were to investigate the incidence and clinical outcomes of patients with VT seen on cross-sectional imaging within 1 year of the TIPS procedure. Images were independently assessed for the presence of thrombosis in the portal and hepatic venous system, as well as the presence of liver infarcts. VT was categorized according to whether it occurred in the right anterior portal vein (RAPV), right posterior portal vein (RPPV), right portal vein (RPV), left portal vein (LPV), main portal vein (MPV), left hepatic vein (LHV), middle hepatic vein (MHV), or RHV. A vein was determined to be thrombosed if it was completely or partially occluded with thrombus or if it was not visible, but the contralateral side was opacified. If the vein was attenuated but opacified, then the vein was classified as patent. The presence of liver infarcts, classified as focal areas of nonenhancement on contrast-enhanced images, was recorded.

Categorical variables were summarized as counts and percentages. The grouping variable of interest was the presence of VT in the portal or hepatic venous bed on follow-up cross-sectional imaging within 1 year after TIPS. The chi-square test was calculated to assess independence between patient characteristics and the presence of VT. The Levene test was used for continuous variables to determine equality of variances; the Student *t*-test was used if variances were equal, and the Mann-Whitney U test was used if nonparametric tests were necessary. Kaplan-Meier analysis with the log-rank test and univariate Cox hazards regression was used to determine associations with survival. All statistical tests were two-sided, and differences were considered statistically significant at $P < 0.05$. Statistical analysis was performed using IBM SPSS version 25.0 (IBM Corp., Armonk, NY, USA).

Results

Patient characteristics

The median age of the 138 included patients was 55 years old (range, 28–76 years old). Most ($n = 96$, 69.6%, $P = 0.117$) were male. The median pre-TIPS MELD score was 13 (4–26, $P = 0.442$), the median post-TIPS MELD score was 15 (6–37, $P = 0.400$). The most common cause of cirrhosis was longstanding alcoholic liver hepatitis (31.2%, $P = 0.118$). Refractory ascites (52.9%, $P = 0.485$) and variceal bleeding (30.4%, $P = 0.842$) were the two most common indications for TIPS. Sixty-three patients (45.7%) had 8-mm portosystemic stents placed and the remaining 75 patients (54.3%) had 10-mm stents placed (Table 1).

The mean time to obtaining cross-sectional imaging after TIPS was 97 days (1–365 days). Eighty-seven (63.0%) patients had VT either in the portal or hepatic venous system 1 year after TIPS. Sixty-four had portal VT and 14 had hepatic VT. The most thrombosed vein was the RPV, at the lobar area in 41 (29.7%), RPPV in 25 (18.1%) and RAPV in two (1.4%) patients (Table 2). Ten patients had VT in the LPV (7.2%). Among these patients, six had combined VT of the RPV and LPV with the MPV being patent. Four patients had a thrombus in the MPV (2.9%). Seventeen patients had VT in the RHV (12.3%) and six in the MHV (4.3%). Nine patients had combined portal branch (7 RPPV, 3 RPV, and 1 LPV) and hepatic VT (8 RHV, and 1 MHV).

Clinical outcomes and survival

The presence of hepatic or portal VT did not significantly affect overall survival. Those with patent veins had a median survival of 24.7 months (95% CI, 5.1–44.3 months) versus 47.7 months (95% CI, 30.9–64.3 months) ($P = 0.085$) for those with VT. The median follow-up time was 36.1 months (27.8–44.3 months). From our survival data, at year 1, 83% of those with VT and 63% of those with patent veins survived. Thirty patients (21.7%) died within 1 year and 58 patients (42.0%) died during the study period. Twenty patients (14.5%) were lost to follow-up, 16 of whom had VT. Sixteen patients (11.6%) received liver transplants, three of which were emergent. Within 90 days of stent placement, 45 patients (32.6%) were diagnosed with clinically significant hepatic encephalopathy that required hospitalization.

Thrombosis in the MPV was associated with worsened mortality, with a median survival of 1.1 months (0.0–4.1 months; $P < 0.0001$). Three of the four patients experienced mortality prior to their TIPS anniversary and the other was lost to follow-up. One patient (MELD 9) with VT in the MPV, thrombosed TIPS, and a concomitant large infarction, died due to complications of pro-

Fig. 1. Patient selection. TIPS, transjugular intrahepatic portosystemic shunt; PACS, picture archiving and communication system; VT, vein thrombosis.

