International Journal of Research in Medical Sciences
Elanchezhian et al. Int J Res Med Sci. 2017 Nov;5(11):5041-5047
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20174967

Original Research Article

Comparison of haematological parameters between alcoholics and non-alcoholics

Elanchezhian[1], Yoganandh T.[1]*, S. Mayilsamy[2], Shankar Radhakrishnan[3]

[1]Department of Medicine, Government Mohan Kumaramangalam Medical College, Salem, Tamil Nadu, India
[2]Department of Surgeon Govt Hospital, Uthukkuli, Tamil Nadu, India
[3]Department of Preventive Medicine, VMKVMCH, Salem, Tamil Nadu, India

Received: 12 September 2017
Accepted: 09 October 2017

***Correspondence:**
Dr. Yoganandh T.,
E-mail: yoganandt@gmail.com

ABSTRACT

Background: Alcohol being one of the most commonly used drug, whose consequences include changes of CBC. The main causes leading to changes of CBC (complete blood count) are: myelosuppression that is accompanying with slight reduction in all blood cells, blood loss from gastrointestinal tract, malnutrition etc. Alcoholics may suffer from moderate anemia, characterized by enlarged, structurally abnormal RBC's; mildly reduced numbers of WBC's, especially of neutrophils; and moderately to severely reduced numbers of platelets. The objective of the study was to study the haematological manifestation among alcoholics based on the quantity and duration of alcohol intake and compare them with non-alcoholics.

Methods: A cross-sectional study was conducted for a period of one year in our medical college hospital in medicine OPD with the collaboration of department of pathology and bio-chemistry. The study was started after getting the approval from the institutional ethical committee. A total of 150 study subjects were included in the study in which 50 were non-alcoholics, 50 were moderate alcoholics (less than or equal to two drinks per day for men and less than or equal to one drink per day for women) and the remaining 50 were severe alcoholics (more than 7 drinks a week in women and more than 14 drinks in a week in men). Blood investigations such as CBC, prothrombin time, liver function test, renal function test, folic acid levels and vitamin B12 levels were measured and the levels were compared between alcoholics and non-alcoholics.

Results: Mean RBC count, mean MCH, MCHC were normal among the non-alcoholic group and it started decreasing among moderate alcoholics and more so with severe alcoholics and a similar type of result was also seen with total count and platelet count and the difference was found to be statistically significant. LFT, RFT, prothrombin time and vitamin B12 levels were found to be high and folate levels were decreased among alcoholics group when compared to non-alcoholics and the difference was statistically significant.

Conclusions: Detection of hematological changes in chronic alcoholics and giving psychiatric counseling and treatment for alcohol dependence will decrease the future complications like cirrhosis liver, cardiac and renal disease, cerebellar degeneration, neuropathy, pancreatitis, etc. and reduce the morbidity and mortality in alcoholics.

Keywords: Alcoholics, Hematological parameters, Non-alcoholics

INTRODUCTION

The use of alcoholic beverages dates from the beginning of civilization in ancient Egypt. As far as we know the first alcoholic drink was used at least in 6000 BC and people have been drinking alcohol all over the world ever since.[1] Alcoholism is one of the most serious global

public health problem. Regarding disease Burden Alcohol is the world's third largest risk factor.

It is estimated that the total number of the population classified as alcohol consumers in the world goes up to 2 billion, while 76.3 million people develop alcohol use disorder.[2,3] The effects of alcohol depend on the amount of ethanol consumed per kg body weight. Levels from 0:02 to 0:03 g / dl are achieved after consumption of one or two standard drinks.[4] Alcoholism (Alcohol use disorder) is defined as repeated alcohol related difficulties in atleast 2 of 11 life areas that cluster together in the same 12 months period.[5] Lifetime risk for an AUD in most of the western countries is about 10-15% for men and 5-8% for women. Approximately 60% of the risk for AUD is attributed to genes. The factors increasing risk of liver disease in alcoholics are quantity and duration of intake, Sex (Females susceptibility twice as males), co-infection with Hepatitis C, Genetic factors, Malnutrition, Obesity, Smoking and Iron over load. Hence alcohol consumption is known for morbidity and mortality, being a serious health hazard of the world. Many a times haematological changes are left undetected and untreated which could progress to cardiac failure.[6]

