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Original Research Article

Correlation of Serum Ascites Albumin Gradient and Endoscopic Parameters of Portal Hypertension in Chronic Liver Disease

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Abstract

Background: Chronic liver disease denotes the disease of the liver which lasts over a period of 6 months or more. The serum ascites albumin gradient (SAAG) is a formula that is used to assist in determining the etiology of ascites. It is also used in detecting ascites of portal hypertension in the adult population. We don't have any research-based information regarding the correlation between serum ascites albumin gradient and endoscopic parameters of portal hypertension in chronic liver disease. ***Aim of the study*:** The aim of this study was to assess the correlation between serum ascites albumin gradient and endoscopic parameters of portal hypertension in chronic liver disease. ***Methods:*** This prospective observational study was conducted in the Department of Hepatology, BSMMU, Dhaka, Bangladesh during the period from January 2005 to December 2005. In total 50 patients with cirrhosis with ascites with high SAAG values (>1.1 gm/dl) were included as the study people. The age of the patients was 15 to 70 years. Both males and females were included in the study. All data were entered into a personal computer, thoroughly checked for any possible error, and then processed and analyzed by the SPSS program. The significance of the test was tested by the chi-square test. P-value of <0.05 was taken as statistically significant. Correlation analysis was done by the Pearson correlation test**.** ***Result:*** In this study, there were three SAAG groups. In SAAG group 1.10-1.49gm/dl, 53.33% of patients had mild grades of PNG, and 33.33% of patients had a severe grade of PNG. In the SAAG group, 1.50-1.99 gm/dl, 44.44% of patients had mild grade and 33.33% had a severe grade of PNG and in the SAAG group >2 gm/dl, 42.30% had mild grade and 49.99% had a severe grade of PNG. But there had not been any significant correlation among the groups regarding SAAG values and PHTN grades because the p-value was greater than 0.05. ***Conclusion:*** It was shown in this study that, oesophageal varices were present in 49 patients, sensitivity was 98%; portal hypertensive gastropathy in 44 patients, sensitivity 88%, and both oesophageal varices and gastropathy in 43 patients, sensitivity 86%. So high SAAG value can be used as an indicator of the presence of portal hypertensive changes especially oesophageal varices and gastropathy in the upper gastrointestinal tract. A weak positive correlation was found in this study between SAAG values and grades of oesophageal varices (r=0.358, p=0.011) and gastropathy (r-0.139, p--0.33) but no correlation was found between SAAG and gastric varices (p=0.4).

Keywords: Ascites albumin, SAAG Portal hypertension Oesophageal varices, Liver disease.

I. INTRODUCTION

Chronic liver disease denotes the disease of the liver which lasts over a period of 6 months or more. The serum ascites albumin gradient (SAAG) is a formula that is used to assist in determining the etiology of ascites. It is also used in detecting ascites of portal hypertension in the adult population. In several studies recently carried out, it was emphasized that serum ascites albumin gradient (SAAG) based on the difference between albumin level of serum and ascitic fluid should be used to determine the aetiology of ascites cases instead of discrimination between transudate and exudates [1]. It was shown that such a classification has a validity rate of 96.7% in detecting ascites of portal hypertension in the adult population whereas distinguishing transudate from exudate based

