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ORIGINAL ARTICLE

Prediction of oesophageal varices in cirrhotic patients by serum ascites albumin gradient.

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ABSTRACT

Background and aim of the work: Portal hypertension (PHT) and the eventual Oesophageal varices is one of the awful complications of liver cirrhosis. Bleeding from OV is a real leading cause of death. This study aims at evaluating serum ascites albumin gradient (SAAG) as a predictor of OV. **Patients and methods:** A descriptive cross-sectional study was conducted on 105 males and 26 females where they were divided into two main groups and sub groups according to the presence and size of OV. All participants were subjected to full history taking, thorough clinical examination, laboratory investigations, liver function tests, complete blood count, kidney function tests, hepatitis markers: HCV-Ab (anti hepatitis c virus antibody) hepatitis B surface antigen (HBs-Ag), abdominal ultrasonography and abdominal diagnostic paracentesis with calculation of SAAG. **Results:** there is highly significant increased SAAG in cirrhotic patients with OV than those without. Moreover, SAAG increases significantly with the grade of OV. **Conclusion:** Serum ascites albumin gradient (SAAG) could be used as a non-invasive predictor for the presence of OV in cirrhotic patients with ascites. Moreover, SAAG value increased significantly with the progression of OV.

Key words: esophageal varices, serum ascites albumin gradient (SAAG), portal hypertension-cirrhosis, HCV, ascites.

INTRODUCTION

Liver cirrhosis is considered to be the end stage of any chronic liver disease, resulting in portal hypertension, which is associated with development of ascites, and esophago-gastric varices. Oesophageal varices (OV) are present at diagnosis in approximately 50% of cirrhotic patients, being more common in Child-Pugh class C patients as compared with Child- Pugh class A patients. Once varices form, they expand from small to large at a rate of 5-12% per year and bleed at a rate of 5-12% per year [1].

Poor results from variceal bleeding with mortality rates between 30-60% were reported [2]. Early diagnosis of varices before the first bled is essential as studies of primary prophylaxis obviously demonstrate that the danger of variceal hemorrhage can be diminished from 50% to about 15% for large esophageal varices [3]. Current guidelines, consequently, recommend that all cirrhotic patients should be screened for

varices at diagnosis of cirrhosis, with follow up every 2-3 years for patients without varices (depending on liver diseases severity) and 1-2 years for patients with small varices, to assess for enlargement of varices and requirement of prophylactic treatment [4]. Upper GI endoscopy remains the gold standard for screening varices development, however this procedure has many limitations [5].

There is conflicting evidence concerning the interobserver agreement for endoscopic analysis of variceal presence, grade, or presence of red signs [6]. This approach places a huge burden and cost to endoscopy units and require patients to have repeated unpleasant procedures. If it were possible to predict esophageal by noninvasive means, this would restrict endoscopy to the population considered to be at most risk and decrease the number of endoscopies required. Such a screening test should be simple, rapid, reproducible and low cost [7].

Many studies have been performed trying to predict esophageal varices by noninvasive means such as evaluating clinical signs and/ or variables related to liver function, liver fibrosis, portal hypertension and hypersplenism. However, no study achieved a sufficiently high level of significance to warrant the broad use of such non- invasive tests [4,8].

Serum-ascites albumin concentration gradient (SAAG) is a good biochemical marker and a better discriminator of portal hypertension (PHT) than ascites protein concentration. Patients with gradients of >1.1 gm/dl have PHT, while those with gradients <1.1 do not, with accuracy rate 97% [9]. Indeed, SAAG is now considered a useful physiological and clinical tool in the work-up of ascites. (high-albumin gradient) (>1.1gm/dl) or low-albumin gradient (<1.1gm/dl) have replaced the terms transudative and exudative in the description of ascites in all recent publications [10].

SAAG was proposed to be an element deciding the level of (PHT) and the prognosis in the patients with alcoholic cirrhosis [11]. Because of the conflicting results, correlation between these two findings SAAG and OV in patients with cirrhosis has attracted attention and needs extensive clarification.

