



Case Series Psychiatry

Archives of Biological Psychiatry



Thrombocytopenia in alcohol use disorder: An essential differential

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Received: 13 October 2024 Accepted: 13 February 2025 Published: 15 March 2025

DOI 10.25259/ABP_14_2024

Quick Response Code:



ABSTRACT

Despite the significant disease burden associated with alcohol use, research on thrombocytopenia (TP) in this context remains limited. This case series explores the relationship between chronic alcohol consumption and TP, a hematological abnormality characterized by low platelet counts, in four male patients. Each patient presented a history of excessive alcohol intake and concomitant TP, which resolved following abstinence. Initial platelet counts varied from 30,000 to $55,000/\mu$ L but showed a marked increase within days of withdrawal. Notably, our findings indicate that TP is transient in nature and does not correlate with liver damage or the severity of alcohol withdrawal. This highlights the importance of monitoring platelet counts in patients with a history of alcohol use and emphasizes the need for awareness of the hematological implications of alcoholism.

Keywords: Alcohol use disorders, Alcoholism, Alcohol withdrawal, Platelet count, Thrombocytopenia

INTRODUCTION

Alcohol consumption accounts for approximately 5.1% of the global disease burden.^[1] While not widely discussed, hematological abnormalities like thrombocytopenia (TP), characterized by platelet count below 150,000/ μ L,^[2] are prevalent in alcoholism and serve as biomarkers. In India, few cross-sectional studies have explored this relationship, focusing on changes in platelet counts rather than TP itself.^[3-6] Understanding this link is crucial in regions like Uttarakhand, where vector-borne diseases such as dengue fever are common, and TP is a frequent hemostatic defect. This case series examines the relationship between chronic alcohol consumption and TP by presenting four patients encountered over the past 6 months. These patients had excessive alcohol use history and concomitant TP, which were resolved after they abstained from alcohol.

CASE 1

A 30-year-old male with an 8-year history of consuming 360 mL of Indian-made foreign liquor (IMFL) daily, increasing to 540 mL in the past 4–5 months, had his last drink 3 days before admission. His platelet count was $55,000/\mu$ L on the 3rd day of withdrawal, with subsequent counts of $30,000/\mu$ L, $45,000/\mu$ L, and $60,000/\mu$ L.

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CASE 2

A 36-year-old male, drinking an average of 10 standard drinks/day for 10 years, last consumption 2 days before admission. His initial platelets were $30,000/\mu$ L, with follow-up counts of $43,000/\mu$ L, $58,000/\mu$ L, and $63,000/\mu$ L.

CASE 3

A 44-year-old male presented with incoherent speech, hallucinations, and disrupted sleep after consuming 560–750 mL of IMFL daily for 15 years. His last intake was 1 day before admission. Platelet counts over 4 days were 41,000/ μ L, 52,000/ μ L, 57,000/ μ L, and 65,000/ μ L.

CASE 4

A 27-year-old male with a 7-year history of consuming 560 mL of IMFL last drank 1 day before presenting with tremors and convulsions. His platelet counts were $53,000/\mu$ L, $62,000/\mu$ L, and $68,000/\mu$ L over 3 days and 74,000/ μ L at discharge [Table 1].

Table 1 and Figure 1 show the baseline and last-day Serum glutamate oxaloacetate transaminase (SGOT), Serum glutamate pyruvate transaminase (SGPT), Gamma-glutamyl transferase (GGT), Clinical institute withdrawal assessment for alcohol, Revised (CIWA-Ar) scores of all four cases. Electrocardiogram (ECG) of all four patients was normal. The mean age of the sample is 34.25 (standard deviation [SD] = 7.500). The mean of platelet counts immediately post-admission and at discharge is 44750.00 (SD = 11615.363) and 66250.00 (SD = 5251.984), respectively, with a 23% increase within 4–5 days. The mean days of lowest platelet count post-admission was 2.25 (SD = 1.258). Complicated withdrawal was reported in two

patients. However, no significant correlation was found between the baseline platelets, final platelet counts before discharge, baseline and final liver enzymes, withdrawal scores, and the presence of complicated withdrawal.

DISCUSSION

This case series aims to elucidate the transient nature of TP during excess alcohol use. Typically, early remission of alcohol-induced TP occurs without significant consequences, as seen in our cases.^[2] Platelet counts stabilized within a week, with a 30% increase within 5 days from the lowest point.^[2] Interestingly, TP did not correlate with liver damage, as confirmed by our findings.^[2] Contrary to reports associating TP (<119,000/µL) with complicated withdrawal, we found no such correlation. The cause of alcohol-related TP is complex. Potential mechanisms include myelosuppression and platelet presence in the bloodstream, with platelet poisoning being suspected.^[5] Alcohol also negatively impacts platelet functionality and size, which is crucial for coagulation issues.^[6] The severity of TP determines management. Anticoagulants and antiplatelet medications may be necessary if bleeding occurs. However, those with mild-to-moderate TP, as in our cases, might not need thorough evaluation. Monitoring TP after alcohol cessation is appropriate.^[7] In addition, obtaining a history of alcohol use is crucial, as it may be concealed due to stigma and fear of insurance non-compliance. TP in alcohol use disorder (AUD) can arise from factors beyond direct alcohol toxicity, including liver cirrhosis with portal hypertension and splenomegaly, folic acid deficiency, viral infections, and alcohol-induced pancreatitis. Understanding TP in AUD is limited by overlapping mechanisms, variability in severity despite similar alcohol intake, uncertain recovery