on criteria requiring total protein level in ascites above 2.5gm/di has a validity rate of 55.6% in presence of portal hypertension [2]. In the last years, the concept that the exudative ascites arises from peritoneal inflammation has been abandoned as total protein level above 4gm/dl were found in peritoneal fluid samples of healthy women. The osmotic pressure gradient between portal venous blood and the peritoneal cavity is a direct function of the corresponding capillary hydrostatic pressure gradient. The difference between serum and ascites albumin concentration (SAAG) is thought to reflect directly colloid osmotic pressure gradient and indirectly degree of portal hypertension. SAAG is a better discriminator of portal hypertension than ascites total protein concentration [2]. Indeed SAAG is now considered a useful physiological clinical tool in the workup of ascites. Patients with SAAG >1.1 gm/dl are considered as high SAAG and indicate the presence of portal hypertension while those with SAAG <1.1 gm/dl are considered as low SAAG and indicate the absence of portal hypertension. Net portal pressure is correlated strongly with SAAG in patients with cirrhotic ascites (r=0.81, p< 0.001) [2] [Portal pressure in mm Hg=7.08xSAAG+3.62]. Serum-ascites albumin gradient, an index of serum-ascites oncotic pressure difference, correlates directly with the pressure gradient between portal capillaries and peritoneal cavity [1]. SAAG is compared with the ascites total protein concentration in the separation of transudation and exudative ascites. SAAG is large in patient with transudative ascites and small in patient with exudative ascites and provided significantly better discrimination of these categories than did the ascites total protein concentration. SAAG does not provide perfect discrimination of any category, however; in patients with mixed causes of ascites, this difference tended to be large, resembling ordinary transudative ascites, a potential source of diagnostic error. Nevertheless, the SAAG has superior discriminatory power and replaced the ascites total protein concentration in the routine diagnostic examination of ascites [1]. Ascites caused by raised hydrostatic pressure in the portal venous bed or diminished oncotic pressure or both is called transudative ascites. A large hydrostatic pressure gradient between portal capillaries and peritoneal cavity characterizes all transudative causes of ascites except nephrotic syndrome, generating large blood to ascites oncotic pressure difference and consequently, a low ascites total protein concentration. A relatively high ascites protein concentration may be seen in patients with transudative ascites when the blood oncotic pressure determined chiefly by the albumin concentration is relatively well preserved as occurs in some patients with cirrhosis and most patients with CCF or constrictive pericarditis. Conversely, a relatively low ascites protein concentration may be found in patients with exudative ascites if there is a severe reduction of the serum albumin concentration. These relationships limit the diagnostic usefulness of the ascites total protein concentration (1). Portal hypertensive changes in upper gastrointestinal endoscopy arc oesophageal varices, gastric varices and portal hypertensive gastropathy. The oesophageal varices are graded according to Japanese classification into three grades (Japanese research society for portal hypertension 1980). There is some red color signs present on oesophageal varices. They are red wale markings, cherry red spot and haematocystic spots. Gastric varices are assessed according to position in stomach and type of varices into tortuous type, tumor type and notched type. Portal hypertensive gastropathy is assessed according to New Italian Endoscopic Club (NIEC) classification into mild and severe type [3]. Mild cases are characterized by a mosaic-like pattern (MLP) with pink in the center. It is characterized by the presence of small, polygonal pinkish areas surrounded by a whitish-yellow depressed border. Severe cases are mosaic-like patterns with redness in the center, redpoint lesion (RPL), cherry red spot (CRS), black-brown spot (BBS). When these signs are present singly or in combination with or without a mosaic-like pattern with pink in the center it is called severe portal hypertensive gastropathy. In several studies on cirrhosis, it was shown that a correlation exists between SAAG and portal hypertension in cirrhotic patients with ascites. SAAG was proposed to be a factor determining the degree of portal hypertension and prognosis of the patients in cirrhosis. In a study performed by Hoefs et al. in 1983 [4], it was shown that an excellent correlation exists between portal hypertension and SAAG. In this study, a numeric formula was established for the first time between portal hypertension and SAAG in 56 patients (r-0.73, p<0.05). The numeric formula was as follows: Portal gradient=7.08 X SAAG +3.62. A similar correlation was found by Rector et al. in 1984 [1] in a study on 18 patients. In this study on patients with alcoholic cirrhosis, a correlation was found between portal hypertension and SAAG (p = 0.001 and r- 0.8). In 1990, Kajani et al. [5] investigated the correlation in patients with alcoholic cirrhosis and with cirrhosis due to other causes separately. In this study, a correlation was found between SAAG and either portal pressure (r=0.62) or oesophageal varices (r=0.53) in alcoholic patients. But in the patients with nonalcoholic cirrhosis, no significant correlation was found between SAAG and portal pressure (r=0.39), while the correlation between SAAG and the degree of varices was found to be weaker (r=0.02). An increased incidence of portal hypertensive gastropathy in patients with Child's C liver disease could possibly be related to humoral substances, neuronal influences, and some unidentified factors [6]. In a recent study by Torres et al. in 1998 [7], the correlation between SAAG and oesophageal varices was studied (p=0.001, r=0.54). In this study, a total of 31 patients were included. Among them, 25 patients had high SAAG. Among the patients of high SAAG value, 17 had oesophageal varices (68%). Varices were present in 4 of 10 (40%) patients with SAAG value 1.10 to 1.49 gm/dl; in 4 of 6 patients (66.7%) with SAAG value 1.50 to 1.99 gm/dl and in 9