AIM OF THE WORK

The aim of this study is to evaluate the role of serum ascites albumin gradient for prediction of esophageal varices in hepatitis C virus (HCV) cirrhotic patients with ascites.

PATIENTS AND METHODS

This is a descriptive cross-sectional study that was carried out in Tropical Medicine Department, Zagazig University Hospitals, during the period from August 2018 to January 2019. The study comprised 131 HCV cirrhotic patients with ascites, during this period. They were 105 males and 26 females. Their age ranged from 45 to 76 years.

Patients were divided into two groups:

- **G I:** comprised 20 cirrhotic patients without O.V.
- **G II:** comprised 111 cirrhotic patients with O.V who were furtherly

Classified according to upper GI endoscopy **findings into:**

- **G (II) a:** Comprised 55 cirrhotic patients with O.V grade 1.
- **G (II) b:** Comprised 38 cirrhotic patients with O.V grade 2.
- **G (II) c:** Comprised 11 cirrhotic patients with O.V grade 3.
- **G (II) d:** Comprised 7 cirrhotic patients with O.V grade 4.

A written informed consent was obtained from all patients and all procedures were approved by the Institutional Review Board (IRB).

The work has been carried out in accordance with the code of Ethics of the World Medication Association (Declaration of Helsinki) for studies involving humans.

Inclusion criteria:

HCV positive cirrhotic ascetic patients who had no past history of hematemesis and /or melena were included in the study.

Exclusion criteria:

Pregnant ladies, Patients who had cirrhosis and ascites other than hepatitis C virus, Patients with spontaneous bacterial peritonitis, Patients

with hepatorenal syndrome, Patients with chronic renal failure, Patients with hepatopulmonary syndrome, Patients with portal or splenic vein thrombosis, Patients with uncontrolled hepatic encephalopathy, Hemodynamic instability, Active or past history of alcohol use, Previous surgery for portal hypertension and /or splenectomy and Previous endoscopic treatments (band ligation, sclerotherapy) or trans jugular intrahepatic portosystemic shunt (TIPS).

Methods:

All patients were subjected to full history taking, thorough clinical examination and laboratory investigations including:

Liver function tests by using (Dimension Rx1 Autoanalyzer from siemens), Complete blood count by using (system xkx21 from Roche diagnosis), Kidney function tests: as serum creatinine, blood urea nitrogen by using (Dimension Rx1 Autoanalyzer from siemens), Hepatitis markers: HCV-Ab (anti hepatitis c virus antibody) by using (VITROS Anti-HCV Reagent Pack on the VITROS 3600 Immunodiagnostic System hepatitis B surface antigen (HBs-Ag) by using the VITROS Anti-HBc Reagent Pack on the VITROS 3600 Immunodiagnostic System, Abdominal ultrasonography, performed by esoate MYLab20Plus to assess liver size, the presence of focal lesions and liver texture: bright or coarse echo pattern. Chronic liver disease and cirrhosis are suspected by presence or absence of periportal thickening, diameter of the portal vein, presence or absence of porto-systemic collaterals, size of the spleen, irregularities of liver surface and gall bladder fossa and presence or absence of ascites and Child Score was calculated for all patients who were categorized into Child B or C according to modified Child Classification [12].

Endoscopic evaluation: all patients had upper gastrointestinal endoscopy performed by **pentax EPM 3500 video esophago-gastro-duodenoscope**. An experienced gastro-enterologist blinded to the patients clinical and laboratory data confirmed all endoscopic findings. Upper gastrointestinal endoscopy: performed to estimate presence of varices, grades of O.V, Fundal varices, Portal hypertensive gastropathy and any other relevant signs and Endoscopic grading was done according to **Thakeb et al.,** [13].