Table 1 Patient Characteristics and Outcomes

Characteristic	All patients	Venous thrombosis*	Patent veins	P-value
Sample size (<i>n</i>)	138	87	51	
Age (yr)	55 (28–76)	55 (28–76)	53 (30–73)	0.451
Pre-TIPS MELD	13 (4–26)	12 (6–26)	13 (4–21)	0.442
Post-TIPS MELD	15 (6–37)	14 (6–37)	13 (7–33)	0.400
Sex				
Male	96 (69.6)	57 (65.5)	39 (76.5)	0.117
Female	42 (30.4)	30 (34.5)	12 (23.5)	
Causes of cirrhosis				
Alcoholic hepatitis	43 (31.2)	23 (26.4)	20 (39.2)	0.118
HCV	20 (14.5)	15 (17.2)	5 (9.8)	0.231
Non-alcoholic steatohepatitis	10 (7.2)	8 (9.2)	2 (3.9)	0.249
Primary biliary cholangitis	7 (5.1)	6 (6.9)	1 (2.0)	0.202
Wilson's disease	2 (1.4)	2 (2.3)	0 (0.0)	0.275
Autoimmune	2 (1.4)	2 (2.3)	0 (0.0)	0.275
Alcoholic and HCV	39 (28.3)	20 (23.0)	19 (37.3)	0.072
Cryptogenic	15 (10.9)	11 (12.6)	4 (7.8)	0.382
Indications for TIPS				
Refractory ascites	73 (52.9)	48 (55.2)	25 (49.0)	0.485
Variceal bleeding	42 (30.4)	27 (31.0)	15 (29.4)	0.842
Hydrothorax	17 (12.3)	8 (9.2)	9 (17.6)	0.145
Budd-Chiari syndrome	1 (0.7)	1 (1.1)	0 (0.0)	0.442
Multifactorial	5 (3.6)	3 (3.4)	2 (3.9)	0.089
Stent size				0.543
8 mm	63 (45.7)	38 (43.7)	25 (49.0)	
10 mm	75 (54.3)	49 (56.3)	26 (51.0)	
Liver infarct	9 (6.5)	9 (10.3)	0 (0.0)	0.018
Liver transplant	16 (11.6)	10 (11.5)	6 (11.8)	0.962
Post-TIPS hepatic encephalopathy at 90 days	45 (32.6)	31 (35.6)	14 (27.5)	0.322

Values are presented as median (range) or number (%).

MELD, Model for End-Stage Liver Disease; TIPS, transjugular intrahepatic portosystemic shunt; HCV, hepatitis C virus.

*Portal or hepatic venous thrombosis on imaging.

Table 2 Overview of Findings of the Segmental, Main Portal, and Hepatic Vein in 138 Patients with Available Imaging

Finding	RAPV	RPPV	RPV	LPV	MPV	RHV	MHV
Thrombosed/absent	2 (1.4)	25 (18.1)	41 (29.7)	10 (7.2)	4 (2.9)	17 (12.3)	6 (4.3)
Patent	136 (98.6)	113 (81.9)	97 (70.3)	128 (92.8)	134 (97.1)	121 (87.7)	132 (95.7)

Values are presented as number (%).

RAPV, right anterior portal vein; RPPV, right posterior portal vein; RPV, right portal vein; LPV, left portal vein; MPV, main portal vein; RHV, right hepatic vein; MHV, middle hepatic vein.

gressive hepatorenal syndrome.

Additionally, older age ($P = 0.028$) and a higher pre-TIPS MELD score ($P = 0.049$) were independently associated with decreased survival. The mean MELD score increased after TIPS by 1.6 (95% CI, 0.7–2.4; $P < 0.001$), from 13.0 to 14.6. Sex, the etiology of cirrhosis, the indication for TIPS, shunt diameter, and the location of thrombosis aside from MPV were not significant or inconclusive, as shown in Table 3.