Alcohol being one of the most commonly used drug, whose consequences include changes of CBC. Due to the fact, that alcohol use, especially in heavy drinkers, can cause different metabolic derangements, it is necessary, to investigate the changes of complete blood count.[7] Alcohol consuming can cause different adverse effects on blood cells, therefore even in their functions. The main causes leading to changes of CBC (complete blood count) are: myelosuppression that is accompanying with slight reduction in all blood cells, blood loss from gastrointestinal tract, malnutrition etc.[8] Chronic excessive alcohol ingestion reduces the number of blood cell precursors in the bone marrow and causes characteristic structural abnormalities in these cells, resulting in fewer-than-normal or non-functional mature blood cells. As a result, alcoholics may suffer from moderate anemia, characterized by enlarged, structurally abnormal RBC's; mildly reduced numbers of WBC's, especially of neutrophils; and moderately to severely reduced numbers of platelets. Although this generalized reduction in blood cell numbers (i.e., pancytopenia) usually is not progressive or fatal and is reversible with abstinence, complex aberrations of hematopoiesis can develop over time that may cause death.[9] As of today only very few studies had been done in India to compare the hemotological manifestations among alcoholics and non-alcoholics, so this study was undertaken to assess and compare the blood parameters among these two groups, which would help when detected earlier in preventing serious complications due to alcoholics.

The aim of the study was to study the haematological manifestation among alcoholics based on the quantity and duration of alcohol intake and compare them with non-alcoholics.

METHODS

A cross-sectional study was conducted for a period of one year in our medical college hospital in medicine OPD with the collaboration of department of pathology and bio-chemistry. The study was started after getting the approval from the institutional ethical committee. A total of 150 study subjects were included in the study in which 50 were non-alcoholics, 50 were moderate alcoholics (less than or equal to two drinks per day for men and less than or equal to one drink per day for women) and the remaining 50 were severe alcoholics (more than 7 drinks a week in women and more than 14 drinks in a week in men). Patients with history of hematological malignancies, hepatic diseases, chronic illnesses such as diabetes, hypertension, TB etc., and patients taking hepatotoxic drugs were excluded from the study. Informed consent was obtained from all the patients before the start of the study. Patients demographic data, history related to their alcohol habits were recorded, complete physical examination was conducted for all patients and the following hematological parameters were measured for all the subjects included in the study.

- Complete Blood Count (by Automated Counter)
  - Haemoglobin in gm%
  -RBC count in million / cmm
  -PCV in %
  -Total WBC count in cells / cmm
  -MCV in fl
  -MCH in pg
  -MCHC in %
  -Platelet count in lakhs/ cmm
- Microscopic peripheral smear study.
- Prothrombin time in seconds.
- Serum vitamin B12 in pg/ml. (by chemiluminescent micro particles immuno assay)
- Serum Folic Acid in ng/ml (by chemiluminescent immuno assay)
- Liver function tests – Total Bilirubin, Direct and Indirect fraction, SGOT, SGPT, Total protein, Albumin and Alkaline phosphatase.
- Renal function tests – Blood urea, Serum Creatinine

All data were entered and analysed by using SPSS version 21. Mean and SD were derived for all parametric variables. Chi square tests was applied for comparing discrete variables and ANOVA was applied for comparing continuous variables and 'p' value of <0.05 was considered as statistically significant.

RESULTS

Table 1 shows the age wise distribution of the study population. It is seen from the table that majority of the study subjects were in the age group between 30 – 50 years and the mean range was between 40 – 42 years and there was no statistically significant difference in the age group between the three groups. Gender wise distribution of the study subjects was not mentioned as all the

subjects involved in our study were males. Malena and abdominal distension was found to be the most common presenting complain among the study subjects with the history of alcoholism, followed by pedal oedema and jaundice and all these presenting complaints were more common among severe alcoholism than moderate alcoholics and the difference was found to be statistically significant. Presenting complaint related to CNS (altered sensorium) was found to be more common among severe alcoholic patients and the difference was found to statistically significant (Table 2).