Grade I: small straight cords of varices continued to lower 1/3 of the esophagus, **Grade II:** moderate sized clubbed varices with well-defined areas of normal mucosa between them forming several distinct vertical cords and confined to lower third of esophagus, **Grade III:** gross varices extending into the proximal half of the esophagus, which are so large and tortuous, that normal mucosa may not be visible in between unless the esophagus is fully distended with air, **Grade IV:** varices are like those of grade III but with dilated capillaries on top or in bet and Abdominal diagnostic paracentesis was done for all patients under complete aseptic conditions through placing the patient supine at the edge of the bed, with the trunk elevated 45 degrees. Accessing to the peritoneal space is usually midline 3 to 4 cm below the umbilicus, halfway between the symphysis pubis and the umbilicus. Alternatively, the entry site can be in left or right lower quadrant between the umbilicus and anterior superior iliac spine or the patients flank depending on the location of the fluid as determined by percussion of fluid wave. Be sure to avoid old surgical scars since the bowel may be adherent to the abdominal wall. For diagnostic paracentesis, collect 50 ml of ascitic fluid, most of operants use the (Z-tract) method, avoid continuous suction [14].

Analysis of 20 cc of ascitic fluid using the standard technique for aspiration, and examined for:

Color, aspect and reaction, Albumin, lactate dehydrogenase (LDH) and glucose levels, Bacteriological examination: gram stain and culture, Cell count and differential and Culture and sensitivity.

Estimation of SAAG where blood sample and ascitic fluid sample were taken for laboratory investigations simultaneously or within 30 minutes [15]. SAAG was calculated by subtracting the albumin level in the ascitic fluid

from the albumin level in a serum sample (serum albumin - ascitic fluid albumin).

Statistical Analysis:

The collected data were computerized and statistically analyzed using SPSS program (Statistical Package for Social Science) version 24. Data were tested for normal distribution using the Shapiro Walk test. Qualitative data were represented as frequencies and relative percentages. Chi square test ($\chi 2$) and Fisher exact was used to calculate difference between qualitative variables as indicated. Quantitative data were expressed as mean ± SD. Independent T test was used to calculate difference between quantitative variables in two groups. One-way ANOVA F-test was used to calculate difference between quantitative variables in more than two groups. All statistical comparisons were conducted with significance level of P-value ≤ 0.05 indicates significant, p <0.001 indicates highly significant difference while, P> 0.05 indicates Non-significant difference. Correlation coefficient.The 95%CI: 95% confidence interval, Positive predictive value (PPV) and negative predictive value (NPV), Area under the ROC curve (AUC)

RESULTS

Out of 131 Hcv-positive cirrhotic patients (100%) who were classified according to upper GI endoscopy there was 20 cirrhotic patients without OV (Group 1)(15.3%) and 111 cirrhotic patients with OV (Group II) (84.7%) who were classified into 4 groups according to the grade of OV in which Group IIa included 55 patients with OV grade 1 ,group IIb included 38 patients with OV grade 2 , Group IIc included 11 patients with OV grade 3 and Group IId included 7 patients with OV Grade 4 .

There was no significant difference between the studied groups as regard sex and residence, while there was significant difference as regard age. Table (1)

There were significant differences between the studied groups as regard jaundice, Child-Pugh class and score, while there was no significant difference as regard the presence of ascites and lower limb edema. Table (2).

There was statistically significant difference between the studied groups as regard spleen diameter, the presence of splenomegaly and shrunken liver. While there was no statistically significant difference among the studied groups regarding portal vein (P.V) diameter. Table (3).

There was highly significant difference regarding SAAG values among studied groups. SAAG value increased significantly with the grade of OV. Table (4).

There was significant positive correlation between SAAG with total bilirubin, direct bilirubin, serum albumin, C- reactive protein, INR and age. among patient with O.V; while There was significant positive correlation between SAAG and total bilirubin and direct bilirubin, age and child score among patients without OV. In addition, there was highly significant negative correlation between SAAG and PLT, ALT, AST, Hb and albumin in ascitic fluid. among patients with OV; while there was highly significant negative correlation between SAAG and albumin in ascites among patients without OV. Table (5).

