Research Article

Study of hematological and biochemical profile in patients with alcoholic liver disease in rural Maharashtra

Vedant Rajendraprasad Awasthi[1,2], Janhavi Jaywant Deshpande[2,3], Pawar Sujata[4]

[1]Director, Noble Hospital and Critical Care Centre, Latur, Maharashtra, India, [2]Department of General Medicine, Pravara Rural Hospital and Medical College, Loni, Maharashtra, India, [3]Department of General Medicine, B.K.L. Walawalkar Rural Medical College and Hospital, Ratnagiri, Maharashtra, India, [4]Department of Pediatric, Samarth Nursing College, Dervan, Maharashtra, India

Correspondence:
Dr. Janhavi Jaywant Deshpande, Department of General Medicine, B.K.L. Walawalkar Rural Medical College and Hospital, Dervan, A/P Sawarde-Kasarwadi, Taluka Chiplun, Ratnagiri – 415 606, Maharashtra, India.
E-mail: dvjaywant@gmail.com

How to cite this article:
Awasthi VR, Deshpande JJ, Sujata P. Study of hematological and biochemical profile in patients with alcoholic liver disease in rural Maharashtra. Innov Pharm Pharmacother 2019;7(2):27-30.

Source of Support: Nil.
Conflicts of Interest: None declared.

ABSTRACT

Background: Alcoholism is condition ensuing from excess drinking of beverages that contain alcohol. The main health risk of alcoholism includes liver disease, heart disease, pancreatitis, central nervous system disorders and certain forms of cancer. Alcohol will be manifested in liver injury from fibrosis to end stage of cirrhosis and should eventually result in liver cancer. The progression of alcoholic disease is characterised by steatosis, inflammation, necrosis and cirrohosis. **Aim:** To study of hematological and biochemical profile in patients with alcoholic liver disease. **Methodology:** The prospective hospital-based case control study was done at Pravara Rural Hospital and Medical College, Loni from September 2013 to September 2015. A total of 100 cases of alcoholic liver disease were included. **Results:** Hematological abnormalities reveals that Anemia (hemoglobin <13 g/dL), MCV >96fl fl and platelet count <150,000/mm^3 were present in 75%, 42% and 62% patients, respectively. Raised SGOT seen in 84% of the patients. Followed by raised prothrombin time seen in 79% of the patients. Raised PT-INR levels seen in 68% patients. And raised SGPT in 63% of patients. Hypoalbuminemia seen in 57% of the alcoholics and raised bilirubin seen in 50% of alcoholics. **Conclusion:** The present study it was concluded that various hematological parameters changed due to alocoholic liver disease.

Keywords: Alcoholic liver, biochemical, hematological

Introduction

Alcohol use is increasing hurriedly in developing regions and could be a major concern among autochthonous folks round the world, showing a higher prevalence of the disease. However, levels and patterns of alcohol consumption do not fully explain the cause of alcoholic liver disease mortality.[1] The global burden of disease project estimated alcohol to be responsible for 1.5% of all deaths and 3.5% of those who live life with a disability.[2] In the USA, 67.3% of the population over 18 years old drinks alcohol annually.[3]

Access this article online	
Website: www.innpharmacotherapy.com	**e-ISSN:** 2321-323X **p-ISSN:** 2395-0781

Alcoholism is a condition ensuing from excess drinking of beverages that contain alcohol. The main health risk of alcoholism includes liver disease, heart disease, pancreatitis, central nervous system disorders, and certain forms of cancer.[4] Alcohol will be manifested in liver injury from fibrosis to end stage of cirrhosis and should eventually result in liver cancer. The liver is especially susceptible to unwellness associated with significant drinking, most typically termed as alcoholic hepatitis or cirrhosis. The progression of alcoholic disease is characterized by steatosis, inflammation, necrosis, and cirrhosis. Once severe cirrhosis happens, death is the outcome.[5] Chronic consumption of alcoholic beverages could be a primary reason behind liver injury. Chronic and excessive consumption of alcoholic beverages provokes membrane lipid-peroxidation due to triglyceride accumulation in hepatocytes.[6] Hence, an attempt has been made to evaluate the hematological and biochemical parameters of alcoholic liver disease patients. Hematologic tests, namely, red blood cell counts, white blood cell counts, hemoglobin levels, and mean corpuscle volumes are strong

indicators of alcoholic liver disease as reported by other researchers.[7] Serum was separated and various biochemical parameters of total bilirubin, conjugated and unconjugated bilirubin, total protein, albumin, and albumin:globulin ratio, aspartate transaminase, gamma-glutamyl transferase tests were done.