Table 1: Age wise distribution of the study population.

Age group	Non-alcoholics (n=50)	Moderate alcoholics (n=50)	Severe alcoholics (n=50)	P value
21 – 30	4 (8%)	5 (10%)	4 (8%)	0.384
31 – 40	14 (28%)	16 (32%)	15 (30%)	
41 – 50	17 (34%)	14 (28%)	13 (26%)	
51 – 60	10 (20%)	11 (22%)	12 (24%)	
>60	5 (10%)	4 (8%)	6 (12%)	
Mean ± SD	41.5±7.8	39.8±8.2	40.8±6.8	

P value derived by applying chi-square test

Table 2: Distribution of the study subjects based on their presenting complaints among alcoholics.

Presenting complaint	Moderate alcoholics (n=50)	Severe alcoholics (n=50)	P value
Jaundice	5 (10%)	12 (24%)	<.001
Pedal edema	7 (14%)	13 (26%)	<.001
Haematemesis	3 (6%)	7 (14%)	<.001
Malena	8 (16%)	17 (34%)	<.001
Abdominal distension	10 (20%)	15 (30%)	<.001
Altered sensorium	2 (4%)	10 (20%)	<.001

P value derived by applying chi-square test

Among the various clinical signs elicited among the alcoholic patients pallor and abdominal distension was found to be more common in severe alcoholic patients and signs of liver cell failure was also present in 25% of the patients in severe alcohol group and when compared with the moderate alcohol group the difference was found to be statistically significant (Table 3).

Table 3: Distribution of various clinical manifestations among alcoholics.

Clinical features	Moderate alcoholics (n=50)	Severe alcoholics (n=50)	P value
Pallor	10 (20%)	15 (30%)	0.003
Icterus	4 (8%)	9 (18%)	<.001
Pedal edema	6 (12%)	13 (26%)	<.001
Ascites	8 (16%)	14 (28%)	<.001
Other signs of liver cell failure	6 (12%)	13 (26%)	<.001

P value derived by applying chi-square test

The complete blood parameters were compared between the three groups among non-alcoholics, moderate alcoholics and severe alcoholics. It is seen from table 4 that Hb%, Mean RBC count, mean MCH, MCHC were normal among the non-alcoholic group and it started decreasing among moderate alcoholics and more so with severe alcoholics and a similar type of result was also seen with total count and platelet count and the difference was found to be statistically significant (Table 4). MCV was found to be very high in severe alcoholics when compared to moderate and non-alcoholics and the difference was found to be statistically significant (Table 4).

The liver function test comparison between the three groups shows that total bilirubin along with direct and indirect bilirubin high in severe alcohol group in comparison with non-alcoholics and moderate alcoholics and the difference was found to be statistically significant and similarly the same type of result was also seen with SGOT, SGPT and alkaline phosphatase levels, whereas the total protein and albumin levels were lower in severe alcoholics when compared to moderate and non-

alcoholics and the difference was found to be statistically significant (p<.01) (Table 5).

Table 4: Comparison of means of the complete blood parameters among the study population.

Blood parameter	Study group	Mean	SD	ANOVA	p
Hb	NA	11.12	2.24	9.30	< 0.001
	Moderate	9.33	1.20		
	Severe	9.37	2.30		
RBC	NA	3.90	0.58	15.04	< 0.001
	Moderate	3.26	0.74		
	Severe	3.13	0.68		
PCV	NA	38.72	2.78	52.50	< 0.001
	Moderate	33.55	4.26		
	Severe	28.30	6.14		
MCV	NA	89.56	3.84	6.24	0.003
	Moderate	85.60	8.35		
	Severe	93.44	12.21		
MCH	NA	30.54	2.27	37.23	< 0.001
	Moderate	25.48	1.62		
	Severe	23.91	3.18		
MCHC	NA	36.18	1.92	35.74	< 0.001
	Moderate	32.57	1.94		
	Severe	30.24	2.00		
T.C	NA	8296.00	1762.30	8.83	< 0.001
	Moderate	7872.00	3537.72		
	Severe	6272.00	4286.17		
Platelet	NA	2.50	0.37	39.93	< 0.001
	Moderate	1.63	0.66		
	Severe	1.47	0.67		

NA – Non-alcoholics

Table 5: Comparison of means of the liver function test parameters among the study population.