of 9 patients (100%) with SAAG values of >2 gm/dl. Grade-I was present in 1 patient (25%), Grade-II in 1 patient (25%), and Grade-Ill in 2 patients (50%) with SAAG value 1.10 to 1.49 gm/dl. Grade-I was present in 2 patients (50%), Grade-II in 2 patients (50%), and Grade-Ill in 0 patients with SAAG value 1.5 to 1.99 gm/dl. Grade-I was present in 2 patients (22.2%), Grade-II in 4 patients (44.4%), and Grade-Ill 3 patients (33.4%) with SAAG value >2gm/dl. Direct portal pressure measurement is an invasive procedure. Portal pressure can be guessed by upper gastrointestinal endoscopy. Portal hypertensive changes like varices and gastropathy are seen by upper gastrointestinal endoscopy. As the portal pressure rises, these changes begin to appear. So, a correlation exists between SAAG and portal hypertensive changes in upper gastrointestinal endoscopy in cirrhotic patients with ascites. We have the aim at evaluating whether such a correlation exists between SAAG values and portal hypertensive changes (varices, gastropathy) in upper gastrointestinal endoscopy in cirrhotic patients with ascites.

II. OBJECTIVES

General Objective

- To evaluate the use of high SAAG value as a preliminary indirect parameter of presence of portal hypertensive changes (varices, gastropathy) in upper gastrointestinal endoscopy.

Specific Objective

- To correlate the degree of high SAAG values with the grades or sizes of oesophageal varices.
- To correlate the degree of high SAAG values with the presence of gastric varix.
- To correlate the degree of high SAAG values with the grades of portal hypertensive gastropathy.