Methods

The prospective hospital-based case–control study was done at Pravara Rural Hospital and Medical College, Loni, from September 2013 to September 2015. A total of 100 cases of alcoholic liver disease were included.

Inclusion criteria

The following criteria were included in the study:

- Patient diagnosed with alcoholic liver disease
- Patients aged above 16 years
- Patients of both sexes will be taken for study.

Exclusion criteria

The following criteria were excluded from the study:

- Patients with hepatitis secondary to other than significant alcohol consumption
- Patients aged below 16 years.

Results

The mean hemoglobin, platelet counts, total leukocyte count, and mean corpuscular volume (MCV) were 10.215 ± 3.339 g/dL, 140627 ± 89899/μL, 10063 ± 5432.7 cumm, and 90.501 ± 11.63 fl, respectively. Anemia (hemoglobin <13 g/dL), MCV >96fl, and platelet count <150,000/mm^3, and total leukocyte count (>11000/cumm) were present in 75%, 42%, and 62%, and 36% patients, respectively [Table 1].

The mean serum glutamic oxaloacetic transaminase (SGOT), serum glutamic pyruvic transaminase (SGPT), SGOT: SGPT, total bilirubin, albumin, A: G ratio, prothrombin time, prothrombin time difference, and prothrombin time and international normalized ratio (PT-INR) were 140.84 ± 119.66 U/L, 69.645 ± 60.03 U/L, 2.02 ± 1.99, 6.152 ± 6.35 mg/dL, 2.816 ± 0.52 g/dL, 0.93 ± 0.64, 19.126 ± 7.09 s, 6.098 ± 5.64 s, and 1.65 ± 0.69, respectively.

The mean Na+, K+, urea, and creatinine were 134.49 ± 6.98 meq/dL, 4.05 ± 0.781 meq/dL, 44.48 ± 31.73 mg/dL, and 1.75 ± 1.68 mg/dL, respectively. Hyponatremia (<130 meq/dL), hypokalemia (<3.5 meq/dL), raised urea (>40 mg/dL), and creatinine (>1.5 mg/dL) were present in 23%, 20%, 41%, and 31% of the patients, respectively [Table 2].

Discussion

In the present study, a total of 100 cases were studied.

Table 1: Hematological abnormality in patients of alcoholic liver disease

Hematological abnormalities	No. of cases (%)
Hemoglobin (<13 g/dl)	75 (75)
Platelet count (<150,000/μl)	62 (62)
Total leukocyte count (>11,000/cumm)	36 (36)
MCV (>96 fl)	42 (42)

MCV: Mean corpuscular volume

Table 2: Biochemical parameters

Biochemical parameters	No. of cases (%)
↑ Bilirubin total	50 (50)
↑ SGOT	84 (84)
↑ SGPT	63 (63)
↑ ALP	14 (14)
↑ PT-INR	68 (68)
↑ Prothrombin time (>14 s)	79 (79)
Hypoalbuminemia	57 (57)

SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, ALP: Alkaline phosphatase, PT-INR: Prothrombin time and international normalized ratio

Hematological abnormality

In the present study, the mean hemoglobin, platelet counts, total leukocyte count, and MCV were 10.215 ± 3.339 g/dL, 140627 ± 89899/μL, 10063 ± 5432.7 cumm, and 90.501 ± 11.63 fl, respectively.

Anemia (<11 g/dl), raised total leukocyte count (more than 11000 cumm), and low platelet count (<150,000/μl), and raised MCV (more than 96 fl) were seen in 75%, 62%, 36%, and 42% of the patients.