The area under the curve (AUC) is (0.985). Receiver operating characteristic (ROC) curve showed that SAAG at a cut off value ≥1.3 gm/dl could predict the presence of esophageal varices with 100% specificity, 100% PPV, 90.09% sensitivity and 64.5% negative predictive value (NPV). Table (6).

Table 1. Demographic Findings among the studied groups.

		Group I N=20	Group IIa N=55	Group IIb N=38	Group IIc N=11	Group IId N=7	Total N=131	Test	P
Age, years*		**59.8 ± 6.5**	**58.1 ± 8.4**	**62.1 ± 7.9**	**58.9 ± 4.3**	**65.7 ± 5.4**	**60 ± 7.8**	**2.7**	**0.033**
Sex	**Female**	**4 (20.0%)**	**15 (27.3%)**	**7 (18.4%)**	**0 (0.0%)**	**0 (0.0%)**	**26 (19.8%)**	**6.4**	**0.17**
	Male	**16 (80.0%)**	**40 (72.7%)**	**31 (81.6%)**	**11 (100.0%)**	**7 (100.0%)**	**105 (80.2%)**		
Residence	**Rural**	**18 (90.0%)**	**52 (94.5%)**	**37 (97.4%)**	**9 (81.8%)**	**7 (100.0%)**	**123 (93.9%)**	**4.6**	**0.328**
	Urban	**2 (10.0%)**	**3 (5.5%)**	**1 (2.6%)**	**2 (18.2%)**	**0 (0.0%)**	**8 (6.1%)**		

- ***All variables were compared using Chi-square X^2 test except (*) One-way ANOVA test.***
- ***All variables were expressed using their No. (%) except (*) by Mean±SD***

Table 2. Clinical Findings & Child score among the studied groups.

		Group I N=20	Group IIa N=55	Group IIb N=38	Group IIc N=11	Group IId N=7	Total N=131	Test	P
Ascites	**Mod**	**7 (35.0%)**	**16 (29.1%)**	**5 (13.2%)**	**3 (27.3%)**	**3 (42.9%)**	**34 (26.0%)**	**5.4**	**0.247**
	Mas	**13 (65.0%)**	**39 (70.9%)**	**33 (86.8%)**	**8 (72.7%)**	**4 (57.1%)**	**97 (74.0%)**		
Jaundice	**Absent**	**11 (55.0%)**	**26 (47.3%)**	**8 (21.1%)**	**0 (0.0%)**	**2 (28.6%)**	**47 (35.9%)**	**16.2**	**0.003**
	Present	**9 (45.0%)**	**29 (52.7%)**	**30 (78.9%)**	**11 (100.0%)**	**5 (71.4%)**	**84 (64.1%)**		
Lowe limb edema	**Absent**	**5 (25.0%)**	**10 (18.2%)**	**5 (13.2%)**	**0 (0.0%)**	**0 (0.0%)**	**20 (15.3%)**	**5.2**	**0.267**
	Present	**15 (75.0%)**	**45 (81.8%)**	**33 (86.8%)**	**11 (100.0%)**	**7 (100.0%)**	**111 (84.7%)**		
Child class	**B**	**14 (70.0%)**	**22 (40.0%)**	**9 (23.7%)**	**4 (36.4%)**	**1 (14.3%)**	**50 (38.2%)**	**13.8**	**0.008**
	C	**6 (30.0%)**	**33 (60.0%)**	**29 (76.3%)**	**7 (63.6%)**	**6 (85.7%)**	**81 (61.8%)**		
Child Score*		**8.9 ± 1.2**	**9.8 ± 1.6**	**10.9 ± 1.5**	**10.7 ± 2.6**	**10.7 ± 1.9**	**10.1 ± 1.8**	**6.4**	**<0.001**

- ***All variables were compared using Chi-square X2 test except (*) One-way ANOVA test***
- ***All variables were expressed using their No. (%) except (*) by Mean±SD***

Table 3. Comparison of ultrasonographic data among the studied groups.