III. METHODOLOGY & MATERIALS

This prospective observational study was conducted in the Department of Hepatology, BSMMU, and Dhaka, Bangladesh during the period from January 2005 to December 2005. In total 50 patients with cirrhosis with ascites with high SAAG values (>1.1 gm/dl) were included as the study people. The age of the patients was 15 to 70 years. According to the exclusion criteria of this study cirrhotic patients with ascites with low SAAG values (<1.1 gm/dl), pregnant women, cases with a space-occupying lesion (SOL) in the liver or intra-abdominal tuberculosis or malignancy, patients received endoscopic treatment for oesophageal varices previously and patients to whom endoscopy was contraindicated were excluded. When a patient is seen to have the features of chronic liver disease with abdominal swelling in the inpatient and outpatient department of Hepatology, then the patient is provisionally selected for the study. After taking written consent from the patient, biochemical liver function tests and viral serological tests that is, serum bilirubin, ALT, AST, prothrombin time, serum albumin, HBsAg, Anti-HCV were done. Ultrasonography of the hepatobiliary system was done to detect the presence of features of chronic liver disease, presence of ascites and to exclude space-occupying lesions in the liver. Ascitic fluid was aspirated from the abdomen through the abdominal wall at the junction between the medial two-third and lateral one-third of the spino-umbilical line under aseptic precaution after asking the patient to evacuate the bladder in the procedure room. The ascitic fluid was sent for cytology, total protein and albumin, and malignant cell. At the same time, venous blood was drawn and sent for serum albumin concentration estimation. After obtaining the report, serum ascites albumin gradient (SAAG) was determined. If the value of the SAAG is high(>l.lgm/dl), the patient is selected for further procedure. If the value is low(<l.lgm/dl), the patient is excluded from the study. Then the endoscopy of the upper gastrointestinal tract was done by Olympus video endoscopy in the endoscopy room after topical anesthesia (10% xylocaine spray) in the presence and under the direct supervision of the thesis supervisor. During endoscopy, the esophagus was surveyed for evidence of oesophageal varices. If oesophageal varices are present, then the number of oesophageal varices, size of oesophageal varices, and presence of any red signs over the varices were noted in the case record form. Then the stomach was surveyed for the presence of portal hypertensive gastropathy and gastric varix. If portal hypertensive gastropathy is present, then the grade of portal hypertensive gastropathy was noted. If the gastric varices were present, then the site of varices and type of varices were noted. All information was noted in the case record form. A detailed history of each patient was taken and preset data was filled for every patient. History of jaundice, blood transfusion, operation, dental procedure, haemetemesis and melaena, endoscopic treatment of oesophageal varices, ingestion of hepatotoxic drugs and alcohol were inquired for every patient. Physical examination was done systematically. All data were entered into a personal computer, thoroughly checked for any possible error, and then processed and analyzed by the SPSS program (version 20.0). The significance of the test was tested by the chi-square test. p-value of <0.05 was taken as statistically significant. Correlation analysis was done by the Pearson correlation test.

IV. RESULT

In this study, the age range of the cirrhotic patients was 15-70 years, and the mean age was 39, 61±14.54 years. Male was 42 (84%) and female were 8(16%) in number. The number of patients in child Grade-B was 11(22%) and child Grade-C was 39(78%). The SAAG values of cirrhotic patients are divided into three groups. The first group was 1.1-1.49 gra/dl, the number of patients in this group was 15 (30%). The second group was 1.5-1.99 gm/dl; the number of patients was 9 (18%). The third group was >2 gra/dl, the number of patients was 26 (52%). In this study,

oesophageal varices were present in 49 (98%) patients; small-sized in 18 (36%) patients, medium-sized in 15(30%) patients, large-sized in 16 (32%) patients. Red signs over oesophageal varices were present in 12 (24%) patients. Gastric varices were present in 2 (4%) patients. Portal hypertensive gastropathy was present in 44 (88%) patients; mild grade in 23 (46%) patients and severe grade in 21 (42%) patients. Both oesophageal varices and portal hypertensive gastropathy were present in 43 patients (86%). In this current study, we observed, in SAAG 1.1-1.49 gm/dl, 14 of 15 patients (93.3%) had oesophageal varices and 13 of 15 patients (86.6%) had gastropathy. 9 of 9 patients (100%) had oesophageal varices and 7 of 9 patients (77.7%) had gastropathy, in SAAG 1.5-1.99 gm/dl. In SAAG>2 gm/dl, 26 of 26 patients (10G%) had oesophageal varices and 24 of 26 patients (92.3%) had gastropathy, a Total of 49 of 50 patients (98%) had oesophageal varices and 44 of 50 patients (88%) had gastropathy. Besides these, in the SAAG group 1.1-1.49 gm/dl, only 13.32% of patients had red signs, in SAAG group 1.5-1.99 gm/dl, no patient had red sign whereas and in the SAAG group >2 gm/dl, 38.46% patients had red signs over the varices. On the other hand, gastric varices were present in only 2 patients. One of them had a SAAG value at 1.50-1.99 gm/dl and the other had a SAAG value at >2 gm/dl. In this study, there were three SAAG groups. In SAAG group 1.10-1.49gm/dl, 53.33% patients had a mild grade of PNG and 33.33% of patients had a severe grade of PHG. In the SAAG group 1.50-1.99 gm/dl, 44.44% patients had mild grades and 33.33% had a severe grade of PNG and in the SAAG group >2 gm/dl, 42.30% had mild grade and 49.99% had a severe grade of PHG. But there had not been any significant correlation among the groups regarding SAAG values and PHG grades because the p-value was greater than 0.05.