Liver function test	Study group	Mean	SD	Anova	P
T. Bilirubin	Na	0.97	0.20	23.50	< 0.001
	Moderate	2.52	2.38		
	Severe	3.95	2.73		
D. Bilirubin	Na	0.32	0.08	23.95	< 0.001
	Moderate	0.86	0.77		
	Severe	1.28	0.87		
Indirect bilirubin	Na	0.65	0.16	22.93	< 0.001
	Moderate	1.65	1.61		
	Severe	2.67	1.88		
T. Protein	Na	7.44	0.46	30.60	< 0.001
	Moderate	6.68	0.39		
	Severe	5.92	0.37		
Albumin	Na	4.44	0.51	67.44	< 0.001
	Moderate	3.55	0.31		
	Severe	3.40	0.28		
SGOT	Na	20.16	4.11	61.38	< 0.001
	Moderate	67.40	27.45		
	Severe	94.44	50.51		
SGPT	Na	27.40	5.08	47.76	< 0.001
	Moderate	48.84	15.33		
	Severe	57.44	21.27		
Alk. Phosphatase	Na	51.92	10.47	2.53	0.085
	Moderate	48.16	8.28		
	Severe	54.52	10.89		

NA – Non-alcoholics

Table 6: Comparison of means of the renal function test parameters among the study population.

Renal function test	Study group	Mean	SD	ANOVA	p
Blood Urea	NA	27.00	6.49	150.98	< 0.001
	Moderate	79.68	17.88		
	Severe	95.68	29.49		
	Total	57.34	35.65		
Serum Creatinine	NA	0.94	0.77	28.16	< 0.001
	Moderate	2.04	0.88		
	Severe	3.06	1.90		
	Total	1.75	1.47		

NA – Non-alcoholics

Table 7: Comparison of means of prothrombin time, vitamin B12 and folic acid levels among the study subjects.

Blood parameter	Study group	Mean	SD	ANOVA	p
P.T	NA	14.08	0.83	149.16	< 0.001
	Moderate	25.76	6.41		
	Severe	31.35	5.39		
Vit. B12	NA	605.12	132.53	47.77	< 0.001
	Moderate	713.80	374.14		
	Severe	1375.60	510.03		
Folic acid	NA	12.18	1.72	91.17	< 0.001
	Moderate	8.71	1.00		
	Severe	8.08	1.01		

The renal function parameters particularly the blood urea and serum creatinine levels were found to be very high among the severe alcoholics whereas it was within normal limits among non-alcoholics and slightly elevate in the moderate alcoholic group and this difference was found to be statistically significant ($p<.05$) (Table 6). Prothrombin time was found to be grossly elevated in severe alcoholic group in comparison with moderate or non-alcoholic group, similarly vitamin B12 levels were also found to be very high among severe alcoholics, whereas folic acid was found to be deficit in severe and moderate alcoholics in comparison with non-alcoholics and these differences were found to be statistically significant ($p<.05$) (Table 7).

DISCUSSION

Alcohol abuse is a growing epidemic in India, especially among men and now a day it is becoming a major problem among young adults. The clinical manifestations of alcohol-induced hematologic disorders are profoundly influenced by the patient's social and economic status, and the presence or absence of other factors, such nutritional deficiency or alcoholic cirrhosis. Most of these changes result, either directly or indirectly, in anemia and when extensive liver disease is present, the patient may develop an abnormally functioning fibrinogen or other coagulation disorders, which may initiate or exacerbate bleeding. Studies had shown that even before anemia appears, approximately 90 percent of alcoholics have a macrocytosis (mean corpuscular volume [MCV] between 100 to 110 femtoliters [fL]) and it was almost in par with our study where we found mean MCV was 93.44 fl among severe alcoholics and it was very high in comparison with moderate or non-alcoholics.[10-12] Alcohol-induced macrocytosis occurs even though patients are folate and cobalamin replete and do not have liver disease. The mechanism is unknown, but it takes two to four months for the macrocytosis to disappear after the patient becomes abstinent.

