RESEARCH ARTICLE

Thrombocytopenia in Alcohol Dependence Syndrome: A Hospital-based Observational Study

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Abstract

Aim and background: Thrombocytopenia is a condition characterized by a decrease in platelet counts less than 1,50,000/µL. Multiple studies have elicited the effect of alcohol on various blood parameters but its effect on platelets is often overlooked. We intend to estimate the prevalence of thrombocytopenia in alcohol dependence syndrome (ADS), to compare the platelet count at baseline with that of the 7th day of admission, to evaluate the association between platelet count with the severity of ADS, and co-morbidities in patients with ADS.

Materials and methods: This is an observational study conducted in a tertiary care Medical College Hospital. We recruited 64 inpatients qualifying for ADS whose last intake of alcohol was within five days before admission. Severity of alcohol dependence questionnaire (SADQ) and Clinical Institute Withdrawal Assessment for Alcohol (CIWA-Ar) scale were administered, platelet count along with CBC was assessed during baseline, and only platelet count was assessed on the 7th day of admission. Statistical analysis paired sample *t*-test, ANOVA, Pearson's correlation were used.

Results: The prevalence of thrombocytopenia at baseline was 18.8% which dropped to 9.4% on day 7 of admission following abstinence from alcohol. At baseline patients with liver disease had significantly lower platelet count than those without liver disease (Mean: 1,93,900/µL vs 2,40,500/µL, *p*: 0.031) but no statistically significant difference on 7th day (*p*: 0.8). Baseline platelet count showed negative correlation with age, duration of alcohol use.

Conclusion: The prevalence of thrombocytopenia is high in patients with ADS.

Clinical significance: Abstinence from alcohol can lead to normalization of platelet count even in patients with co-morbid liver disease.

Keywords: Addiction, Alcohol dependence, Platelet count, Substance use, Thrombocytopenia.

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Introduction

Platelet is considered to be very crucial for the normal functioning of the human body and plays a key role in various processes like inflammation, thrombosis, hemostasis, and wound healing. Balance of platelet creation, maintenance in circulation, and final clearance from the blood are required to ensure optimal functioning of the human body. Thrombocytopenia is a medical condition characterized by a decrease in the platelet count to less than 1,50,000 per microliter (Normal 1,50,000–4,00,000 per microliter). The risks associated with thrombocytopenia range from no risk at all to bleeding risks and thrombosis. The correlation between the severity of thrombocytopenia and bleeding risk is uncertain. Spontaneous bleeding can occur with a platelet count under 10,000/µL and surgical bleeding with counts below 50,000/µL.[1] Multiple studies have elicited the effect of alcohol on various blood parameters including hemoglobin, various enzymes of liver function tests (LFT) and components of renal function tests (RFT), but the effect of alcohol on platelets in particular is often the most overlooked parameter. Even though thrombocytopenia in people using alcohol has been reported since the 1960s, there are only a handful of studies that have tried eliciting the phenomenon of thrombocytopenia due to alcohol use, and mostly they were in the form of case reports and case series while some other had relatively smaller sample.[2,3] In a study done in 1968 on a group of 43 heavy alcoholics at the beginning of hospitalization, 15 (34.9%) patients were noted to have a drop in platelet count below 1,00,00/µL whereas another 20 (46.5%) patient's platelet count ranged between 1,00,000 and 1,50,000/µL, only 8 patients had their platelet count within normal range, i.e., (>1,50,000/µL).[4] Most of the earlier studies were retrospective and were done on the western population who were termed as "alcoholics, alcohol abusers, A lot of drinkers, chronic alcohol users" etc. who may or may not be qualifying for alcohol dependence syndrome (ADS). A prospective Indian study evaluated the pattern of platelet count among 40 patients during the course of alcohol withdrawal and its relationship with liver enzymes.[5] In that study, they excluded patients with withdrawal seizures; they also did not include common medical co-morbidities like pancreatitis and liver diseases. Very few studies were conducted on patients diagnosed with alcohol dependence syndrome and took into consideration other factors such as duration of alcohol intake, the severity of alcohol dependence, and other common co-morbid medical conditions. The number of studies available in this area is scarce, especially in the Indian population. We aim to study the impact of alcohol on platelet count and to evaluate the

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association between platelet count and various clinical variables in patients with ADS. The primary objective is to estimate the prevalence of thrombocytopenia in patients with ADS. Secondary objectives are to compare the platelet count at baseline with that of the 7th day of admission, to evaluate the association between platelet count and the severity of ADS, and to determine the effect of common co-morbidities on platelet count in patients with ADS.

