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Research Article

DETERMINATION OF ESOPHAGEAL VARICES IN PATIENTS OF CIRRHOSIS WITH SERUM-ASCITES ALBUMIN GRADIENT

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Abstract:

Objective: To determine commonest SAAG “serum-ascites albumin gradient” value in cirrhotic disease patients having esophageal varices and to evaluate serum albumin and serum ascites values.

Study Design: A cross sectional study.

Place and Duration: In the East Medical Ward of Mayo Hospital Lahore for one year duration from July 2017 to July 2018.

Methods: This section was performed on hundred patients of cirrhosis with ascites to calculate the SAAG level in ascitic and serum fluid, SAAG value (≥ 1.1 g / dl) was examined and high SAAG value ≥ 1.1 g / dl when <1.1 g / dl to classify the esophagus varices SAAG under. All patients were done with upper GI endoscopy.

Results: Of the hundred patients, 62 were male and 38 were female. The SAAG was 1.099 ± 0.62 . In 87 patients, Esophageal varices (EV) were noted and were not present in thirteen subjects. The esophageal varices grading, SAAG degree ($p < 0.01$) and correlation coefficient $r = 0.56$ ($p < 0.01$) stressed significant correlation. Using the ROC curve, the SAAG value, ie, 1.64 ± 0.014 g / dl, was an accurate indication of the EV onset; for the highest predictive value, the cut-off points were 96% negative and 98% positive.

Conclusion: In cirrhosis patients having ascites, the onset of EV is only related with SAAG, and the EV size is associated with SAAG degree. SAAG value $\geq 1.65 \pm 0.014$ g / dl is helpful tool in predicting the onset of EV in subjects having cirrhosis with ascites.

key words: Esophageal varices, SAAG, cirrhosis, portal hypertension.

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INTRODUCTION:

Chronic liver disease is a common disease in South Asia. Increased prevalence should be assessed and updated from time to time for HbsAg and HCV¹⁻³. In a region-based study, 31% of the patients had antibodies against the hepatitis B nucleus and 4.5% had a positive HBsAg antigen. Chronic liver disease associated with HCV also increased in Pakistan and additional available data show that approximately 59-71% of CLD patients are positive for HCV antibodies. Patients with CLD eventually lead to cirrhosis of liver and related problems such as portal hypertension⁴. The esophageal varices development is one of the problems of portal hypertension⁵. Chronic upper GI bleeding in patients with cirrhosis is common due to portal hypertension observed in between 30% and 40%. Western studies show that the upper part of the variceal hemorrhage varies between 17 and 57%, but in our cases (5 to 10%). In the past, on portal hypertension Baveno international consensus argued that total cirrhosis patients for varicose veins must be checked by diagnostic endoscopy⁶. Investigation of gastroesophageal varices (VEG) in patients with cirrhosis increases the cost and implies an approved degree of invasiveness and discomfort for patients. Some retrospective studies have shown a possible non-invasive esophageal symptom in cirrhotic patients such as SAAG and serum albumin levels⁷. The serum albumin-albumin gradient (SAAG) has resulted in a series of studies as an oblique marker in the approach of portal hypertension and its problems⁸. For SAAG participation, the calculation, which evaluates the concentrations of serum albumin and ascitic liquid samples, was kept on a parallel day and the ascitic removed the value of the liquid from the serum values⁹. If SAAG was equal to or more than 1.1 gm / dl (11 mg / L), the patient covered portal hypertension with this correction reducing the risk of bleeding (97%)¹⁰. It is likely that the SAAG prediction is still present in a small business laboratory without assumption and is available for new ways to recognize patients with a high risk of bleeding.

