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Research Article

ASSESSMENT OF EFFECT OF ANTICOAGULATION FOR PORTAL VEINRECANALIZATION IN NON-TUMORAL PORTAL VEIN THROMBOSIS IN CIRRHOSIS

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A B S T R A C T

Background and Aims: Portal vein thrombosis is common consequence of cirrhosis and portal hypertension. Recent studies have showed that prophylactic anticoagulation prevented portal vein thrombosis (PVT) and decreased episodes of decompensation of cirrhosis. We aim to assess effect of anticoagulation for portal vein recanalization in non-tumoral portal vein thrombosis with cirrhosis and its effect on prognosis.

Methods: 45 patients of cirrhosis with nontumoral PVT were included in study. PVT was diagnosed by PV Doppler study. Decision to start anticoagulation was taken at the discretion of the clinician managing the patient. The effect of anticoagulation on PVT recanalization was analyzed.

Results: The mean age was 52.8± 9.26 years and 26 (57.8%) were males. Severity of cirrhosis was assessed by Child–Pugh (CP) score & MELD score. Anticoagulation (LMWH–9, heparin–16) was administered in 30/45(66.7%) patients. 24/30 (80%) attained recanalization (Total–16, partial – 8) of the portal vein. By Cox regression analysis, factors associated with mortality at the end of follow-up were: Age (HR 0.021, 95% C.I. 0.943–1.106, p= 0.608), CP score (HR-2.305, 95% C.I. 0.007–1.487, p= 0.095), MELD score (HR 0.582, 95% C.I. 0.352–9.102, p= 0.483), bilirubin (HR -0.362, C.I. 0.175-2.777, p=0.608). Portal vein recanalization was more frequent in patients on anticoagulation than no anticoagulation (80% vs. 13.3%) (p= 0.005).

Conclusions: Anticoagulation in patients with cirrhosis and PVT appears to be safe and associated with higher portal vein recanalization rates and significantly lower mortality.

INTRODUCTION

Portal vein thrombosis (PVT) is a common event in patients with cirrhosis. Routine evaluations of the portal vascular system are performed semiannually in many patients with cirrhosis as they undergo hepatocellular carcinoma screening with Doppler ultrasound. Because the sensitivity and specificity of ultrasound, contrast enhanced computed tomography (CECT) and magnetic resonance imaging (MRI) vary for PVT detection, incidence and prevalence estimates for PVT in the cirrhosis population vary widely based on the imaging modality used for diagnosis. Epidemiologic and cross sectional studies estimate the true prevalence of nonmalignant main trunk PVT to be between 2 and 8 percent in patients with various stages of cirrhosis. [1-7]Cross sectional and epidemiologic data support the intuitive argument that PVTs which are completely occlusive and have greater extension (*i.e.*, mesenteric involvement) are associated with higher morbidity, increased technical difficulty during liver transplantation, and increased mortality after liver transplantation.[8,9] More intriguingly, a highly criticized but randomized controlled trial in patients at high risk for PVT using enoxaparin *in prevention* of PVT over the course of a year showed that low molecular weight heparin (LMWH) was not only highlyeffective at preventing the formation of PVT in the treatment arm but also resulted in reduced rates of decompensation and improved all cause survival in that group.[10]

In cirrhosis patients, literature suggests that more than 70% of partial or nonocclusive PVTs spontaneously resolve when initially discovered on Doppler ultrasonography. [11]This high rate of spontaneous resolution is likely due to the inherent weaknesses in Doppler techniques. Unlike the deep veins in the extremities, the deep veins of the abdomen are not externally compressible; thus, differentiating true thrombosis versus sluggish flow is difficult. For this reason, before therapeutic decisions can be made regarding PVT, diagnosis must be verified by contrast enhanced imaging such as CT or MRI. Importantly, malignant tumor thrombus, a common finding with hepatocellular carcinoma (HCC), does not

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respond to antihemostatic medications and HCC with tumor thrombus should be diagnosed and treated differently than bland, nonmalignant PVT. Once the diagnosis of nonmalignant PVT is confirmed, it is reasonable to decide on the treatment strategies and urgency based on the extent of thrombosis and presentation.