Materials and Methods

This is a prospective observational study done in a tertiary care medical college hospital in South India. The study was conducted after getting the Institute's human ethical committee approval (IHEC/2021/Appr/FB/030; Ref project no: 21/120). We screened all consecutive patients admitted to the de-addiction ward between September 2021 and November 2022. We included all patients aged between 18 and 65 years of age fulfilling the criteria for alcohol dependence syndrome according to ICD 10 diagnostic criteria for research (DCR) whose last intake of alcohol is within five days before admission in our de-addiction ward. We excluded patients with other substance use except for nicotine and patients who were on chronic use of drugs that can potentially affect platelets (Valproate, Vancomycin, H2 Blockers, and Proton Pump Inhibitors). Patients who were already diagnosed with serious hematological diseases or had confirmed coagulation disorders (Idiopathic Thrombocytopenic Purpura, Thrombotic Thrombocytopenic Purpura), Infectious diseases (dengue, leptospirosis), and carcinomas were excluded from the study.

All patients who fulfilled our inclusion and exclusion criteria were recruited for the study after taking written informed consent. In the case of patients who were not in a condition to give consent due to delirium or other complications, consent was initially taken from their family members after explaining the study protocol to them, and later consent was taken from the patients after they had recovered. After recovery, if a patient did not consent then they were removed from the study. Patient's sociodemographic details; relevant clinical details like medical history, surgical history, and medication details; and details related to alcohol use like frequency of alcohol intake, duration of alcohol intake, the quantity of alcohol intake, details of last drink, history of abstinence etcetera were collected using a semi-structured proforma. The (CIWA-Ar) scale was used to assess withdrawal symptoms. The severity of alcohol dependence was assessed using the SADQ. Blood was collected on the day of admission to assess baseline platelet count, along with other routine investigations like complete blood count (CBC), liver function test (LFT), renal function test (RFT), and random blood sugar (RBS) were carried out. The platelet count alone was checked again on the 7th day of admission. For analyzing blood count, 2 mL of venous blood was collected in an ethylene-diamine tetra-acetic acid vacutainer, CBC was analyzed on a coulter LH780 hematology analyzer within 2 hours of blood collection. Patients were treated with lorazepam or chlordiazepoxide if required, and all patients received thiamine and multivitamin supplementation. Gastroenterologists, physicians, and other specialists' opinions were sought if necessary. Patients with co-morbid liver or pancreatic diseases were diagnosed and treated as per the advice of Gastroenterologists. Mostly alcohol-induced liver disease patients received T.Ursodeoxycholic acid and pancreatitis patients received symptomatic treatment like painkillers and antibiotics which did not have any significant effect on platelet count.

We carried out Statistical Analysis using the software IBMt SPSS Statistics for Windows, Version 23.0 (Armonk, NY: IBM Corp). For descriptive data frequency analysis, percentage analysis was used for categorical variables and Mean and Standard Deviations were used for continuous variables. To find the significant difference between the bivariate samples in paired groups the paired sample *t*-test was used and for independent groups, the independent sample *t*-test was used. For the multivariate analysis, the one-way ANOVA with Turkey's *post hoc* test was used. To assess the relationship between the variables Pearson's Correlation was used. In all the above statistical tools the probability value 0.05 is considered a significant level.