In comparison with study by Dr. Parmar and Vyas, the mean hemoglobin, platelet counts (×103/μL), and MCV were seen in 10.6 ± 3.45 g/dL, 176.8 ± 89.23, and 100.42 ± 9.09 fl, respectively.[8]

In study by Suthar *et al*., the mean hemoglobin, total leukocyte count, platelet count, and MCV were 10.1%, 9521, 148000, 97.6, respectively.[9]

Renal function test

In present study, the mean Na+, K+, urea, and creatinine were 134.49 ± 6.98 meq/dL, 4.05 ± 0.781 meq/dL, 44.48 ± 31.73 mg/dL, and 1.75 ± 1.68 mg/dL, respectively. Hyponatremia (<130 meq/dL), hypokalemia (<3.5 meq/dL), raised urea (>40 mg/dL), and creatinine (>1.5 mg/dL) were present in 23%, 20%, 41%, and 31% of the patients, respectively.

It is compared with the study by Suthar *et al*., which shows that the mean serum sodium was 132.1 meq/L. The mean serum creatinine was 1.74 mg/dl. Hyponatremia (<130 meq/dL) was seen in 31% of the patients, hypokalamia was seen in 22% of the patients, and serum creatinine (>1.5 mg/dl) was seen in 32% of the patients.[9]

The present study compared with study by Dr. Parmar and Vyas which shows that the mean Na+, K+, urea, and creatinine were 130.08 ± 8.43 meq/dL, 3.8 ± 1.04 meq/dL, 70.04 ± 64.86 mg/dL, and 1.8 ± 1.2 mg/dL, respectively. Hyponatremia (<130 meq/dL), hypoka¬lemia (<3.5 meq/dL), raised urea (>40 mg/dL), and creatinine (>1.5 mg/dL) were present in 20%, 30%, 52%, and 42% of the patients, respectively.[8]

Liver function test

Bilirubin total

In present study, the mean bilirubin total 6.152 ± 6.35 mg/dL and raised blirubin total were seen in 50% of the patients.

In comparison with Dr. Walter and Ashraf, study hyperbilirubinemia was found in 40% of the patients.[10]

In Sarmistha *et al*., study hyperbilirubinemia was found in 34% of cases.[11]

In the study of Suthar *et al*., the mean S. total bilirubin was 3.17 ± 1.81. Serum bilirubin is more than 5 mg/dl in 36% of patients.[9]

In Nand *et al.* study, hyperbilirubinemia was seen in 85% of patients with mean bilirubin was 5.28 ± 6.03 mg/dL.[12]

SGOT, SGPT, and SGOT: SGPT ratio

The mean SGOT, SGPT, and SGOT:SGPT were 140.84 ± 119.66 U/L, 69.645 ± 60.03 U/L, and 2.02 ± 1.99, respectively.

Raised SGOT levels were seen in 84% of the patients and raised SGPT levels were seen in 63% of the patients in the present study.

This is correlated with Dr. Walter and Ashraf, study which shows 42% of the subjects had elevated SGOT levels and 8% of the subjects had elevated SGPT levels. The SGOT to SGPT ratio in the study group was more than 2 in 40%.[10]

In Suthar *et al*., study the mean SGOT, SGPT, and SGOT: SGPT ratio were 134.6 IU/L, 56.1 IU/L, and 2.11, respectively.[9]

SGOT: SGPT ratio in Nand *et al.* study was 2.15 ± 0.88 and Suthar *et al*. study had ratio more than 2 in 32% of the cases.[9,12]

Alkaline phosphatase (ALP)

In the present study, the alkaline phosphatase raised in 14% of patients with a mean of 206.4 ± 112.8 IU/L.

It is compared with Dr. Walter and Ashraf, study which showed raised ALP levels in 12% of the patients and in Suthar *et al*., study the mean ALP was 208 IU/L.[9,10]

Albumin

In the present study, hypoalbuminemia was seen in 57% of the patients. With an incidence of 55.5% in cirrhosis without portal hypertension, 78.57% in cirrhosis with portal hypertension, 23.07% in fatty liver, and 22.07% in hepatitis. The mean albumin was 2.816 ± 0.52 g/dL.