Currently, the optimal management of PVT in cirrhosis remains unclear and no definitive recommendations have been reported in clinical guidelines or consensus conferences. The therapeutic strategies available are anticoagulation therapy and, in some technically suitable patients, Transjugular intrahepatic portosystemic shunt (TIPS). Although anticoagulation therapy is associated with a high rate of recanalization, the indications for treating PVT in cirrhotic patients are still not strictly defined and neither is the optimal treatment duration. Accordingly, not all PVT patients have been treated with anticoagulants in the past decade [12]. In fact, patients with cirrhosis are considered at risk of bleeding events due to the common findings of a low platelet count and prolonged prothrombin time,although the quantification of this risk does not appear to be well defined. The safety of anticoagulation in cirrhosis is still a matter of debate. At the same time, the pro-thrombotic potential of cirrhotic patients is often underestimated and, in the clinical practice, it is difficult to assess where, on the bleed/clot spectrum a patient is at any time [13]. We have used this gray area of clinical management to collect cirrhotic patients with non-tumoral PVT and to compare those who were treated with anticoagulants to those who were left untreated.

The aim of this study was toprospectively assess effect of anticoagulation for portal vein recanalization in non-tumoral portal vein thrombosis with cirrhosis.

PATIENTS AND METHODS

All patients of cirrhosis irrespective of etiology and PVT, diagnosed by portal vein Doppler or contrast enhanced CT or MRI abdomen were included in study. This study was conducted at Institute of Medical Gastroenterology, RGGGH & Madras Medical College from October 2016 to September 2018.

The Exclusion Criteria were: Patients lost to follow up (<3 months), intra-abdominal malignancy including hepatocellular carcinoma (HCC) at the time of the diagnosis of the PVT and patients already on anticoagulants due to other reasons; anticoagulants were not given to recurrent variceal bleed, multiple collaterals and cavernous transformation, underlying kidney disease, frequent encephalopathy and critically ill cases, unwillingness for anticoagulation.

Clinical, epidemiologic, laboratory and radiologic data were collected at the time of the diagnosis of the PVT.Decision to start anticoagulation was taken at the discretion of the clinician managing the patient. Anticoagulation was given as LMWH/heparin, then converted to warfarin once INR reaches 2-3. Anticoagulation was given upto 6 months.Follow-up of anticoagulated cases were done on monthly basis by portal venous (PV) Doppler and laboratory parameters till 6 months and then 3 monthly upto end of study period (September 2018) or death. 3 monthly follow up was done for those who were not anticoagulated. The study was approved by the Ethics Committee and all participants had given written informed consent.

Statistical Analysis

Continuous variables are expressed as mean ± standard deviation (SD) and categorical variables as numbers and frequencies. Variable distribution was assessed by the Kolmogorov–Smirnov test, and continuous variables were compared using analysis of variance (ANOVA). Categorical variables were compared using the $\chi 2$ test with a Yates' correction. Survival was calculated as the time from the PVT diagnosis to death or the last follow-up visit (censoring events) and was expressed as medians and 95% confidence intervals (95% CIs). Survival curves were generated by using the Kaplan–Meier method and were compared with the log rank test.Cox univariate analysis was carried out to assess the degree of association between survival and the above-mentioned variables. Variables associated ($p \leq 0.10$) with survival at the univariate analysis were tested using the Cox multivariate regression model. The hazard ratio (HR) and 95% CI were calculated for independent predictors of survival.

Endpoints: The primary endpoints of this study were to explore the safety ofanticoagulant treatment of PVT in terms of the rate and patternof the bleeding events, and its efficacy, in terms of rate of PVTrecanalization, in aprospectivecohort of cirrhotic patients. The secondary endpoints were to explore the rate of PVT, recurrence after recanalization and to identify the clinical factors significantly influencing survival.

RESULTS

Total60 patients were identified to have PV thrombosis in the enrollment period, out of which 15 were excluded from the study because of underlying intra-abdominal malignancy or short period of follow up (< 3months). Thus, 45 cirrhotic patients with PVT were finally included in the analysis.

The median follow-up was 21.7±4.98months (range 8–24) after PVT detection. The main characteristics of the study populationat baseline are shown in Table 1.Alcohol related cirrhosis is the most common cause 20 (44.4%), followed by cryptogenic cirrhosis 10 (22.2%).Table 2, shows etiological profile of study population with relation to treated and untreated group. The Child–Turcotte–Pugh (CTP) class was significantly better in the treated than in the untreated group (CTP-B 46.6 vs. 40%, $p = 0.074$; CTP-C 13 vs 60% $p = 0.016$). The Model for End-Stage Liver Disease (MELD) score was also lower in the treated group (MELD 14.7 vs. 19.07%, $p = 0.001$).Table 3 shows extent of portal vein thrombosis at diagnosis & treatment status. Main trunk of portal vein was involved in 44/45(97.7%) cases. Partial thrombosis was noted in 20(44.4%) and complete thrombosis in 25(56.6%) cases. Treatment was given to 18/20 (90%) cases of partial thrombosis and 12/24(50%) in complete thrombosis. Cavernoma was present in 9 cases, none of them were given treatment.