Results

A total of 80 ADS patients were screened, out of which 6 patients wanted to get discharged early, 6 patients have done blood investigation outside recently, 3 patients were not willing for investigation, 1 patient was not interested to participate in the study, and finally, we were able to recruit 64 male patients for our study. Age distribution of the study population, among 64 patients 11 (17.2%) belonged to the age group 21–30 years; 19 (29.7%) between 31 and 40 years; 22 (34.4%) between 41 and 50 years; 10 patients (15.6%) between to 51 and 60 years of age; and only 2 patients (3.1%) were above 60 years of age. The mean duration of alcohol intake in our sample was 16.5 years (ranging from 2 years to 40 years). Out of 64 patients, 20 were mildly dependent; 20 were moderately dependent; and 24 patients were severely dependent on alcohol. Co-morbid liver and pancreatic diseases were found in 31 and 7 patients respectively. The prevalence of thrombocytopenia at baseline was 18.8% (12/64) which dropped to 9.4% (6/64) on day 7 of admission. The range of platelet count at baseline was 58,200/µL–5,16,000/µL. The mean platelet count at baseline was 2,17,900/µL and on the 7th day it was 2,78,400/µL this difference in mean platelet count is statistically very significant ($p < 0.01$).

Out of 64 patients, 31 (48.4%) had co-morbid alcoholic hepatitis, 3 patients were also diagnosed to have cirrhosis of the liver; 1 patient had de-compensated liver disease with grade 2 hepatic encephalopathy. In our sample 23 patients had raised total bilirubin levels (levels in our sample ranged from 0.3 mg/dL to 13.1 mg/dL), 26 patients had more than twice the normal value (5–38 U/L) of SGOT (levels in our sample ranged from 23 U/L to 264 U/L), and 23 patients had more than twice the normal value (5–41 U/L) of SGPT (levels in our sample ranged from 14 U/L to 440 U/L). At baseline patients with liver disease had significantly lower platelet count than those without liver disease (Mean platelet count 1,93,900/µL vs 2,40,500/µL, *p*: 0.031).

Whereas by day 7 there is no significant difference in the platelet count between the groups due to the rapid rise in platelet count in patients with liver disease (Mean 2,81,800/µL vs 2,75,200/µL, *p*: 0.80) (Table 1). Among 64 patients 7 (10.9%) patients had co-morbid pancreatic disease (4 patients had acute pancreatitis; 3 patients had chronic pancreatitis with atrophic pancreas). There was no statistically significant difference in platelet count between those with and without pancreatic disease either at baseline or by day 7 (*p*: 0.44 and 0.92 respectively). About 33 patients had co-morbid nicotine dependence. There was no statistically significant difference in platelet count between those with and without nicotine dependence either at baseline or at day 7 (*p*: 0.77 and 0.42 respectively) (Table 1).

Table 1: Platelet count among patients with and without co-morbidity

Co-morbidity	*Time of assessment*	*N (Total 64)*		*Mean (cells × 1000/μL)*	*SD*	*t-value*	*p-value*
Liver disease	Baseline	Present	31	193.9	79.9	2.205	0.03*
		Absent	33	240.5	88.5		
	Day 7	Present	31	281.8	120.8	0.245	0.80
		Absent	33	275.2	90.5		
Pancreatic disease	Baseline	Present	7	193.7	76.3	0.778	0.44
		Absent	57	220.9	88.4		
	Day 7	Present	7	274.3	113.9	0.106	0.91
		Absent	57	278.9	107.7		
Nicotine dependence	Baseline	Present	33	214.7	64.6	0.229	0.76
		Absent	31	221.4	106.9		
	Day 7	Present	33	267.8	94.7	0.808	0.42
		Absent	31	289.6	120.1		

*p-value < 0.05 is significant

Table 2: Comparison of platelet count at baseline and by day 7 based on the severity of alcohol dependence syndrome assessed using SADQ

Platelet count	*Severity of dependence*	*N (Total 64)*	*Mean (cells × 1000/μL)*	*SD*	*f-value*	*p-value*
Baseline	Mild	20	238.1	57	3.006	0.06
	Moderate	20	237.8	122.8		
	Severe	24	184.5	61.1		
Day 7	Mild	20	285.8	93.8	0.075	0.92
	Moderate	20	272.6	104.5		
	Severe	24	277.1	123.5		

