



## Original Article

# Association between Serum-Ascites Albumin Gradient and Esophageal Varices in Patients of Cirrhotic Ascites

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### Abstract

**Background:** Serum-Ascitic Albumin Gradient (SAAG) can identify ascites related to portal hypertension. Esophageal varix (EV) is another feature of portal hypertension in cirrhosis. This study was done to find out the relation between SAAG and esophageal varices.

**Materials and methods:** It was a cross-sectional descriptive study of 50 cases of cirrhotic ascites. SAAG was measured and upper gastrointestinal endoscopy was done for esophageal varices.

**Results:** Among 50 cases, mean SAAG value 1.77 ( $\pm 0.45$  SD). Esophageal varices were found in 43(86%) cases. All cases with SAAG value  $>2.0$  gm/dl had EV. A significant statistical association was found between level of SAAG and the presence of esophageal varices ( $p=0.01$ ).

**Conclusion:** In patients of cirrhosis, the level of SAAG has positive association with the esophageal varices. This finding may permit us to use SAAG as a preliminary parameter of esophageal varices (EV) especially where endoscopy facilities are not available.

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### Introduction

Patients with ascites may accompany several diseases but approximately 85% have cirrhosis and 15% have non-hepatic causes of fluid retention<sup>1</sup>. Most common cause of ascites in cirrhosis of liver is portal hypertension (PHTN)<sup>2, 3</sup>. Esophageal varix is a manifestation of portal hypertension (PHTN). Esophageal varices are present in about 45% of compensated cirrhosis and in 80% of uncompensated cirrhosis with ascites<sup>3,4</sup>. Traditionally ascites are two types: exudative and transudative (ascitic fluid total protein  $\geq 2.5$  gm/dl in exudates and  $< 2.5$  gm/dl in transudates)<sup>4</sup>. This classification, however, is unable to correctly identify the etiological factors responsible for its

causation, as ascitic fluid total protein (AFTP) is  $>3$  gm/dl in 30% of cirrhotic ascites. Different studies show that SAAG can be used as a parameter of PHTN to differentiate ascitic fluid into two categories. When SAAG is  $\geq 1.1$  gm/dl indicates ascites with portal hypertension and  $< 1.1$  gm/dl is unrelated to portal hypertension<sup>5, 6</sup> with 97% accuracy<sup>7, 8</sup>. This study was designed to find out association between SAAG and of esophageal varices in order to use of SAAG as a preliminary parameter for presence of esophageal varices in cirrhotic patients.

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## Materials and Methods

This is an observational and cross-sectional study of 50 diagnosed cases of cirrhotic ascites attending in Medicine unit of Rajshahi Medical College Hospital from January 2012 to December 2012. Cirrhotic ascites cases were diagnosed clinically and by ultrasonographically. Esophageal varices were detected by Upper GI endoscopy and classified into three grades according to Japanese classification<sup>8</sup>. Albumin concentration in serum and ascitic fluid was measured simultaneously. The SAAG was calculated by subtracting ascitic fluid albumin from serum albumin and was classified into three ranges: 1.10-1.49 gm/dl; 1.50-1.99 gm/dl and >2.0 gm/dl to find out association with grades of EV. Data were analyzed by SPSS-16 (statistical package for social science). Chi-square test was applied to see association between variables and *p* value <0.05 was considered as statistically significant.

## Results

Mean age was 53.4 ( $\pm$ 11.98 SD) yrs. Male cases were 38 (76%) and female were 12 (24%). Male-female ratio was 31:1. Esophageal varices were found in 43 cases (86%). All the patients had SAAG value >1.10 gm/dl. Mean SAAG value was 1.77 ( $\pm$ 0.45 SD). Among 43 cases of esophageal varices 8 (18.5%) cases had grade-I, 17 (39.5%) cases had grade-II and 18 (42%) cases had Grade-III esophageal varices (table-1). In SAAG value between 1.10gm/dl and 1.49gm/dl, 8 out of 13 cases had EV; in SAAG value between 1.50gm/dl and 1.99gm/dl, 18 out of 20 cases had EV and in SAAG value >2.0gm/dl all of 17 cases had EV. A significant statistical correlation was found between level of SAAG and the presence of EV ( $p=0.009$ ). It was remarkable that all cases with SAAG value >2.0gm/dl had EV and it was observed that the more the level of SAAG the more the presence of EV (Table 2). So there was a positive correlation with the presence of EV and the level of SAAG.