Changes of RBC from chronic and heavy drinking have been studied in many respects, not only regarding to changes of the size of RBC(macrocytosis), but even the presence of defectuose RBC in the blood and their production from the bone marrow.[7] As a result of these changes, anemia is a common finding in alcoholics.[7] The above statement was very much supported by our study in which we found the mean Hb among the severe alcoholics was 9.33 gm% which was significantly lessin comparison with the non-alcoholic group. Anemia was found in approximately 50% of the alcohol abusers in a study performed in Finland.[13] The same study revealed that elevated MCV and MCH was a common finding among alcohol abusers both in absence, or in presence of anemia.[13] In our study we found a statistically significant decrease of WBC count among the severe alcoholics in comparison with non-alcoholics and a similar result were also shown by Esmeralda Thoma et al.[14]

This substantiates that alcohol abusers are at high risk to develop various diseases, including infections, or cancer, though the exact mechanism how alcohol is related to cancer it is not yet well established. Our study further proves that thrombocytopenia is common among severe alcoholics as the mean platelet count was found to be lower than the non-alcoholics and it was almost in par with the studies done by Esmeralda Thoma et al and in another study done in in Kebbi State in Nigeria.[14-20] A case-control study performed in Nigeria had observed significant reduction of WBC, RBC, haemoglobin, haematocrit and platelet count, while MCV values are significantly elevated.[21] Another study done in India had shown a significant reduction of haemoglobin, RBC, WBC, haematocrit and PLT, while MCV and MCH were significantly elevated.[22] Thus the complete blood count picture had shown that anemia, leucopenia and thrombocytopenia are common abnormalities that are associated with alcohol abuse with respect to time of alcohol abuse and the quantity of alcohol consumed.[21,22] These findings suggest that alcohol abuse can cause bone marrow suppression, or ethanol has cytotoxic effects.

As the previous studies [23-25] quoted, the common clinical manifestations of alcoholism were jaundice, pallor, anorexia, fever, abdominal distension, altered sensorium and in our study the patients with moderate to severe alcoholism had almost the similar complaints.

Total bilirubin abnormalities in chronic alcoholics may result from isolated or combined increases of the non-conjugated and conjugated fractions. An increase in the conjugated fraction always denotes hepatocyte damage.[26] Elevation of the non-conjugated fraction may result from various causes, including increased absorption from enterohepatic pools for reasons that are not entirely clear.[27,28] Our study shows a statistically significant increase of bilirubin among the severe alcoholics and serum bilirubin levels were best correlated with the severity of alcoholic hepatitis. Prevalence of undernutrition in alcoholics with alcoholic hepatitis due to both protein and caloric deficiency resulting from low ingestion and inadequate utilization of nutrients absorbed and in the present study we found serum albumin and total proteins level was significantly lower among the severe alcoholics.[29] The fact that the test for aminotransferases (AST) had high sensitivity demonstrates its capability for indicating liver damage in alcoholics; additionally, its high positive and negative predictive values allow the differentiation with high probability of individuals with and without liver disease. Whereas the results of our study which showed a positive correlation of AST levels among severe alcoholics is contradicting Rosman and Lieber where they state that activities of liver enzymes, including AST, have limited diagnostic usefulness in predicting the histologic staging.[30]

Studies had mentioned that in alcoholics, alkaline phosphatase (AP) alterations are more significant than those of GGT (Gamma-glutamyl transferase). AP activity is elevated when cholestatic processes are present.[31] In the current study we found both the AP and GGT were elevated among the severe alcoholics.