*p-value < 0.05 is significant

The platelet count at baseline was low in patients with severe alcohol dependence compared to those with mild or moderate alcohol dependence but this difference was not statistically significant (Mean value was 2,38,100/μL, 2,37,800/μL, and 1,84,500/μL in mild, moderate, and severe alcohol dependence patients respectively; *p*: 0.057). After a period of abstinence of at least a week, there is a recovery in platelet count in all three groups but the rate of recovery was very drastic in the severe alcohol dependence group compared to the other two (Mean value was 2,85,800/μL, 2,72,600/μL, and 2,77,100/μL in mild, moderate, and severe alcohol dependence patients respectively; *p*: 0.92) (Table 2). Hemoglobin (Hb) and red blood cells (RBC) were assessed only at baseline; the ANOVA test showed a significant difference in Hb and RBC levels among patients with different severity in ADS (*p*-value: 0.013 and < 0.01 respectively), following which *post hoc* Tukey HSD test was used to measure the difference between groups. Both hemoglobin and red blood cells were significantly low in patients with severe alcohol dependence compared to those with mild alcohol dependence (*p*: 0.013 and 0.002 for Hb and RBC respectively) (Table 3).

Baseline platelet count showed a negative correlation with age, duration of alcohol use, total bilirubin (TB), direct bilirubin (DB), indirect bilirubin (IB), and SGOT which was statistically significant at *p*-value < 0.01. Whereas Hemoglobin level and total RBC count showed a positive correlation with baseline platelet count which was statistically significant as well with a *p*-value < 0.01 (Table 4).

After a period of abstinence for 7–10 days, i.e., platelet count done on day 7 of admission showed no significant positive or negative correlation with any of the above factors.

Table 3: Severity of alcohol dependence and hemoglobin, RBC levels

Post hoc Tests–Tukey HSD-Multiple comparisons					
				95% Confidence interval	
Dependent variable	*Mean difference (I–J)*	*Std. error*	*p-value*	*Lower bound*	*Upper bound*
Hemoglobin					
Mild					
Moderate	0.5846	0.5357	0.523	–0.706	1.875
Severe	1.5148*	0.5067	0.011*	–0.294	2.735
Moderate					
Severe	0.9302	0.4985	0.158	–0.271	2.131
RBC					
Mild					
Moderate	0.66173	0.30206	0.082	–0.0659	1.3893
Severe	1.08205*	0.28567	0.001*	0.3939	1.7702
Moderate					
Severe	0.42031	0.28107	0.301	–0.2567	1.0973

*Significant at *p*-value < 0.05

Discussion

The phenomenon of thrombocytopenia due to alcohol use is under-described and under-researched and there is a need for further studies.[6] This prospective observational study is one of

Table 4: Correlation of baseline and day 7 platelet count with age, duration of alcohol intake, and liver function test

	Pearson's correlation r / *Significance (2 tailed) p*		
Variables	*N*	*Baseline platelet count*	*Day 7 platelet count*
Age	*r*	−0.34	−0.04
	P	<0.01	0.75
	N	64	64
Duration of alcohol use	*r*	−0.34	−0.05
	P	<0.01	0.67
	N	64	64
Hemoglobin	*r*	0.38	−0.02
	P	<0.01	0.91
	N	58	58
Total RBC	*r*	0.35	−0.13
	P	<0.01	0.34
	N	58	58
Total bilirubin	*r*	−0.37	−0.18
	P	<0.01	0.17
	N	64	64
Direct bilirubin	*r*	−0.33	−0.12
	P	<0.01	0.35
	N	64	64
Indirect bilirubin	*r*	−0.44	−0.17
	P	<0.01	0.17
	N	64	64
SGOT	*r*	−0.36	0.17
	P	<0.01	0.19
	N	64	64
SGPT	*r*	−0.22	0.23
	P	0.19	0.06
	N	64	64
ALP	*r*	−0.19	−0.13
	P	0.13	0.31
	N	64	64
GGT	*r*	−0.19	0.08
	P	0.14	0.52
	N	62	62

ALP, alkaline phosphatase; GGT, gamma glutamyl transferase; SGOT, serum glutamic oxaloacetic transaminase; SGPT, serum glutamate pyruvate transaminase; RBC, red blood cell; Significance at p-value < 0.05.

the very few studies that systematically evaluated the association between alcohol dependence and platelet count. As we commonly encounter in our clinical practice, all the patients were male and most of them were between 30 and 50 years of age.