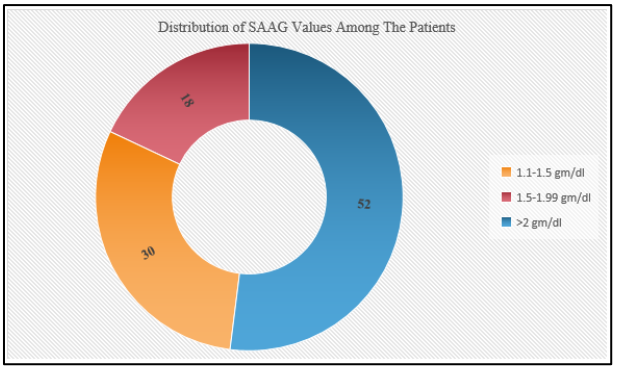

Fig-I: Distribution of SAAG values among the participants (N=50)

Table-I: Endoscopic profile of the participants (N=50)

Endoscopic findings	Size/grade	n (%)
OesophagealVarices (OV)	Absent	01(2.0)
	Small	18(36.0)
	Medium	15(30.0)
	Large	16(32.0)
OVRS	Present	12(24.0)
	Absent	38(76.0)
Gastric Varices	Present	2(4.0)
	Absent	48(96.0)
PHG	Absent	6(12.0)
	Mild	23(46.0)
	Severe	21(42.0)
Both OV and PHG	Present	43(86.0)

OVRS: Oesophageal variceal red signs, PHG: Portal hypertensive gastropathy

Table-II: Distribution of oesophageal varices and portal hypertensive gastropathyin different SAAG group (N=50)

SAAG (gm/dl)	Patient (n)	Oesophageal varices		PHG	
		Present	Absent	Present	Absent
		n (%)	n (%)	n (%)	n (%)
1.1-1.49	15	14 (93.3)	1 (6.6)	13 (86.6)	2 (13.3)
1.5-1.99	9	9 (100.0)	0 (0.0)	7 (77.7)	2 (22.2)
>2	26	26 (100.0)	0 (0.0)	24 (92.3)	2 (7.6)
Total	50	49 (98.0)	1 (2.0)	44 (88.0)	6 (12.0)

Table-III: Correlation between SAAG group and grades of portal hypertensive gastropathy cirrhotic patient (N=50)

SAAG (gm/dl)	Patient (n)	PHG grades			r	p-Value
		absent	mild	severe		
		n (%)	n (%)	n (%)		
1.1-1.49	15	2 (13.33)	8 (53.33)	5 (33.33)	0.139	0.334^{ns}
1.5-1.99	9	2 (22.22)	4 (44.44)	3 (33.33)		
>2	26	2 (7.69)	11 (42.3)	13 (49.99)		
Total	50 (100%)	6 (12.0)	23 (46.0)	21 (42.0)		