Anticoagulant therapy was administered to 30 patients (66.6%). 19 (63.3%) received unfractionated heparin, 11(36.6%) patients were given LMWH initially. All 30 treated cases were switched over to warfarin for 6 months when INR of 2-3 was achieved. Prior to anticoagulation, all patients had received an esophagogastroduodenoscopy for variceal screening. 13/45(28.8%) patients had large varices; variceal banding was done prophylactically till eradication of large varices in both treated and untreated patients. Anticoagulation therapy was given after banding in treated group.

Table 4 shows various outcomes in treated vs nontreated group. 24/30 (80%) attained recanalization of the portal vein in anticoagulation group, after median period of 4 months (range 2- 8 months). In 16/24 (66.6%) complete recanalization & 8/24(33.3%) partial recanalization was achieved. Spontaneous recanalization occurred in 2/15(13.3%) patients those who were not anticoagulated. The recanalization rate was significantly higher in the anticoagulation group than in the untreated group (80 vs. 13.3 % $p < 0.00002$) (Table 4). The rates of partial and complete recanalization were both higher in treated than in untreated patients (Table 4). Multivariate analysis revealed no predictive factors of recanalization in treated patients (data not shown). Recanalisation status after treatment in various CTP class is shown in Table 5. In CTP class B patients 13/14(92.9%) attains recanalization in treated group, whereas 1(7.1%) in untreated group, Similarly in CTP class A 8/12(66.7%) achieved recanalization in treated group. In class C although study sample is less but 75% cases attained recanalization in treated group.

The recurrence of PVT in recanalized group was seen in 11/24(45.8%), and the median time from the cessation of anticoagulation to recurrence was 4.2 months (range: 1.4 - 7.0 months). 2/6 (33.3%) who did not achieve recanalization at the time of anticoagulation discontinuation showed additional progression of the PVT after treatment discontinuation.

During the follow-up, 26.6% untreated patients presentedwith events of bleeding, whereas 20% in treated group (Table 4). In all cases, the bleeding events were relatedto portal hypertension, supposedly aggravated by PVT. Among these cases 2 had variceal bleeding, 1 had severe portal hypertensive gastropathy (PHG). 2 out of 4 patients had a concurrent progressionof the thrombosis seen in portal vein Doppler study. Bleeding events were reported in 6/24 (25%) in treated group.4 related to portal hypertension (2 variceal bleeding, 1 PHG and 1 due to GAVE), and 2 probably favored by anticoagulant treatment (following trauma or accidental falls). There were no significant differences in the rate of bleeding complications between the two groups ($p = 0.612$).The mean overall survival (Fig. **1**) in treated group was 22.8 months and for the untreated group 19.4 months. Kaplan–Meier curve analysis revealed higher cumulative survival in the treated group than in the untreated group ($p = 0.005$). Univariate analysis showed that age, anticoagulant treatment, liver function (expressed in CTP classes) and MELD score at the diagnosis of PVT were associated with survival.

Multivariate analyses carried out to identify factors predictive of survival in treated patients and in the subgroup of the treated patients who experienced progression or relapse of PVT after stopping anticoagulant treatment did not identify any significant factor.Finally, a subgroup analysis of the treatment outcomes according to the CTP class revealed significantly shorter survival and higher rates of bleeding along with the progression of the CTP class with the worst outcomes in CTP-C patients, as expected according to the natural history of the disease. The rate of recurrence after treatment discontinuation was lower in CPT-B than CPT-A class, although without reaching the statistical significance.

Table 1 Baseline characteristics of study population

Variables	Treated (30)	Untreated (15)	Total (45)	P value
Age (Years) (Mean, SD)	47.97 ± 8.79	62.47 ± 10.842	52.8± 9.26	.568
Male gender (N, %)	19 (63.3)	7 (46.7)	26 (57.8%)	.286
CTP Class				.001
A(N, %)	12 (40%)	0%	12 (26.7%)	.074
B (N, %)	14 (46.7%)	6 (40%)	20 (44.4%)	.166
C(N, %)	4 (13.3%)	9 (60%)	13 (28.9%)	
MELD Score (Mean, SD)	14.70±2.654	19.07 ± 3.751	16.156± 2.94	.186
Bilirubin(mg/dL) (mean, SD)	4.220±2.0179	4.753±2.4092	4.398± 2.725	.406
INR(mean, SD)	1.20 ± .188	1.783±.307	1.402± .246	.235
Platelet-mean,(SD)	125.87 (63.74)	65.13 (26.18)	105.622(42.63)	.000
Varices (N, %)	16 (53.3%)	13 (86.7%)	29 (64.4%)	.028
Follow-up period(months)	22.80±3.727	19.47± 6.424	21.7 ± 4.98	.033
Therapy duration (months)	6	0		