In the present study, the prevalence of thrombocytopenia in ADS is 18.8% which is very high compared to the prevalence of thrombocytopenia in the general population in India (5–12%).[7] The prevalence in our study is much less than that of the Poland study which reported 46% of their subjects admitted for detoxification had thrombocytopenia.[8] Another retrospective cohort study done on 334 patients, showed a prevalence of 32% with patients who had complicated withdrawal having a higher proportion of thrombocytopenia compared to uncomplicated withdrawal patients.[9] In the present study as the patient remained abstinent from alcohol, the platelet count increased which is evident by a drop in the prevalence of thrombocytopenia to 9.4% on the 7th day of admission, and this increase in count is noted even in patients who did not have thrombocytopenia at baseline. This finding is in line with a previous Indian study that assessed the patterns of platelet count in 40 ADS patients and found that platelet count increased gradually from baseline till the 10th day of alcohol withdrawal.[5] In this study, we found that people who are old and those who were consuming alcohol for a long duration are at more risk of having a lower platelet count or developing thrombocytopenia compared to those who are young and were not consuming alcohol for a very long time. This finding is in contrast to that of a previous Indian study which did not find any significant correlation between platelet count and age or duration of alcohol dependence.[5] However, in our study, we did not find any significant correlation between these factors and platelet count on the 7th day of admission suggesting

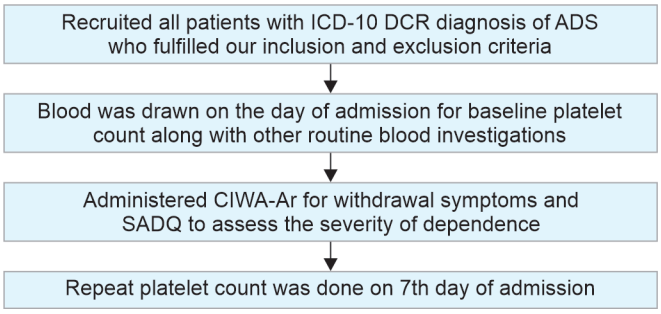

Fig. 1: Flowchart of study design

that even in aged and chronic alcohol users' platelet count rises after a period of abstinence. But whether this rate of recovery in platelet count is comparable between old and young individuals; and between chronic users and people who had a short duration of alcohol use needs to be studied further (Fig. 1).

In our study, the platelet count is lower in patients with severe ADS compared to patients with mild or moderate ADS, with a trend toward significance (*p*: 0.057). However, this trend was not seen when the platelet count was repeated on the 7th day of admission due to a rise in platelet count following abstinence. It is also worth noting that the rate of recovery is much more rapid in the severe ADS group and the platelet count is almost similar to the mild to moderate ADS group. This finding is similar to another study that showed platelet count during abstinence from alcohol is not affected by the quantity of daily intake of absolute alcohol.[5]

Thrombocytopenia is the most common hematologic abnormality seen in liver disease and is multifactorial. The most notable contributor is portal hypertension, which leads to congestive splenomegaly and the sequestration of platelets in the spleen.[10] We found that co-morbid liver disease patients had significantly lower platelet count compared to those without liver disease at the time of admission, with a significant negative correlation with Total bilirubin; direct and indirect bilirubin; and serum glutamic oxeloacetic transaminase (SGOT). But neither liver disease nor any of the liver enzymes had any effect on reverse thrombocytosis seen in patients during abstinence from alcohol.