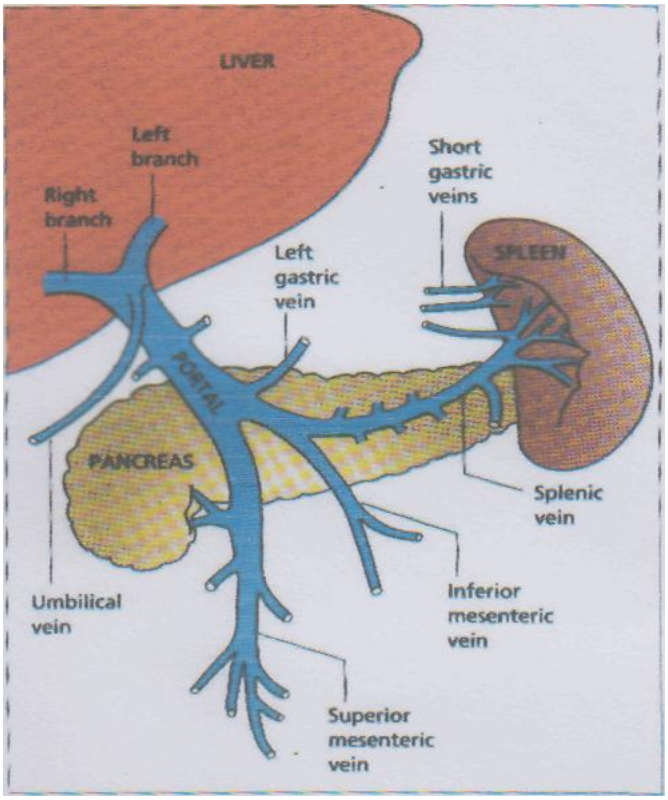

Fig-2: The anatomy of the portal venous system

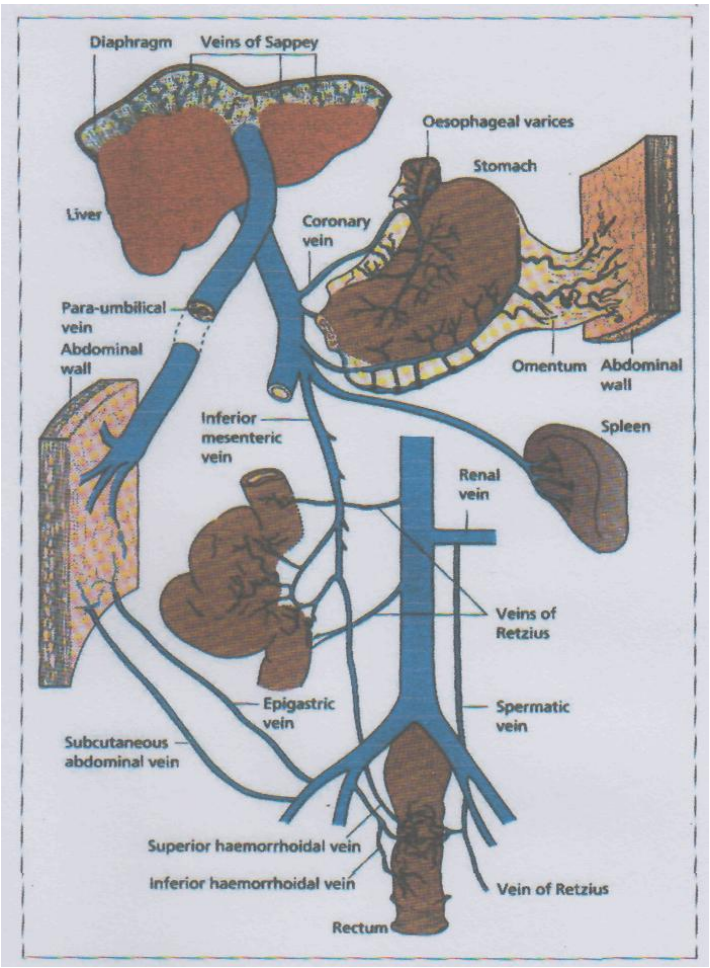

Fig-3: The sites of the portal-systemic collateral circulation in cirrhosis of the Liver

