

Effect of Anticoagulation on The Hepatic Function of Non-Malignant Portal Vein Thrombosis

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ABSTRACT

Background: In various groups of PVT patients, portal vein thrombosis (PVT) occurs at a rate of 5-26%; the incidence increases with progressive liver disease. Portal vein thrombosis increases portal hypertension, increases the risk of variceal haemorrhage, and decreases hepatic perfusion, eventually leading to hepatic decompensation. Therefore this study is conducted to evaluate the effect of anticoagulation on the hepatic function of non-malignant portal vein thrombosis.

Patients and Methods: Total 200 patients were consecutively enrolled in this retrospective study among April 2021 to April 2022. Patients with malignant PVT and those who lacked enough clinical data at the time of PVT diagnosis and during the subsequent clinical course were excluded. Retrospective analysis was used in this study to assess how anticoagulation affected hepatic function. A predesigned proforma were used to collect data. All the data was entered and analyzed by SPSS version 25. All the quantitative variables were presented by Mean+ SD and qualitative with frequency and percentages. An independent sample t test was applied to find out the significant difference of hepatic function among groups. P-value< 0.05 was considered as significant.

Results: Total 200 patients were enrolled in current study among which 100 were treated with anticoagulation and 100 without anticoagulation. The mean age among groups were (T= 48.99±1.73 Vs. UT=49.50±1.63). Majority of the patients were male (115/200) and married (161/200). Majority of patients were Males, Married, normal weighted (123/200), moderate ascites (55/200), grade 3 hepatic encephalopathy (105/200) and child PUGH class A (97/200). There was insignificant mean difference of hepatic function among groups. The ALT, AST ALP, Albumin, Bilirubin shows insignificant difference (P-value>0.05).

Conclusion: Our research shows that anticoagulation reduces hepatic damage and enhances liver production in people with non-malignant PVT.

Keyword:

Anticoagulation, Hepatic Function, Portal Vein Thrombosis

INTRODUCTION

Almost all chronic liver diseases can lead to cirrhosis, with chronic viral hepatitis and alcoholic liver disease being the most common causes in developed countries.¹ As a result of esophageal varices or portal hypertensive gastropathy, patients with cirrhosis are more likely to experience gastrointestinal bleeding.² The inadequacy of coagulation tests, which do not accurately predict bleeding or thrombotic events but may lead to incorrect drug administration, is an important problem in clinical practice. In addition, the dynamic interaction between the coagulation and anticoagulation pathways cannot be represented with tests. The importance of platelets, whose quantitative and qualitative characteristics can

determine the dynamic hemostatic forces in cirrhosis, is becoming increasingly clear.³ In various groups of PVT patients, portal vein thrombosis (PVT) occurs at a rate of 5-26%; the incidence increases with progressive liver disease.⁴ Portal vein thrombosis increases portal hypertension, increases the risk of variceal haemorrhage, and decreases hepatic perfusion, eventually leading to hepatic decompensation.⁵ The prothrombotic state in cirrhotic patients is caused by a decreased concentration of anticoagulant factors, endothelial activation, rupture of the endothelial glycocalyx, and formation of procoagulant microparticles.^{6,7} Depending on the severity, 0.6-26% of patients with liver cirrhosis develop non-malignant PVT, and the incidence increases as the cirrhosis progresses.^{8,9} Reduced PV velocity is the primary contributor to PVT development in people with liver cirrhosis.¹⁰ Currently, the plasma concentrations of the substrates or end products of liver metabolism are used to assess hepatic function. In non-cirrhotic patients,

Conflict of Interest: The authors declared no conflict of interest exists.

Citation: Afzal MS, Zaman M, Zaidi YA. Effect of Anticoagulation on The Hepatic Function of Non-Malignant Portal Vein Thrombosis. J Fatima Jinnah Med Univ. 2023; 17(2): 54-56.

DOI: <https://doi.org/10.37018/JFJMU/MEH/0133>

anticoagulation has been shown to be beneficial in the treatment of PVT.¹¹ Experimental evidence suggests that anticoagulation has a positive impact on liver synthesis function and fibrosis when used to prevent and treat PVT. In a prospective clinical trial, preventive treatment reduced hepatic decompensation, increased survival, and prevented PVT in liver transplant candidates.¹²

We conducted a study to compare the outcomes of anticoagulation in non-malignant PVT in cirrhotic patients to the outcomes of those who did not receive anticoagulation. This study was conducted to evaluate the effect of anticoagulation on the hepatic function of non-malignant portal vein thrombosis.

