Prospective Study to Evaluate Anemia and its Covariation with Hypoalbuminemia in Patients Suffering from Ascites Cirrhosis

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Anemia is the reduction in quantity of haemoglobin pigment in circulating blood. It is the manifestation of chronic inflammation, infection or malignancy in the body. The study sample comprised of total 30 participants in two groups. The mean haemoglobin of cirrhotics (9.86 ± 1.8 mg/dl) was lesser in comparison to haemoglobin (13.09 ± 1.76 mg/dl) of controls significantly ($p<0.0001$). Further, sample t-test revealed a significant ($p<0.0001$) reduction in concentration of haemoglobin of cirrhotics at a cut off (13 mg/dl). The anemia in cirrhotics covariated significantly ($p=0.05$), with hypoalbuminemia, as revealed in Pearson's coeffiecient of correlation ($r=0.043$).

Keywords: Anemia, Haemoglobin, Cirrhosis, Hypoalbuminemia.

1. INTRODUCTION

Cirrhosis is the chronic liver disease. The term cirrhosis was first introduced by Laennec in 1826. It is derived from the Greek term 'scirrhus' and it means 'the orange or tawny surface of the liver seen at autopsy'.

Cirrhosis is defined as diffuse inflammatory process characterized by fibrosis, scarring and formation of regenerative nodules in the liver lobules and the liver architecture is altered along with abnormality in liver vasculature. The progression of liver injury to cirrhosis may occur over weeks to years [1,2]. Vasodilating substances produced in body in cirrhosis have profound effect on blood circulation and haemodynamics. Nitric oxide, endothelin, endotoxins, CGRP and prostaglandins are the powerful vasodilating substances. These are implicated in vasodilation of splanchnic and peripheral vascular beds [3,4]. The high pressor baroreceptors in carotid sinus and aortic arch are stimulated to activate SNS, thus inducing the release of norephenephrine (NE) from adrenal medulla. This potent vasoconstrictor causes renal vasoconstriction [5]. Renal hypoperfusion activate Renin–Angiotensinogen-Aldosterone System. The patients with ascites cirrhosis have essentially, the features of anemia. The causes can be destruction of red blood cells, enlarged spleen, blood loss due to damage to duodenum, rupture of esophageal varices and decreased production of red blood cells due to its lack of stimulation by erythropoietin.

2. AIM AND OBJECTIVES

To assess anemia and its covariation with hypoalbuminemia in patients suffering from ascites cirrhosis.

The objectives of haemoglobin concentration, serum albumin level estimation in ascites cirrhosis parients and to assess the covariation of both, were fulfilled.

3. MATERIALS AND METHODS

3.1. Research Design: Prospective, observational, single centre, case control study was undertaken.

3.2. Sampling Design:

- Sample: The study sample was constituted of total 30 participants, which were divided into two groups. The Group 1 (ascites cirrhosis patients) consisted of 15 patients, while, Group 2, was composed of 15 participants as controls as shown in Table 1.
- Sampling method: The simple random sampling was utilized for sample selection.
- Sample selection criteria: The following criteria were used, for both, cases and controls.

A. Criteria for selection of patients in Group 1

Inclusion criteria:

- Patients with ascites cirrhosis (as diagnosed by case history, clinical examination and confirmed by lab. Investigation).
- Patients above 18 years of age.
- Patients of both gender.

Exclusion criteria:

- Patients suffering from liver disease other than ascites cirrhosis.
- Patients suffering from any systemic/metabolic disorder.
- Patients with AIDS.
- Patients with liver malignancy.
- Patients already enrolled in any other clinical trial.

B. Criteria for selection of patients in Group 2

Inclusion criteria:

- Healthy controls (as confirmed by case history, clinical examination and lab values).
- Age matched controls.
- Case: Control proportion was (1:1).
- Hospital based selection of controls.

Exclusion criteria;

- Drug addicts and blood transfused controls were excluded.
- Controls outside hospital population.

3.3. Data Collection

Primary data were collected, at single time point, from amongst participants who visited hospital. The data were collected by utilizing following methods:

- Pre-structured clinical Interview method was used for demographic data.
- Data related to clinical signs and symptoms were collected by Pre-structured Schedules. Biochemical markers were estimated by laboratory investigation of collected blood specimens.

3.4. Cut off Values

- Serum Albumin : 3.4 mg/dl
- Serum Bilirubin : 1.2 mg/dl
- Serum Aminotransferases : >40 IU/L
- Haemoglobin : 13 mg/dl [6].

3.5. Collection of Blood Specimens

Blood specimens were collected from participants, sera separated and investigated by following methods:

- Estimation of serum Albumin: by Bromocresol green dye method [6].
- Estimation of serum Bilirubin: by Diazotised sulphinilic acid method [6].
- Estimation of serum Aminotransferases: by Colorimetric method by Reitman and Frankel [8].
- Estimation of haemoglobin: by Sahli's method [6].

4. RESULTS

(i) The mean age (50.6 ± 8.5 y) of Group 1 (ascites cirrhotics) showed difference with mean age (49 ± 8.4 y) of the Group 2 (control), but proved statistically non significant (p=0.60) as shown in Table 1.