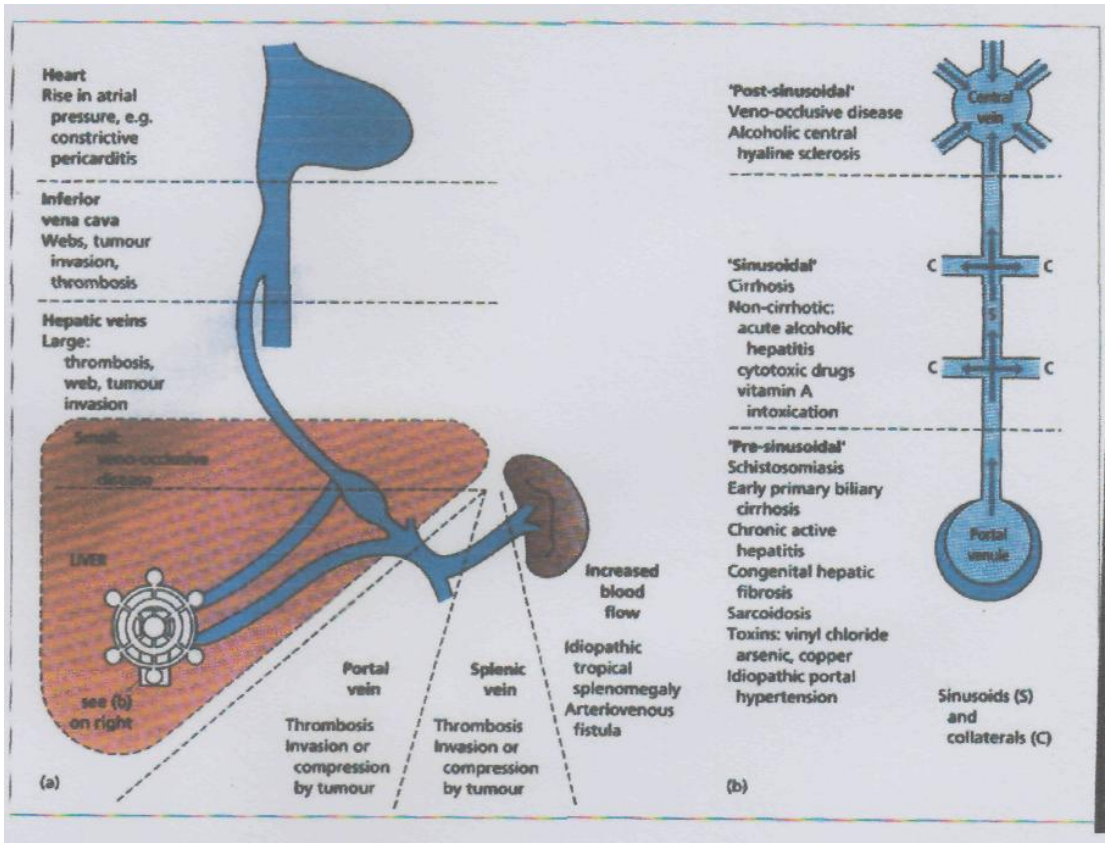

Fig-4: Aetiology of portal hypertension (a) Pre- and post- hepatic, (b) Intrahepatic