Studies done on smokers have shown nicotine can significantly increase platelet count, platelet crit, and platelet distribution width.[11] Nicotine dependence syndrome as a variable in itself in patients with ADS may have a direct effect by increasing platelet adhesiveness.[12] However, in this study, we did not find any significant effect of nicotine dependence syndrome or co-morbid pancreatitis on platelet count either at baseline or on day 7.

The mechanisms of alcohol-induced hematotoxicity are unclear and it is likely to be complex.[7,13] Alcohol can indirectly affect folic acid and vitamin B12 deficiency, or cirrhosis of the liver leading to splenomegaly and sequestration of cells in the spleen.[14–16] But earlier studies have pointed out that the reduction in the platelets in alcohol users may have a different origin than in the case of megaloblastic anemia.[4,17,18] In alcohol-related thrombocytopenia two possible mechanisms are hypothesized: Myelosuppression and the direct toxic effect of alcohol on platelets circulating in the bloodstream (Platelet apoptosis).[7,13,19–21] When we look at the overall findings of the current study both hemoglobin and RBC levels had a positive correlation with baseline platelet count and were significantly low in patients with severe ADS; the presence of co-morbid liver disease though affected the baseline platelet count did not have any influence on recovery; severe dependence patients had relatively lower baseline platelet count but the rate of recovery is much rapid in them than the mild or moderate ADS patients; all these points towards myelosuppression being the main reason for thrombocytopenia in ADS. The more severe the alcohol use the more severe the bone marrow suppression which in turn leads to a stronger rebound phenomenon (Reverse thrombocytosis) in them once alcohol is stopped. However, trying to explain or understand the mechanism behind alcohol-induced thrombocytopenia is beyond the scope of this study.

Some potential limitations of this study are the small sample size is a major limitation we could not recruit more patients due to the drastic reduction in admissions in general due to the COVID pandemic and also because of our stringent inclusion and exclusion criteria, we only excluded patients with known medical or surgical illness and could not exclude all conditions that might affect platelet count, we assessed only the quantity of platelet and not the quality or platelet functioning, we did not assess the general nutritional status of the patient, we only quantitatively assessed the severity of dependence using SADQ and found platelet count was low in patients with severe dependence but severity of dependence is also based on various psychosocial factors, having a control arm without alcohol use and with or without liver disease would have added more credibility to the study, and tests like bone marrow analysis would have also helped us in understanding the mechanism of alcohol-induced thrombocytopenia.

Some of the strengths are this is one among the very few studies, especially in the Indian context to systematically evaluate thrombocytopenia in patients using alcohol, the homogenous sample we recruited only those patients who qualify for alcohol dependence using standard diagnostic guidelines, including patients with common co-morbid conditions, we followed consecutive sampling method to avoid selection bias, we only took Inpatients thereby restricting dropouts and ensuring patients were indeed abstinent between assessment.

Conclusion

The prevalence of thrombocytopenia is high in patients with alcohol dependence syndrome. The severity of alcohol dependence and duration of alcohol intake can lower the platelet count which should be kept in mind even though it may not reach dangerous levels. Abstinence from alcohol can lead to normalization of platelet count even in patients with co-morbid liver disease. Alcohol may have a direct effect on platelets and bone marrow, but to understand the exact mechanism of the impact of alcohol on platelets, we need more systematic studies.

Clinical Significance

In clinical settings often we come across ADS patients with low platelet count, instead of becoming anxious and aggressively evaluating and treating this condition just waiting for a week of abstinence (except in severe cases) and repeating platelet count may be enough.