MATERIALS AND METHODS:

This cross sectional study was held in the East Medical Ward of Mayo Hospital Lahore for one year duration from July 2017 to July 2018. In this study, 100 cirrhotic patients with ascites were included, and all cases were diagnosed based on a biopsy report of cirrhosis and previous biopsy because of previous ultrasonography, biochemical and clinical

examinations. Exclusion criteria included all patients who had already been treated with drugs, all patients used as a prophylactic drug for variceal bleeding, patients who were not included in this study and had previously had portasystemic anterior anastomosis, portal vein thrombosis, hepatocellular carcinoma and causes rather than cirrhosis. The cirrhosis examination was based on previous liver biopsy or on ultrasonography, clinical and biochemical observations. The full protocol of the study was explained to patients with written consent was taken. All subjects had complete physical examination, followed by a blood sample, ie, liver function test, complete blood count, liver biochemistry, prothrombin time, serum albumin and viral profile (Anti-HCV, HBsAg). Under aseptic measures diagnostic paracentesis was done within 30 minutes after blood samples were taken. The ascitic fluid was sent to the main laboratory for a detailed report in a disposable 10 cc syringe. Serum ascitic albumin gradient was recorded. To evaluate the hepatic parenchyma, splenomegaly, and echogenic tissue of the ascites, abdominal ultrasound was done. All patients were done with flexible upper gastrointestinal endoscopy in the Medicine Department.

The classification system of DeFranchis for the classification of esophagus varies with: F1, F1 as small flat varicose veins, F2 as enlarged convoluted varices covering less than 1/3rd of the lumen. F3 consists of large, spiral-shaped varicose veins covering above of 1/3rd of the lumen. All data taken from 100 cases were entered into SPSS version 17.0 statistical program and analyzed. To compare the rates between EV and ascitic serum albumin gradient X2 test was applied. A regression analysis was performed to evaluate the positive and negative correlation between SAAG and the esophageal varices degree. $P < 0.05$ was considered as significant statistic.

RESULTS:

Of 100 patients, male were 62 (62%) and female were 38 (38%). Mean age was SD (44.59 ± 13.55). The causes were HCV in 53 patients (53%), 34 patients (34%) HBV, HBV and HCV infection in 5 patients (8%) and no virus in 5 patients (5%). They were able to detect HBV and HCV markers. Mean albumin + SD serum level (2.87 ± 0.35) (2.09 to 3.6 g / dl), ascitic albumin level (0.87 ± 0.51) (0.2 to 2.3 g / dl) and mean value + SD SAAG (2.01 ± 0.52) SAAG value was 1.10 g / dl in 3 patients (3%), SAAG b / w intervals in 11 patients (11%) (10) and SAAG range

b / w in 33 patients (33%). and 1.99 g / dl) in 29 patients (29%) had SAAG range b / w (1.90 and 2.50 g / dl) and 24 (24%) patients were SAAG ranks (87 patients (87 2.5 g / dl EV) (13 %) and 13 patients (13%).

In 24 patients, SAAG values were ≥ 2.5 g / dl, 5 of them (20.8%) had varicose veins of grade F1, 7 (29.2%) had varicose veins with grade F2 and 12 (50%) had varicose veins F3. (Table I).

TABLE I: DISTRIBUTION OF THE CASES ACCORDING TO DEGREE OF SAAG & GRADE OF EV

SAAG Group	No. of Patients	Esophageal varices (Grades)			
		Absent	F1	F2	F3
< 1.10	3	3 (100%)	-	-	-
1.10 - 1.49	11	7 (63.6%)	4 (36.4%)	-	-
1.50 - 1.99	33	3 (9%)	5 (15.2%)	15 (45.5%)	10 (30.3%)
2.00 - 2.49	29	-	7 (24.1%)	16 (55.2%)	6 (20.7%)
≥ 2.5	24	-	5 (20.8%)	7 (29.2%)	12 (50%)
Total	100	13	21	38	28