Liver infarct and vein thrombosis

Nine patients had liver infarcts and VT ($n = 8$ portal, $n = 1$ portal and hepatic). VT in the portal and hepatic veins was associated with the presence of liver infarction ($P = 0.018$). Neither portal/hepatic VT ($P = 0.085$) nor liver infarcts ($P = 0.200$) were associated with overall survival. The median 1-year survival of patients with liver infarcts and VT versus patients without liver infarcts and VT was 8.5 months and 10.9 months, respectively ($P = 0.081$). Except for liver infarcts, there was no independent rela-

Table 3 Univariate Kaplan-Meier and Cox Analysis Overall Survival in Months

Variable	P-value	Hazard ratio	Median survival	95% confidence interval
Overall survival			40.9	33.394–48.339
Age*	0.028	1.035		1.004–1.068
Pre-TIPS MELD score*	0.049	1.068		1.000–1.141
Post-TIPS MELD score*	0.005	1.082		1.024–1.143
Sex	0.932			
Female			46.4	39.115–53.645
Male			39.9	15.991–63.743
Presence of VT	0.085			
Venous thrombosis			47.7	30.942–64.339
Patent veins			24.7	5.061–44.339
Causes of cirrhosis				
Alcoholic hepatitis	0.517		39.9	8.104–71.630
HCV	0.739		46.4	21.631–71.169
NASH/NAFLD	0.965		39.8	4.262–75.338
PBC [†]	0.807		–	–
Wilson disease [†]	0.055		5.4	–
Autoimmune hepatitis [†]	0.473		–	–
Alcoholic and HCV	0.695		15.5	0.000–37.257
Cryptogenic	0.942		40.9	39.225–42.508
Indications for TIPS				
Refractory ascites	0.311		40.9	29.112–52.622
Variceal bleeding [†]	0.093		58.0	–
Hydrothorax	0.584		26.2	9.694–42.706
Budd-Chiari syndrome [†]	0.029		5.4	–
Multifactorial	0.695		11.1	0.000–37.257
Shunt diameter	0.695			
8 mm			39.9	16.557–63.176
10 mm			46.4	25.980–66.820
Encephalopathy at 90 days	0.071			
Yes			39.8	7.635–71.965
No			47.7	33.924–61.409
Liver infarct	0.200			
Yes			26.2	0.000–58.621
No			46.4	38.985–53.815
Portal and hepatic bed thrombosis				
Right posterior portal vein	0.691		40.2	29.057–51.277
Right anterior portal vein [†]	0.600		–	–
Right portal vein [†]	0.059		51.3	–
Main portal vein	< 0.001		1.1	0.000–4.171
Left portal vein	0.716		58.0	0.000–135.002
Superior mesenteric vein [†]	< 0.001		1.1	–
Middle hepatic vein	0.500		15.6	0.000–61.954
Right hepatic vein [†]	0.258		40.2	–

TIPS, transjugular intrahepatic portosystemic shunt; MELD, Model for End-Stage Liver Disease; VT, vein thrombosis; HCV, hepatitis C virus; NASH, non-alcoholic steatohepatitis; NAFLD, nonalcoholic fatty liver disease; PBC, primary biliary cholangitis.

*Hazard ratio with 95% confidence intervals reported.

[†]Greater than 50% of the data are censored, and the standard error and confidence intervals were indeterminate.

tionship between any other patient characteristic and VT (Table 1).

Of the nine patients with liver infarcts, three were lost to follow-up. Two patients underwent liver transplantation for elevated MELD scores approximately 1 month (MELD 23) and 10 months (MELD 16) after the TIPS procedure, respectively. Three patients died from complications unrelated to the TIPS procedure; one patient (MELD 20) died within 1 month of the same hospital admission for spontaneous bacterial peritonitis, refractory ascites, and volume overload, and two patients (MELD 9, 10) died from progressive hepatorenal syndrome 1 month and 2 years after the TIPS procedure, respectively. One patient (MELD 10) died 4 months after TIPS without a known documented etiology at an outside facility.

Discussion

Portal and hepatic VT is a common imaging finding in contrast-enhanced axial images after TIPS, mainly affecting the RPV branches and less frequently the LPV branches, MPV, and the hepatic veins. Associated parenchymal changes including lobar infarcts were observed in a minority of patients. The presence of neither VT nor liver infarcts showed a statistically significant relationship with survival unless the thrombus was in the MPV. Older age, a higher pre-TIPS MELD score, and a higher post-TIPS MELD score were expectedly associated with poorer survival.

Hemodynamic changes in the portal circulation after TIPS have been well described in Doppler studies. Early after TIPS, the flow in the MPV increases dramatically,⁸ thus explaining the increased mortality with MPV thrombi. Flow in the intrahepatic portal branches is usually reversed and directed preferentially toward the shunt due to its lower resistance than the liver parenchyma.⁹ However, bidirectional flow and stagnant undetectable flow in the intrahepatic branches have also been described.^{10,11} These flow alterations in the intraparenchymal portal branches may be responsible for segmental small branch portal VT, as seen in our study. Thrombosis of the umbilical portion of the LPV was frequently seen in one Doppler study.⁸ In our study, the incidence of VT was predominantly within the right segmental branches of the portal and hepatic veins.