Only three studies in the recent past looked at B vitamin status; Van der Gaag *et al* showed no effect of any alcohol intervention on vitamin B_{12}, a fall in folate with spirits consumption and an increase in vitamin B_6 with all alcohol interventions.[32] In contrast, Laufer et al only showed an effect of ethanol on vitamin B_{12}, with no effect on folate.[33] Beulens et al showed that beer consumption increased pyridoxal-5-phosphate (the active form of vitamin B_6), seemed to decrease vitamin B_{12}, but had no effect on folate.[34] In our study we found both vitamin B12 and folate levels were grossly reduced among severe alcoholics in comparison with non-alcoholic group.

CONCLUSION

Anemia, neutropenia and thrombocytopenia along with increase in liver enzymes and reduction in vitamin B12 and folate levels are the common hematological abnormalities among the patients with severe alcoholism. Detection of hematological changes in chronic alcoholics and giving psychiatric counseling and treatment for alcohol dependence will decrease the future complications like cirrhosis liver, cardiac and renal disease, cerebellar degeneration, neuropathy, pancreatitis, etc. and reduce the morbidity and mortality in alcoholics.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. The Social Issues Research Centre. Social and Cultural Aspects of Drinking; A report to the European Commission; 1998.
2. Besheer J, Grondin JJ, Salling MC, Spanos M, Stevenson RA. Interoceptive effects of alcohol require mGlu5 receptor activity in the nucleus accumbens. J Neurosci. 2009;29:9582-91.
3. World Health Organization Global status report on alcohol. Geneva: World Health Organization. 2004.March (assessed on 6th June 2017)
4. Dan L Longo, Anthony S Fauci, Dennis L Kasper, Stephen L Hauser, J Larry Jameson, etal Harrison's Internal Medicine 18-th edition; Alcohol and Alcoholism. The McGraw-Hill Medical, Companies 2011. Chapter 392.
5. Robert M. Morse, MD; Daniel K. Flavin, MD, The Definition of Alcoholism JAMA. 1992;268(8):1012-4.
6. Albano E. Alcohol, oxidative stress and free radical damage. Proceedings of the Nutrition Society. 2006;65(3):278=290.