Figure 1: Survey plots of platelet counts of 4 cases (immediately post admission and pre-discharge (5th day)). Blue color indicates: Area under the graph

Table 1: Sociodemographic and clinical characteristics of patients with alcohol withdrawal (n=4).							
Case No.	Age (years)	Family history	Past history	Physical comorbidity	Day of withdrawal	Day of admission	Platelet Count (mm ³)
1	30	+	+	+	3 rd	1^{st}	55,000
					4^{th}	2 nd	32,000
					5 th	3 rd	45,000
					6 th	$4^{ m th}$	63,000
2	36	-	-	-	2^{nd}	1^{st}	30,000
					$3^{\rm rd}$	2^{nd}	43,000
					4^{th}	$3^{\rm rd}$	58,000
					5 th	$4^{ m th}$	63,000
3	44	+	+	+	2^{nd}	1^{st}	41,000
-					3 rd	2 nd	52,000
					4 th	3 rd	57,000
					5 th	4^{th}	65.000
4	27	+	-	-	1 st	1 st	53.000
-	27	·			2 nd	2 nd	62,000
					3 rd	3 rd	68.000
					$4^{ m th}$	$4^{ m th}$	74,000
Case No.	Day of withdrawal with least platelet count	SGOT	SGPT	GGT	CIWA-Ar	Withdrawal seizures	Withdrawal delirium
1	$4^{ m th}$	250 U/L	212 U/L	385 U/L	15 (on day of	-	-
		(on day of	(on day of	(on day of	admission)		
		admission)	admission)	admission)	,		
		180 U/L	145 U/L	275 U/L	2 (on day of		
		(on day of	(on day of	(on day of	discharge)		
		discharge)	discharge)	discharge)	0,		
2	2 nd	287 U/L	226 U/L	412 U/L	14 (on dav of	-	-
_		(on day of	(on day of	(on day of	admission)		
		admission)	admission)	admission)	···· ,		
		195 U/L	147 U/L	330 U/L	0 (on day of		
		(on day of	(on day of	(on day of	discharge)		
		discharge)	discharge)	discharge)	C C		
3	2^{nd}	293 U/L	245 U/L	430 U/L	30 (on day of	-	+
		(on day of	(on day of	(on day of	admission)		
		admission)	admission)	admission)			
		205 U/L	159 U/L	353 U/L	6 (on day of		
		(on day of	(on day of	(on day of	discharge)		
		discharge)	discharge)	discharge)	-		
4	1^{st}	223 U/L	202 U/L	368 U/L	25 (on day of	+	-
		(on day of	(on day of	(on day of	admission)		
		admission)	admission)	admission)			
		185 U/L	146 U/L	287 U/L	3 (on day of		
		(on day of	(on day of	(on day of	discharge)		
		discharge)	discharge)	discharge)	-		
SGOT: Serum glutamate oxaloacetate transaminase, SGPT: Serum glutamate pyruvate transaminase, GGT: Gamma-glutamyl transferase, CIWA-Ar:							

Clinical Institute withdrawal assessment for alcohol (revised), +: Presence, -: Absence, U/L: Units per liter

rates after cessation, and a bias toward liver disease. While research identifies key pathways, gaps in its pathophysiology and management remain. Future studies and integrated care are crucial to improving outcomes in AUD. The absence of information regarding the possible history of past hematological abnormalities represents a limitation of the study, as such factors could potentially confound the observed findings. The unavailability of PT values is another limitation of this study, as their inclusion could have provided additional insights into the findings.^[8,9]

CONCLUSION

In conclusion, this case series demonstrates that thrombocytopenia (TP) associated with chronic alcohol use and withdrawal is typically transient, with platelet counts normalizing within a week. TP was not linked to liver damage or complicated withdrawal, highlighting the need for careful monitoring and a thorough alcohol use history. Future studies should explore the underlying mechanisms of alcohol-induced TP, the long-term effects of alcohol cessation on platelet counts, and potential correlations between TP and other withdrawal-related complications.

Ethical approval: Institutional Review Board approval is not required.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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How to cite this article: Bhandari S, Chowdhry S, Garg S, Dhyani M, Bhatia A. Thrombocytopenia in alcohol use disorder: An essential differential. Arch Biol Psychiatry. 2024;2:63-6. doi: 10.25259/ABP_14_2024