Table 2 Diagnosis & Treatment status

Diagnosis	No. of patients (%)		Total
	Treated	Non treated	
ALD	15 (50.0%)	2 (13.3%)	17 (37.8%)
ALD &HCV	0%	1 (6.7%)	1 (2.2%)
ALD&HBV	0%	2 (13.3%)	2 (4.4%)
AUTOIMMUNE	1 (3.3%)	0%	1 (2.2%)
Cryptogenic	2 (6.7%)	8 (53.3%)	10 (22.2%)
HBV	2 (6.7%)	0%	2 (4.4%)
HCV	2 (6.7%)	0%	2 (4.4%)
HBV & HCV	0%	1 (6.7%)	1 (2.2%)
NASH	8 (26.7%)	1 (6.7%)	9 (20.0%)

Table 3 Extent of portal vein thrombosis at diagnosis & Treatment status

Extent of portal vein thrombosis at diagnosis	No. of patients (%)		Total
	Treated	Non treated	
Both main trunk and branches	10(33.3%)	3(20%)	13(28.9%)
Main trunk with SMV	4(13.3%)	0%	4(8.9%)
Main trunk with splenic vein	10(33.3%)	3(20%)	13(28.9%)
Only main trunk	5 (16.7%)	9 (60%)	14 (31.1%)
Only intrahepatic branches	1 (3.3%)	0%	1 (2.2%)
Cavernoma formation	0%	9 (60%)	9 (20%)
Present	30 (100%)	6 (40%)	36(80%)
Absent			
Thrombosis		2 (13.3%)	
Partial	18 (60%)	13 (86.7%)	20 (44.4%)
Complete	12 (40%)		25 (56.6%)

Table 4 Various outcome in treatment Vs. Non- treated group

Variables	Treated (30)	Untreated (15)	Total (45)	p-value
Recanalization				
Partial	8 (26.7%)	0(.0%)	8 (17.8%)	
Complete	16 (53.3%)	2 (13.3%)	18 (40.0%)	.000
No	6 (20.0%)	13 (86.7%)	19 (42.2%)	
Recurrence		0 %		
Present	11 (45.8%)	2 (100%)	11 (42.3%)	.207
absent	13 (54.2%)		15 (57.7%)	
Bleed	6 (20%)	4 (26.7%)	10 (22.2%)	.612
Survival time in months (mean, SD)	22.800 ± .669	19.467 ± 1.731	21.689 ± .774	.005
Death	3 (10%)	7 (46.7%)	10 (22.2%)	.005

Table 5 Recanalisation status after treatment in various CTP class

1Treatment status				Recanalisation status		p-value
				Yes	No	
Treated	CTP class	A	Count (%)	8 (66.7%)	4 (33.3%)	
		B	Count (%)	13 (92.2%)	1 (7.1%)	.24
		C	Count (%)	3 (75%)	1 (25%)	
	Total		Count (%)	24 (80%)	6 (20%)	
Non treated	CTP class	B	Count (%)	0 %	6 (100%)	.215

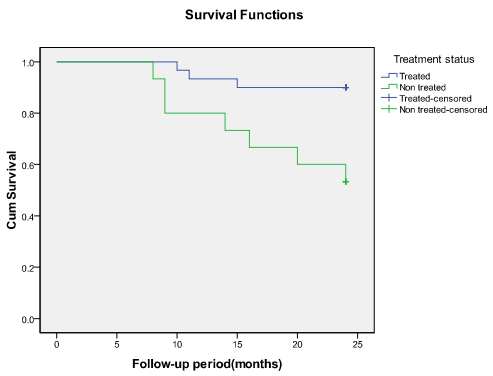

Fig 1 Survival analysis according to treatment allocation

DISCUSSION

Portal vein thrombosis is a frequent complication in liver cirrhosis but its natural history as well as therapeutic management have not yet been clearly addressed by either international guidelines or consensus conferences. The present study confirmed that recanalization of PVT may occur spontaneously, but it is significantly favored by anticoagulant treatment. Of note, limited to the study observation timeframe, anticoagulation was not associated with an increased risk in major bleeding complications and it positively influenced survival.