V. DISCUSSION

The aim of this study was to assess the correlation between serum ascites albumin concentration and endoscopic parameters of portal hypertension in chronic liver disease. Cirrhosis of the liver is a medical problem in Bangladesh. Portal hypertension is a complication of cirrhosis of the liver. It is manifested by the development of ascites, oesophageal varices, gastric varices, and portal hypertensive gastropathy. Serum ascites albumin gradient (SAAG) is a physiological clinical diagnostic tool for the evaluation of ascites. High SAAG (>1.1 gm/dl) indicates the presence of portal hypertension which is detected by observing portal hypertensive changes in the upper gastrointestinal tract. Direct portal pressure measurement is an invasive and cumbersome procedure. So minimal invasive endoscopy helps us to detect the development of portal hypertension by observing changes in the upper gastrointestinal tract. There is a correlation between SAAG and portal hypertensive changes in the upper gastrointestinal tract in cirrhotic patients with ascites [4]. Several studies have been conducted in the different parts of the world on alcoholic cirrhosis but studies on nonalcoholic cirrhosis are scanty. The study was done in the department of Hepatology, BSMMU on nonalcoholic cirrhosis. Hoefs et al. 1983 [4] studied 56 patients, 52 of whom were alcoholic cirrhosis. An excellent correlation was found between SAAG and portal pressure ($r=0.73$, $p<0.05$). In this study, a numeric formula was established for the first time. Kajani et al. in 1990 [5] investigated correlations between SAAG and portal pressure in alcoholic cirrhosis and cirrhosis due to nonalcoholic causes separately. In this study, a correlation was found between SAAG and portal pressure($r=0.62$) or, oesophageal varices in alcoholic cirrhosis but no significant correlation was found between SAAG and portal pressure($r=0.39$) while the correlation between SAAG and variceal grades was found to be weaker($r=0.02$) in nonalcoholic cirrhosis. In our study, there were three SAAG groups. In SAAG group 1.10-1.49gm/dl, 53.33% of patients had mild grades of PHG, and 33.33% of patients had a severe grade of PHG. In the SAAG group 1.50-1.99 gm/dl, 44.44% of patients had mild grade and 33.33% had a severe grade of PHG and in the SAAG group >2 gm/dl, 42.30% had mild grade and 49.99% had a severe grade of PHG. But there had not been any significant correlation among the groups regarding SAAG values and PHG grades because the p-value was greater than 0.05. These findings are comparable with that of some other studies [1]. In our study, a total 50 patients of cirrhosis of various aetiology were included. A correlation was studied between SAAG and portal hypertensive changes in the upper gastrointestinal tract like oesophageal varices, gastric varices, and gastropathy. Oesophageal varices were present in 49 patients (98%), of the small-sized in 18 patients (36%), medium-sized in 15 patients (30%), large-sized in 16 patients (32%). Red signs over the oesophageal varices were present in 12 patients (24%). Gastric varices were

present in only 2 patients (4%). All these findings are comparable with findings of the previous studies done in other centers in the world. Hoefs first introduced SAAG, reporting that SAAG can reflect portal vein pressure and improve the accuracy of ascites identification [4]. Thereafter, several other investigators demonstrated the superiority of SAAG (11g/L) for distinguishing portal hypertensive ascites and non-portal hypertensive ascites. More recently, Rector et al. [1]. Reported an excellent correlation between SAAG and portal pressure [1]. Demirel et al. also suggested that patients with alcoholic cirrhosis with SAAG >20 g/L showed a significant increase in esophageal varicose veins [5]. Forhad Hossain MdShahed et al. [8] found a correlation between SAAG and esophageal varicose gastric varicose veins. A total of 50 patients with cirrhosis of various etiologies were included. The relationship between SAAG and portal hypertensive changes in the upper gastrointestinal tract were examined, including esophageal varices, gastric varices, and gastropathy.

VI. CONCLUSION AND RECOMMENDATIONS

Cirrhosis of liver is a medical disease in Bangladesh. The unavoidable complication of cirrhosis of liver is development of portal hypertension that leads to development of oesophageal varices, gastric varices and portal hypertensive gastropathy. So, it is important to detect portal hypertensive changes in upper gastrointestinal tract as early as possible, thereby prophylaxis and treatment of portal hypertension keep cirrhotic patients' symptom free life. It was shown in this study that, oesophageal varices were present in 49 patients, sensitivity was 98%; portal hypertensive gastropathy in 44 patients, sensitivity 88% and both oesophageal varices and gastropathy in 43 patients, sensitivity 86%. So high SAAG value can be used as an indicator of presence of portal hypertensive changes especially oesophageal varices and gastropathy in upper gastrointestinal tract. A weak positive correlation was found in this study between SAAG values and grades of oesophageal varices(r=0.358, p=0.011) and gastropathy (r-0.139, p=0.33) but no correlation was found between SAAG and gastric varices (P=O.4).

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