(ii) Mean serum albumin (2.4 ± 0.29 mg/dl) of Group 1 was significantly (p<0.0001) lower than mean serum albumin (4.2 ± 0.63 mg/dl) of Group 2 as shown in Table 2.

(iii) Mean serum bilirubin (4.6 ± 2 mg/dl) of Group 1 was proved significantly (p<0.0001) higher than mean serum bilirubin (0.96 ± 0.18 mg/dl) of Group 2 as shown in Table 2.

(iv) SGPT (87.4± 40.7 IU/L) and SGOT (110.7 ± 50.5 IU/L) of Group 1, were significantly (p<0.0001) higher than SGPT (27.3 ± 10.15 IU/L) and SGOT (24.8 ± 9.3 IU/L) of Group 2 respectively as shown in Table 2.

(v) Mean haemoglobin (9.86 ± 1.8 mg/dl) of Group 1 was lower to mean haemoglobin (13.09 ± 1.76 mg/dl) of Group 2 with high statistical significance (p<0.0001) as shown in Table 2.

(vi) Pearson's coefficient of correlation (r=+0.43) was found to be significant (p=0.05), between serum albumin and haemoglobin in ascites cirrhosis as shown in Figure 1.

(vii) Scatter plot as in Figure 1 showed that readings were moving from lower left corner of graph to right upper corner, thus it indicated positive, direct correlation between haemoglobin and serum albumin as shown in Figure 1.

Table 1: Demographic characteristics of participants.

Characteristics	Group 1 (Ascites cirrhosis)	Group 2 (Controls)
Number of participants	15	15
Age (mean ±SD)	50.6 ± 8.5 y	49 ± 8.4 y
Gender (M/F)	15/0	15/0
Cause of disease (n)%	(12/15) 80% alcoholic cirrhosis (3/15) 20% HCV induced cirrhosis	Nil
Duration of Alcoholism >10 y expressed as (n)%	100 %	Nil

Table 2: Clinical parameters for ascites cirrhotics and controls.

Parameters	Group 1 (Ascites Cirrhosis)	Group 2 (Controls)	Significance p value (0.05)
Serum Albumin(3.5-5 mg/dl)	2.4 ± 0.29	4.2 ± 0.6	<0.0001
Serum Bilirubin (0.2-2.0mg/dl)	4.6 ± 2.0	0.96 ± 0.18	<0.0001
SGPT (0-40 IU/L)	87.4 ± 40.7	27.3 ± 10.15	<0.0001
SGOT (0-40 IU/L)	110.7 ± 50.5	24.8 ± 9.3	<0.0001
Haemoglobin (cut off 13 mg/dl)	9.86 ± 1.8	13.09 ± 1.76	<0.0001

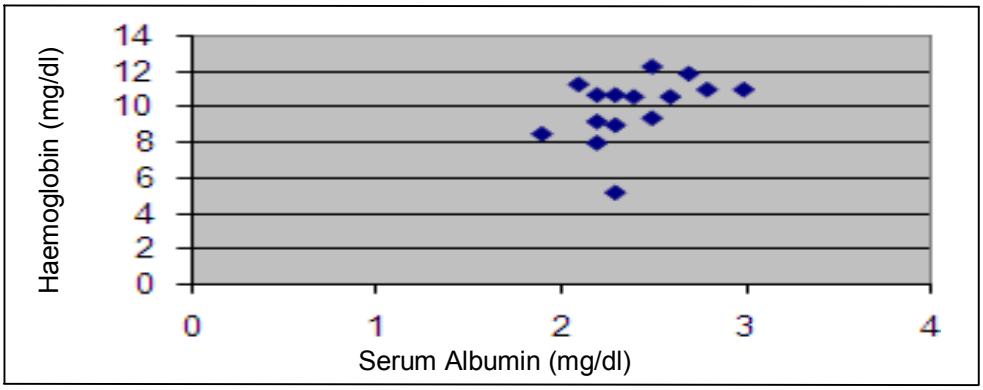

Fig. 1: Scatter plot between Haemoglobin and Serum Albumin

5. DISCUSSION

(i) Anemia as revealed by haemoglobin concentration was found significantly in the ascites cirrhosis patients. The possible explanation for this fact is the multifactorial nature of Anemia in cirrhotics. These patients have poor dietary intake, impaired hepatic functions and alcoholic consumption, all lead to deficiency of vitamin B12, iron, folic acid. Further, oesophageal varieces, haematemesis would lead to blood loss. Hypervolumic anemia is also seen in ascites cirrhosis. It is due to increased volume of plasma as compared to circulating RBC mass [7].

(ii) Hypoalbuminemia as revealed in ascites cirrhotics, also a common consequence of advanced cirrhosis. The explanation for this fact is the renal inability to excrete free water and sodium from body. This results into plasma hypervolumia, ↓ plasma osmolarity, ↓ serum albumin and ↓ haemocrit values. Further, serum albumin decrease is also attributed to increased volume of distribution [8,9]. The positive direct correlation between albumin and haemoglobin in Group 1 may have prognostic significance in advanced cirrhosis.

6. CONCLUSION

The ascites cirrhosis is associated with significant reduction in haemoglobin concentration. Further, the hypoalbuminemia is covariated directly with anemia in cirrhosis.

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