7. Ballard HS. The hematological complications of alcoholism. Alcohol Health Res World. 1997;21:42-52.
8. EO Akanni, VO Mabayoje, TO Zakariyahu, DP Oparinde. Haematological Characterization among Heavy Alcohol Consumers in Osogbo Metropolis. Research Journal of Medical Sciences. 2010;4:48-52.
9. Deitrich, RA., Erwin, GV. Pharmacological Effects of Ethanol on the Nervous System. Boca Raton, FL: CRC Press, 1996. pp. 383-408.
10. Savage D, Lindenbaum J. Anemia in alcoholics. Medicine (Baltimore). 1986;65:322.
11. Girard DE, Kumar KL, McAfee JH. Hematologic effects of acute and chronic alcohol abuse. Hematol Oncol Clin North Am. 1987;1:321.
12. Seppä K, Sillanaukee P, Saarni M. Blood count and hematologic morphology in nonanemic macrocytosis: differences between alcohol abuse and pernicious anemia. Alcohol. 1993;10:343.
13. Latvala J. Effect of alcohol consumption and acetyladehde on blood cells and molecules: Pathogenic and diagnostic implications. Tampere University press Universitaties Tamperensis. 2005. 1035.
14. Esmeralda Thoma1, Sonila Bitri, Klodeta Mucaj, Andrin Tahiri and Irida Pano.. Changes of Some Blood Count Variables in Correlation with the Time of Alcohol Abuse. J Addict Res Ther. 2015;6:2
15. Gacouin A, Legay F, Camus C, Volatron AC, Barbarot N. At-risk drinkers are at higher risk to acquire a bacterial infection during an intensive care unit stay than abstinent or moderate drinkers. Crit Care Med. 2008;36:1735-41.
16. Bagnardi V, Blangiardo M, La Vecchia C, Corrao G. Alcohol consumption and the risk of cancer: a meta-analysis. Alcohol Res Health. 2001;25:263-70.
17. Gacouin A, Roussel M, Gros A, Sauvadet E, Uhel F.Chronic alcohol exposure, infection, extended circulating white blood cells differentiated by flow cytometry and neutrophil CD64 expression: a prospective, descriptive study of critically ill medical patients. Ann Intensive Care. 2012.2:50.
18. Corrao G, Bagnardi V, Zambon A, Arico S. Exploring the dose-response relationship between alcohol consumption and the risk of several alcohol-related conditions: A meta-analysis. Addiction. 1999;94:1551-73.
19. Bosetti C, La Vecchia C, Negri E, Franceschi S. Wine and other types of alcoholic beverages and the risk of esophageal cancer. Eur J Clin Nutr. 2000;54: 918-20.
20. O Erhabor A, Yakubu IZ, Isaac AL, Sule A, Wase AC. Effect of Alcohol Consumption on Platelet, Prothrombin Time and Activated Partial Thromboplastin Time of Alcoholics in Birnin Kebbi, Kebbi State, Nigeria, International Journal of Tropical Disease Health. 2014;4.
21. Adias Teddy Charles, Egerton Everton, Erhabor Osaro. Evaluation of haematological parameters among alcoholics in Port Harcourt Nigeria. British Journal of Medical and Health Sciences. 2013;5:32-44.
22. Das SK, Mukherjee S, Vasudevan DM, Balakrishnan V. Comparison of haematological parameters in patients with non-alcoholic fatty liver disease and alcoholic liver disease. Singapore Med J. 2011;52:175-81.
23. Stickel F, Seitz HK. Update on the management of alcoholic steatohepatitis. J Gastrointestin Liver Dis. 2013;22:189-97.
24. Mathurin P, Lucey MR. Management of alcoholic hepatitis. J Hepatol. 2012;56 Suppl 1:S39-S45.
25. Hamberg KJ, Carstensen B, Sørensen TI, Eghøje K. Accuracy of clinical diagnosis of cirrhosis among alcohol-abusing men. J Clin Epidemiol. 1996;49:1295-1301.
26. Becker U, Deis A, Sørensen TI, Grønbaek M, Borch-Johnsen K, Müller CF, Schnohr P, Jensen G. Prediction of risk of liver disease by alcohol intake, sex, and age: a prospective population study. Hepatology. 1996;23:1025-9.
27. Gartner U, Goeser T, Wolkoff A W. Effect of fasting on the uptake of bilirubin and sulfobromophthalein by isolated perfused rat liver. Gastroenterol. 1997;113:1701-13.
28. Palmer R H. Fasting hyperbilirubinemia (Letter). Gastroenterol. 1998;115:799.
29. Lieber C S - Alcohol-Nutrition interaction. Bol Asoc Med P Rico 1984;76:445-7.
30. Rosman A S, Lieber C S. Diagnostic utility of laboratory tests in alcoholic liver disease. Clin Cem. 1994;40:1641-51.
31. Schaffner F, Popper H. Alcoholic hepatitis in the spectrum of ethanol-induced liver injury. Scand J Gastroent 1970;Suppl.7:69-78.
32. Van der Gaag MS, Ubbink JB, Sillanaukee P, Nikkari S, Hendriks HF. Effect of consumption of red wine, spirits, and beer on serum homocysteine. Lancet. 2000;355:1522.
33. Laufer EM, Hartman TJ, Baer DJ, Gunter EW, Dorgan JF, Campbell WS, et al. Effects of moderate alcohol consumption on folate and vitamin B(12) status in postmenopausal women. Eur J Clin Nutr. 2004;58:1518-24.
34. Beulens JW, Sierksma A, Schaafsma G, Kok FJ, Struys EA, Jakobs C, et al. Kinetics of homocysteine metabolism after moderate alcohol consumption. Alcohol Clin Exp Res. 2005;29:739-45.

Cite this article as: Elanchezhian, Yoganandh T, Mayilsamy S, Radhakrishnan S. Comparison of haematological parameters between alcoholics and non-alcoholics. Int J Res Med Sci 2017;5:5041-7.