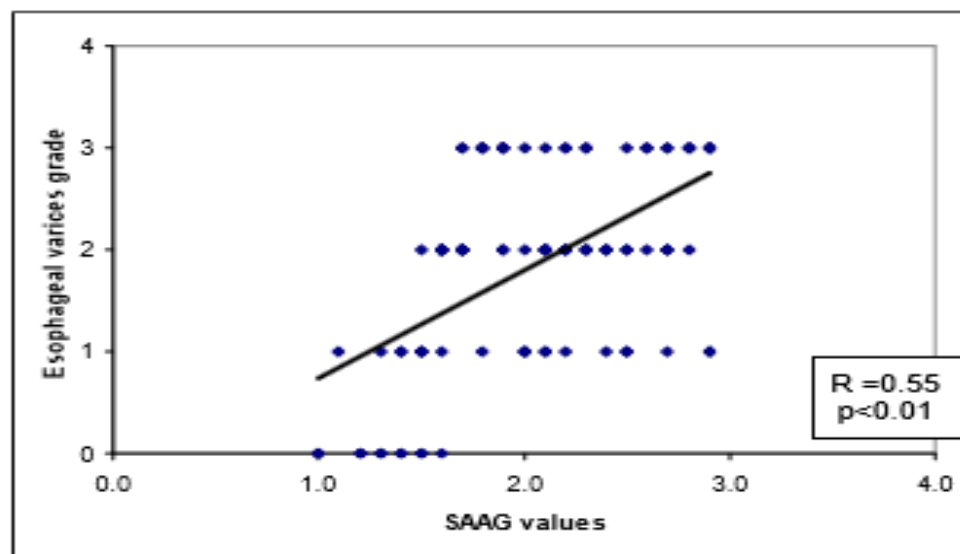
Grade of esophageal varices were significantly associated with the level of SAAG ($p < 0.01$)

The mean SAAG of the 38 patients was 2.1 g / dl \pm 0.38 (SD), and it was found that 28 patients with varicose veins of F3 grade had an average of 2.3 g / dl \pm 0.44 (SD) of SAAG. (Table II)

TABLE II: SAGG VALUE ACCORDING TO ESOPHAGEAL VARICES

Esophageal Varices	No. of Patients	SAAG Mean \pm SD
Absent	13	1.3 \pm 0.20
F1	21	2.0 \pm 0.54
F2	38	2.1 \pm 0.38
F3	28	2.3 \pm 0.44
P-value		0.001

Using the receiver operating characteristics (ROC) curve, $\geq 1.65 \pm 0.014$ g / dl SAAG range was an excellent indicator of EV onset; Breakpoints for the highest estimated range.

FIGURE I: CORRELATION BETWEEN SSAG VALUES AND GRADES OF ESOPHAGEAL VARICES**DISCUSSION:**

The esophageal varices prevention primary finding in subjects with cirrhosis are critical problems. It is suggested that cirrhosis patients should be checked for varices at the time of diagnosis¹¹⁻¹². However, in all our hospitals, healthcare system lacks endoscopic facilities so in our configuration this approach is not recommended. SAAG is the indirectly less invasive parameters that detect the esophageal varices presence as a sign of portal hypertension¹³. Several analysis highlight the SAAG value in determining portal hypertension and thus predicting the esophageal varices presence. According to Hoefs et al 56 patients showed much better association between SAAG and portal hypertension with $r = 0.73$ and $p = 0.05$. The Rector and others found the same results in their study of 18 patients with cirrhosis due to alcohol¹⁴. Demirel et al. Conducted a study in 45 patients to determine the EV and SAAG ratio b / w patients with cirrhosis. In all patients who supported my study, it was found that the SAAG values were above 1.1 g / dl, and the sum of patients with SAAG higher than 2.0 had EV, but SAAG and EV. Masroor et al.'s studies in Karachi have shown a strong relationship between SAAG grade and classification of esophageal varices¹⁵. In our study, EVAG, SAAG value was between 1.10-1.49 g / dl, in 4 (28.6%) of 11 patients and in SAAG value of 1.50-1.99 g / dl. (100%) were 2.0-2.49g / dl and 24 of 24 (100%) SAAG values were 2.5 g / dl. These values indicate that as the SAAG grade increases, the likelihood of esophageal varices increases. Therefore, the SAAG degree is related directly to the esophageal varices' presence.

CONCLUSION:

Patients with EV-onset cirrhosis and ascites associated with high SAAG value (≥ 1.1 g / dl) only. The formation and size of EV in ascitic patients are directly related to the degree of SAAG and the SAAG value is $\geq 1.65 \pm 0.014$ g / dl.

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