In a study of Nand *et al*., mean albumin levels were 2.79 ± 0.62 g/dL and severe hypoalbuminemia was seen in 65%.[12]

In a study by Dr. Walter and Ashraf, 40% of the subjects were observed to have hypoalbuminemia.[10]

In a study by Suthar *et al*., the mean S. albumin was 2.41 g/dl. Hypoalbuminemia was found in 80% of patients.[9]

Albumin:globulin ratio

In the present study, A: G ratio is 0.93 ± 0.64. It is compared with the study by Dr. Parmar and Vyas, which shows ratio of 0.97 ± 0.51.77.[8]

Prothrombin time

In the present study, the raised prothrombin time is seen in 79% of the patients with a mean of 19.126 ± 7.09 s. This finding supports the diagnosis of cirrhosis of the liver. In the present study, it is raised in 92.85% of patients with cirrhosis of liver with portal hypertension and 100% in cirrhosis of liver without portal hypertension. It is compared with study by Dr. Walter and Ashraf, in which it is present in 28% of the patients.[10]

In the present study, the mean of prothrombin time difference is 6.098 ± 5.64 s. In study by Suthar *et al*. shows prothrombin time difference of 5.6 s in 72% of the patients.

Furthermore, it is 6.29 ± 6.16 s in the study by Dr. Parmar and Vyas.[8,9]

PT-INR

In the present study raised PT-INR seen in 68% of the patients with a mean of 1.65 ± 0.69. It is compared with the study by Nand *et al.* shows mean PT-INR 2.08 ± 0.89. The mean INR was 1.92 seen in the study of Suthar *et al*.[9,12]

Conclusion

From the present study, it was concluded that various hematological parameters changed due to alcoholic liver disease. There is raised SGOT seen in patients followed by raised prothrombin time. Furthermore, there is an increase in PT-INR levels and SGPT. Hypoalbuminemia is seen in 57% of the alcoholics and raised bilirubin seen in 50% of alcoholics. In cases of cirrhosis of the liver with portal hypertension, there is an increase in prothrombin time and SGOT levels. In hepatitis and fatty liver disease raised SGOT are important findings 90.9% and 53.84%, respectively.

References

1. Leon DA, McCambridge J. Liver cirrhosis mortality rates in Britain from 1950 to 2002: An analysis of routine data. Lancet 2006b;367:52-6.
2. Mandayam S, Jamal MM, Morgan TR. Epidemiology of alcoholic liver disease. Semin Liver Dis 2004;24:217-32.
3. Sofair AN, Barry V, Manos MM, Thomas A, Zaman A, Terrault NA, *et al.* The epidemiology and clinical characteristics of patients with newly diagnosed alcohol-related liver disease: Results from population-based surveillance. J Clin Gastroenterol 2010;44:301-7.

4. Usharani B, Vennila R, Nalini N. Biochemical changes in alcoholics a case control study. IJAM 2012;3:201-5.
5. Felver ME, Merzey E and Herlong HF. Plasma tumor necrosis factor, a predicts decreased long term survival in severe alcoholic hepatitis. Alcohol Clin Exp Res 1990;31:117-34.
6. Tuma DJ. Serial review: Alcohol, oxidative stress and cell injury free. Radic Biol Med 2002;32:303-8.
7. Ryback RS, Eckardt MJ, Felsher B, Rawlings RR. Biochemical and hematologic correlates of alcoholism and liver disease. JAMA 1982;248:2261-5.
8. Parmar C, Vyas M. Retrospective study of the clinical profile and prognostic indicators in patients of alcoholic liver disease admitted to a tertiary care teaching hospital. Int J Sci Res 2013;2:394-408.
9. Suthar H, Suthar K, Mewada B. Clinical profile of cases of alcoholic liver disease. Int J Med Sci Public Health 2013;2:394-8.
10. Walter A, Ashraf M. A study correlating the quantity and duration of alcohol consumption with liver function tests. IOSR J Dent Med Sci 2014;13:70-5.
11. Sarmistha B, Sujat P, Abu S, Shahriar MM, Ashik IK, Das GR, *et al*. Spectrum of alcoholic liver disease in tribal alcoholics of Chittagong hill tracts of Bangladesh. J Med 2011;12:7-11.
12. Nand N, Malhotra P, Dhoot DK. Clinical profile of alcoholic liver disease in a tertiary care centre and its correlation with type, amount and duration of alcohol consumption. J Assoc Physicians India 2015;63:14-20.