A potential explanation for the predominant tendency for the right portal branches to be affected, especially the RPPV, is that TIPS at our institution, like many other centers, are created between the RHV and the RPV branches, with the initial needle access targeting the posterior branches of the RPV in most cases. In a minority of procedures, the puncture is performed in the LPV. Manipulation of the right portal system and RHV during the standard TIPS procedure accounts for the expected higher incidence of VT within the involved vasculature. In most patients, the RHV is used for the initial puncture, and the MHV is also not infrequently catheterized during TIPS. Hence, in our study there was a greater proportion of patients with VT in the RHV (17 patients) than patients with VT in the MHV (6 patients).

We hypothesize that thrombosis of the hepatic and/or portal vein branches is due to a combination of trauma during the access of the branch, partial blockage of blood flow by the presence of the metallic mesh and or graft, and/or alterations of the flow dynamics in smaller veins caused by the shunt. A significant portion of the portal segments in this study had attenuated flow with lower contrast enhancement than other segments. Trauma and graft occlusion are known mechanisms that cause thrombosis. However, it is unclear whether decreased attenuation in smaller vessels results in an increased propensity for thrombus formation or if it is a transient phenomenon.

Liver failure is one of the most feared complications after TIPS and the most common cause of early mortality after the procedure. Even some patients with apparently good hepatic reserve may develop this serious complication.^{4,12,13} Liver ischemia is mostly related to acute diversion of portal vein flow created by the TIPS. It has been shown that the flow in the hepatic artery increases to compensate for the altered portal flow.^{14,15} In theory, if major branches of the portal vein thrombose after TIPS, this may worsen liver ischemia. We observed this in the MPV, and it trended close to significance in the RPV. This unusual complication is frequently associated with liver failure and poor outcomes.^{12,13,16} The pathological process of liver infarction after TIPS is not definitively known but is thought to be multifactorial, with VT as a potential factor. Disruption of the dual blood supply of the liver and trauma related to the procedure have been hypothesized. Hepatic intraparenchymal trauma related to TIPS creation and arterial or venous complications may result in ischemia post-procedure with evolution to liver infarction. Multiple mechanisms, including direct injury to the arterial branches by needle puncture, extrinsic compression from the adjacent TIPS stent graft, and systemic hemodynamic changes (hypotension)^{13,16–18} have been discussed. Furthermore, infarction related to thrombosis of the portal system and hepatic vein has been reported.^{12,19–21} Post-TIPS liver infarction was found to be correlated with segmental perfusion defects associated with the presence of a venous thrombus in the portal and hepatic veins.⁷ The clinical outcome of patients with liver infarction could range from short-lived post-TIPS changes to acute liver failure with resultant mortality. Our study with a larger sample did not find an overall difference in survival with either venous thrombosis or liver infarct. We improved upon prior studies by analyzing a larger sample size that could show worsened mortality with MPV thrombus.

This study has several limitations. One of the main limitations is its retrospective nature. The follow-up CT and MRI scans available for study analysis were obtained at different time intervals and for multiple clinical reasons, including transplant candidacy evaluation, surveillance for liver cancer, and deterioration of liver function, among others. Follow-up images were not obtained at fixed time intervals as part of routine surveillance after TIPS. As happens in other venous segments, VT is probably a dynamic process with thrombosis and recanalization over time. It is possible that some of the portal and/or hepatic vein branches that were thrombosed initially after the procedure were later recanalized, or that patent branches with stagnant flow after TIPS were later thrombosed. Another limitation of this study includes a limited sample size. The presence of Budd-Chiari syndrome, Wilson's disease, and superior mesenteric vein VT was associated with significantly lower 1-year survival and overall survival in the univariate analysis. However, these findings are limited due to a sample size of a single patient in each of those groups. Another limitation is that segments with patent but very slow or stagnant flow were never opacified with contrast and may have been misclassified as thrombosed. Unfortunately, duplex ultrasound scans were not performed at the time of the contrast-enhanced scans in most patients.

In conclusion, segmental portal and hepatic VT is a common imaging finding after TIPS without worsened clinical outcomes, unless VT involves the MPV. VT can lead to liver infarction, but infarcts were not independently associated with poorer survival.

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Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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