## Discussion

Esophageal varices may cause life-threatening bleeding. Mortality rate is still above 45% in those with poor liver function (Child-Pugh grade-C patients)<sup>9</sup>. The early detection of esophageal

varices is critical for prevention of bleeding. Endoscopy is still advocated for accurate diagnosis of bleeding from esophageal varices. SAAG is less costly, feasible and minimally invasive method that indicates the presence or absence of portal hypertension with 97% accuracy<sup>8</sup> especially where endoscopy facilities are not available or contraindicated. In this study, EV was present in 61.5%, 90% and 100% cases when SAAG level was between 1.10 – 1.49 gm/dl, 1.50 – 1.99 gm/dl and >2.0 gm/dl respectively (table 2). It was observed that the more value of SAAG the more the presence of EV. Significant statistical association was found between level SAAG and the presence of esophageal varices ( $p=0.009$ ). This study had also showed that various grade of esophageal varices were present within different level of SAAG. The more the gradient (SAAG), the more the possibility of grades of EV. So grade of EV numerically increases with level of SAAG. Torres et al<sup>10</sup> found that the presence of EV in patients with cirrhotic ascites was directly related to the degree of SAAG ( $p=0.049$ ). Another study by Gurubacharya et al<sup>11</sup> in Nepal showed that the degree of SAAG was directly related to the presence of EV ( $p=0.037$ ) These finding is also consistent with this study.

## Conclusion

In patients of cirrhosis, the level of SAAG has positive correlation with the presence of esophageal varices. It can assist clinicians in determining the urgency of care and can help to identify patients who need early referral for endoscopy especially where this facility are not available.

Table-1: Grading of esophageal varices among the patients.

Grade of Esophageal varices	No. of patients	Percentage (%)
Grade-I	8	18.5
Grade-II	17	39.5
Grade-III	18	42
Total	43	100

Table-2: Level of SAAG and esophageal varices

Level of SAAG	Esophageal varices		Total
	Present No. of patients (%)	Absent No. of patients (%)	
1.10— 1.49 gm/dl	8 (61.5%)	5 (38.5%)	13 (100%)
1.50— 1.99 gm/dl	18 (90%)	2 (10%)	20 (100%)
>2.0 gm/dl	17 (100%)	0	17(100 %)
Total	43	7	50

$X^2=9.49, df=2, p=0.009$

### References

1. Heidelbaugh, J.J. &Sherbondy, N 2006, 'Cirrhosis and chronic liver failure (part-ii) complication and treatment', *Am Fam Physician*, vol. 74, pp. 770-76.
2. Collier, JD &Webstar, G 2010, 'Liver and biliarytract disease', In NR Colledge, B R Walker, SH Ralston (eds), *Davidson's Principal & Practice of Medicine*, 21<sup>st</sup>edn, Churchill Livingstone. pp. 936-42.
3. Garcia-Tsao, G 2007, 'Cirrhosis and its sequelae', In L Goldman, D, Ansiello (eds), *Cecil Medicine*, 23<sup>rd</sup>edn, Saunders Elsevier, Philadephia. p.1140-41.
4. Schepis F, Camma C, Niceforo D 2001, 'Which patients with cirrhosis should undergo endoscopic screening for esophageal varices detection?' *Hepatology*, vol. 33, pp.333-8.
5. Hoefs JC 1983, 'Serum protein concentration and portal pressure determine the ascites fluid protein concentration in patients with chronic liver disease', *J Lab Clin Med*, vol.102.
6. Rector WG, Reynolds TB 1984, 'Superiority of the serum-ascites albumin difference over the ascites total protein concentration in separation of 'transudative' and 'exudative' ascites', *Am J Med*, vol.77,pp. 83-5.
7. Robert MG &Rajapaksa R 2008, 'Abdominal Swelling and Ascites', In : Fauci AS, Braunwald E, Kasper DL et al (eds) *Harrison's Principles of Internal Medicine*, 17<sup>th</sup>edn, McGraw Hill, p.266
8. Runyon BA 1993, 'Ascites and spontaneous bacterial peritonitis', In: Sleisenger MH, Fordtran JS, (eds), *Gastrointestinal diseases: pathophysiology, diagnosis, management*. Vol II, W.B. Saunders Company; Philadelphia, pp. 1977–2003.
9. Collier JD and Webstar G. Liver and biliarytract disease. In: Nicki RC, Brian RW, Stuart HR eds. *Davidson's Principal & Practice of Medicine*. 21<sup>st</sup> Edition. Churchill Livingstone; 2010. p.936-42.
10. Torres E, Barros P, &Calmet F 1998, 'Correlation between serum-ascites concentration gradient and endoscopic parameter of portal hypertension', *American Journal of Gastroenterology*, vol.98, pp. 2172-78
11. Gurubacharya DL, Mathura KC, Karki DB 2005, 'Correlation between serum ascites albumin concentration gradient and endoscopic parameter of portal hypertension', *Kath Uni Med J*, vol. 3, no. 4, issue 12, pp. 327-33.

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