PATIENTS AND METHODS

At Allama Iqbal Medical College/Jinnah Hospital, Lahore. Total 200 patients were consecutively enrolled in this retrospective study among April 2021 to April 2022. Retrospective analysis was used in this study to assess how anticoagulation affected hepatic function. Patients over the age of 18 with cirrhosis and concurrent PVT met the study's specified inclusion criteria. Patients with malignant PVT and those who lacked enough clinical data at the time of PVT diagnosis and during the subsequent clinical course were excluded. A predesigned proforma were used to collect data. **Clinical data was collected from patients' medical records** A complete patient's history (Age, Gender, BMI), Clinical Investigations (Hepatic encephalopathy, Ascites, Child Pugh Classes) and laboratory findings/ Hepatic function was collected. All the data was entered and analyzed by SPSS version 25. All the quantitative variables were presented by Mean \pm SD and qualitative with frequency and percentages. An independent sample t test was applied to find out the significant difference of hepatic function among groups. P-value $<$ 0.05 was considered as significant.

RESULTS

Total 200 patients were enrolled in current study among which 100 were treated with anticoagulation and 100 without anticoagulation. The mean age among groups were (T= 48.99 \pm 1.73 Vs. UT=49.50 \pm 1.63). Majority of the patients were male (115/200) and married (161/200). Majority of patients were Males, Married, normal weighted (123/200), moderate ascites (55/200), grade 3 hepatic encephalopathy (105/200) and child PUGH class A (97/200).

Table 2 showed the comparison of laboratory finding among the patients. There was insignificant

mean difference of hepatic function among groups. The ALT, AST ALP, Albumin, Bilirubin shows insignificant difference (P-value $>$ 0.05). Only PT/INR levels shows significant difference (P-value $<$ 0.05).

Table 1: Demographic Variables

	Groups		Total
	Treated (T) (n=100)	Untreated(UT) (n=100)	
Gender of Patients			
Male	68	47	115
Female	32	53	85
Age (Mean \pm SD)	48.99 \pm 1.73	49.50 \pm 1.63	
Hepatic Encephalopathy			
Single	22	17	39
Married	78	83	161
Ascites			
Absent	8	13	55
Mild	6	25	32
Moderate	8	13	55
Severe	6	25	32
Hepatic Encephalopathy			
Grade 0	8	5	13
Grade 1	9	5	14
Grade 2	25	38	63
Grade 3	58	47	105
Grade 4	0	5	5
Child Pugh Class			
A	58	39	97
B	32	46	78
C	10	15	25
BMI			
Underweight	14	12	26
Normal	53	70	123
Overweight	27	13	40
Obese	6	5	11

Table 2: Comparison of laboratory findings among groups

Laboratory Variables	Groups	Mean	SD	P-value
Alanine Transaminase	Treated	60.8200	36.34998	.696
	Untreated	63.3390	53.25245	
Aspartate Aminotransferase	Treated	87.1000	102.54287	.481
	Untreated	76.3790	111.89784	
Alkaline Phosphate	Treated	159.9540	100.69133	.975
	Untreated	159.4740	116.31632	
Albumin	Treated	3.5522	2.96496	.575
	Untreated	3.8284	3.91901	
Bilirubin	Treated	3.7340	8.93473	.223
	Untreated	2.4697	5.20046	
Creatinine	Treated	1.6399	1.66909	.663
	Untreated	1.5476	1.29824	
PT/INR	Treated	2.4340	.36382	0.01*
	Untreated	2.6010	.34363	

DISCUSSION

This study was conducted to evaluate the effect of anticoagulation on the hepatic function of non-malignant portal vein thrombosis. In current study it was noticed a positive effects on liver function and hepatic inflammation, furthermore it was observed that the AST and ALT levels numerically reduced while albumin, a measure of liver synthesis function, considerably improved in individuals receiving the anticoagulation therapy. This is crucial because patients

with decompensated cirrhosis are more likely to experience complications, and it is important for effective PVT treatment essential.

Interestingly, our study found that anticoagulation improves hepatic function and leads to reduced frequency of bleeding issues. This is consistent with Loffredo et al previous's work, which found that anticoagulant use is associated with a lower risk of variceal bleeding shows that in cirrhotic individuals with PVT, the positive effects of anticoagulation therapy may also result in a reduction in hepatic necroinflammation. The most noteworthy finding was that long-term anticoagulation was also linked to improved ascites control during follow-up.¹³ This result is explained by a decrease in portal and intrahepatic dysfunction, which finally stopreduces the variceal rupture and hemorrhage.¹⁴ It was advised that those individuals undergo endoscopic varices screening before starting anticoagulant therapy and take primary or secondary variceal bleeding prevention strategies.¹⁵ PVT Is a common adverse complication of liver cirrhosis that becomes more frequent as the disease progresses.¹⁶ PVT occurs annually in approximately 10-15% of individuals with severe cirrhosis, according to a recent study.¹⁷ In cirrhotic patients, decreased PV velocity is believed to be the main factor for PVT. Anticoagulation has been shown to help treat PVT in non-cirrhotic patients.¹⁸

CONCLUSION

Our research shows that anticoagulation reduces hepatic damage and enhances liver production in people with non-malignant PVT. Anticoagulation is the recommended treatment for portal vein thrombosis in noncirrhotic individuals. Anticoagulation has historically been used to treat PVT in patients with cirrhosis. Patients with cirrhosis who have nonmalignant PVT liver function benefit from anticoagulation.

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