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References

1. Jinna S, Khandhar PB. Thrombocytopenia 2022; pp. 1. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023.
2. Eichner ER. The hemotologic disorder of alcoholism. Am J Med 1973;54(5):621–630. DOI: 10.1016/0002-9343(73)90120-4.
3. Peltz S. Severe thrombocytopenia secondary to alcohol use. Postgrad Med 1991;89(6):75–85. DOI: 10.1080/00325481.1991.11700918.
4. Lindenbaum J, Hargrove RL. Thrombocytopenia in alcoholics. Ann Intern Med 1968;68(3):526–532. DOI: 10.7326/0003-4819-68-3-526.
5. Harshe DG, Thadasare H, Karia SB, et al. A study of patterns of platelet counts in alcohol withdrawal. Indian J Psychol Med 2017;39(4): 441–444. DOI: 10.4103/0253-7176.211766.
6. Silczuk A, Habrat B. Alcohol-induced thrombocytopenia: Current review. Alcohol 2020;86:9–16. DOI: 10.1016/j.alcohol.2020.02.166.
7. Nisha S, Amita D, Uma S, et al. Prevalence and characterization of thrombocytopenia in pregnancy in Indian women. Indian J Hematol Blood Transfus 2012;28(2):77–81. DOI: 10.1007/s12288-011-0107-x.
8. Silczuk A, Habrat B, Starowicz ML. Thrombocytopenia in patients hospitalized for alcohol withdrawal syndrome and its association to clinical complications. Alcohol and Alcoholism 2019;54(5):503–509. DOI: 10.1093/alcalc/agz061.
9. Berggren U, Fahlke C, Berglund KJ, et al. Thrombocytopenia in early alcohol withdrawal is associated with development of delirium tremens or seizures. Alcohol 2009;44(4):382–386. DOI: 10.1093/alcalc/agp012.
10. Jandl JH, Aster RH, Forkner CE, et al. Splenic pooling and the pathophysiology of hypersplenism. Trans Am Clin Climatol Assoc 1967;78:9–27. PMID: 5339085.
11. Pujani M, Chauhan V, Singh K, et al. The effect and correlation of smoking and platelet indices, neutrophil lymphocyte ratio, and platelet lymphocyte ratio. Hematol Transfus Cell Ther 2021;43(4): 424–429. DOI: 10.1016/j.htct.2020.07.006.
12. Silverstein P. Smoking and wound healing. Am J Med 1992;93 (1A):22S–24S. DOI: 10.1016/0002-9343(92)90623-j.
13. Latvala J, Parkkila S, Niemela O. Excess alcohol consumption is common in patients with cytopenia: Studies in blood and bone marrow cells. Alcohol Clin Exp Res 2004;28(4):619–624. DOI: 10.1097/01.alc.0000122766.54544.3b.
14. Sullivan LW, Herbert V. Suppression of hematopoiesis by ethanol. J Clin Invest 1964;43(11):2048–2062. DOI: 10.1172/JCI105079.
15. Eichner ER, Hillman RS. The evolution of anemia in alcoholic patients. Amer J Med 1971;50(1):218–232. DOI: 10.1016/0002-9343(71)90151-3.
16. Herbert V, Zalusky R, Davidson CS. Correlation of folate deficiency with alcoholism and associated macrocytosis, anemia and liver disease. Ann Intern Med 1963;58:977–988. DOI: 10.7326/0003-4819-58-6-977.
17. Sullivan LW, Herbert V. Suppression hematopoiesis by ethanol. J Clin Invest 1964;43(11):2048–2062. DOI: 10.1172/JC1105079.
18. Post RM, Desforges JF. Thrombocytopenia and alcoholism. Ann Intern Med 1968;68(6):1230–1236. DOI: 10.7326/0003-4819-68-6-1230.
19. Cooper GW, Dinowitz H, Cooper B. The effects of administration of ethyl alcohol to mice on megakaryocyte and platelet development. Thromb Haemost 1984;52(1):11–14. PMID: 6541816.
20. Levine RF, Spivak JL, Meagher RC, et al. Effect of ethanol on thrombopoiesis. Br J Haematol 1986;62(2):345–354. DOI: 10.1111/j.1365-2141.1986.tb02938.x.
21. Smith C, Gasparetto M, Jordan C, et al. The effects of alcohol and alcohol dehydrogenases on disorders of hematopoiesis. Adv Exp Med Biol 2015;815:349–359. DOI: 10.1007/978-3-319-09614-8_20.