The rate of spontaneous partial or complete recanalization of PVT rates ranged from 5 to 48% in the literature [14, 15], the wide range due to heterogeneity in terms of thrombus extension, study populations and imaging methods. In a study by Luca *et al*. involving 42 patients with cirrhotic PVT, a spontaneous reduction in thrombosis occurred in 47.6% of those with partial thrombosis [14] while Senzolo *et al*. reported a spontaneous rate of improvement in thrombosis in 5% [15]. In the present study, 13.3% of untreated subjects showed spontaneous recanalization.

The rates of recanalization under anticoagulant therapy in the present study (80 %) were in keeping with those reported to date (37–93%) [16] and in particular with a recent metanalysis by Loffredo *et al*. [17]. After the discontinuation of therapy, 11/24(45.8%) of our patients who had a partial or a complete recanalization subsequently reported a recurrence/progression of PVT. These results are in line with those reported in the literature [18, 19] and to those of Delgado *et al* [18] who showed recurrence or progression of PVT in approximately one third of patients who discontinued treatment. Complete recanalization was achieved in 18/30 (60%) patients after a median of 4 months after starting anticoagulant therapy. Partial recanalization was achieved in 6/30 (20%) patients after a median of 4 months of therapy. As described in an observational study [19], where full recanalization was reached in33% and partially in 50% of patients after 6 months of anticoagulant therapy, continued treatment might perhaps subsequently allow complete recanalization. Therefore, the continuation of anticoagulant therapy for at least 6 months could have a role in the secondary prophylaxis of PVT recurrence after having achieved recanalization. Whether similar results of secondary prophylaxis could be achieved with lower doses of anticoagulants remains to be explored.Nevertheless, in our study progression of the thrombosis occured in 2/30 (6.6%) in treatment group and 5/15(33.3%) in non-treated group, which is similar to recent metanalysis of Loffredo *et al*. [17] which showed a 9% rate of progression during therapy (vs. 33% of untreated patients). Further studies are needed to clarify whether these are truly refractory patients or simply sub-maximally treated patients.

Currently, due to risk of acute variceal hemorrhage in portal hypertension andan increased risk of bleeding due to the compromised coagulation function, fear for the use of anticoagulation exists in liver disease. The present study has shown that individualized anticoagulant therapy in cirrhotic patients with PVT is safe as bleeding rate is similar in treated and untreated patients, and the bleeding complications are mainly due to portal hypertension rather than anticoagulant therapy itself. In the present study, 6/24(25%) treated patients had hemorrhagic complications, but only 2 were likely related to the anticoagulant treatment. It is important to mention that all patients with high-risk varices at the time of the diagnosis of PVT underwent prophylactic therapy (variceal band ligation and/or beta-blockers) before initiating anticoagulant therapy. The rate of hemorrhagic complications associated with therapy for PVT reaches 18% in the literature [17, 18]. The bleeding rate seems to correlate with severe thrombocytopenia and the use of VKAs (vitamin K antagonists)[18]. No such correlation emerged from our data whereas nearly all bleeding events were related to portal hypertension and the incidence rates were similar in both the treated (25%) and the untreated (26.6%) groups, hence without any additional hazard related to anticoagulation.

Our data also shows that rates of recanalization is higher in CTP-B (92.9%) than CTP-A (66.7%) patients. Apparently CPT-B patients were the category benefitting most of the anticoagulation, showing a lower rate of recurrence after stopping treatment and longer survival than the untreated subjects, in keeping with a previous prospective study which showed survival benefit with enoxaparin in such patients [20].
In conclusion, anticoagulant therapy appeared to be effective in cirrhotic patients with PVT, reaching recanalization rates of 80%, more than doubling the spontaneous recanalization rates. Recanalization was commonly obtained in the first 6 months after the start of treatment. Prior to initiating treatment, either beta-blocker therapy was started or prophylactic endoscopic band ligation of high-risk varices was carried out to protect from the risk of variceal bleeding during treatment. Finally, anticoagulant treatment significantly improved the survival of cirrhotic patients with PVT. However, although the treatment response occurred mainly within 6 months, precocious discontinuation for the presumed lack of treatment response should be avoided. In patients who responded to anticoagulation treatment, its discontinuation was associated with a high risk of PVT recurrence. For this reason, it could be speculated that some patients might benefit from a secondary prophylactic strategy which should be maintained after recanalization. Finally, anticoagulant treatment in cirrhotic patients with PVT was shown to be safe in the short/mid-term. These findings highlighted the need for randomized prospective trials to test the safety and efficacy of a long-term secondary prophylaxis with anticoagulants for PVT in cirrhosis.

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None,
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