		Group I N=20	Group IIa N=55	Group IIb N=38	Group IIc N=11	Group IId N=7	Total N=131	Test	P
Spleen Diameter, mm*		**16 ± 1.1**	**16.4 ± 1.2**	**16.3 ± 1.1**	**17.2 ± 0.8**	**17 ± 0.8**	**16.4 ± 1.2**	**2.7**	**0.036**
Splenomegaly	**Absent**	**1 (5.0%)**	**3 (5.5%)**	**2 (5.3%)**	**2 (18.2%)**	**2 (28.6%)**	**10 (7.6%)**	**7.9**	**0.048**
	Present	**19 (95.0%)**	**52 (94.5%)**	**36 (94.7%)**	**9 (81.8%)**	**5 (71.4%)**	**121 (92.4%)**		
Portal vein diameter, mm*		**15.1 ± 0.8**	**15.1 ± 0.7**	**15.4 ± 0.8**	**15.1 ± 0.7**	**15.9 ± 0.9**	**15.2 ± 0.8**	**2.3**	**0.06**
Liver	**Average**	**9 (45.0%)**	**15 (27.3%)**	**4 (10.5%)**	**2 (18.2%)**	**0 (0.0%)**	**30 (22.9%)**	**11.6**	**0.02**
	Shrunken	**11 (55.0%)**	**40 (72.7%)**	**34 (89.5%)**	**9 (81.8%)**	**7 (100.0%)**	**101 (77.1%)**		

- ***All variables were compared using Chi-square X2 test except (*) One-way ANOVA test***
- ***All variables were expressed using their N (%) except (*) by Mean±SD***

Table 4. comparison of SAAG values among the studied groups

	Group I N=20	Group IIa N=55	Group IIb N=38	Group IIc N=11	Group IId N=7	F	Sig.
SAAG	1.2 ± 0.1	1.5 ± 0.2	2 ± 0.2	2.5 ± 0.2	2.8 ± 0.1	255.7	**<0.001**

- ***All variables were compared using One-way ANOVA (F) test & expressed by Mean± SD***

Table 5. Correlations between calculated SAAG value and other studied parameters in GI and GII

	No OV		OV	
	R	**P**	**R**	**P**
Age(years)	0.459	0.008	0.257	0.011
Portal vein diameter(mm)	0.332	0.064	0.191	0.06
Spleen Diameter(mm)	0.143	0.433	0.174	0.086
WBCs (×10³/Ul)	0.058	0.752	0.033	0.75
Hemoglobin (gm/dl)	-0.12	0.514	-0.309	0.002
Platelets (×10³/Ul)	0.166	0.364	-0.458	<0.001
Total bilirubin(gm/dl)	0.503	0.003	0.44	<0.001
Direct bilirubin(gm/dl)	0.535	0.002	0.405	<0.001
Serum albumin(g/dl)	0.068	0.712	0.545	<0.001
ALT (unit/liter)	-0.21	0.249	-0.396	<0.001
AST (unit/liter)	-0.111	0.546	-0.419	<0.001
INR	-0.015	0.936	0.29	0.004
Prothrombin time (second)	0.258	0.155	0.169	0.096
C- reactive protein (mg/dl)	0.339	0.058	0.49	<0.001
Child Score	0.461	0.008	0.199	0.05
Albumin in ascitic fluid	-0.725	<0.001	-0.574	<0.001

- ***P ≤ 0.05= significant P <0.001 highly significant and P >0.05 Non-significant. r = Correlation Coefficient***

Table 6. Cut off value of SAAG in prediction of OV.

Predictor for	Cut-off	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)	AUC (95% CI)	P
OV	**>1.3**	**90.09 (83.0 - 94.9)**	**100 (83.2 - 100.0)**	**100**	**64.5 (50.9 - 76.1)**	**0.985 (0.946 - 0.998)**	<0.001

- ***The 95%CI: 95% confidence interval, Positive predictive value (PPV) and negative predictive value (NPV), Area under the ROC curve (AUC).***

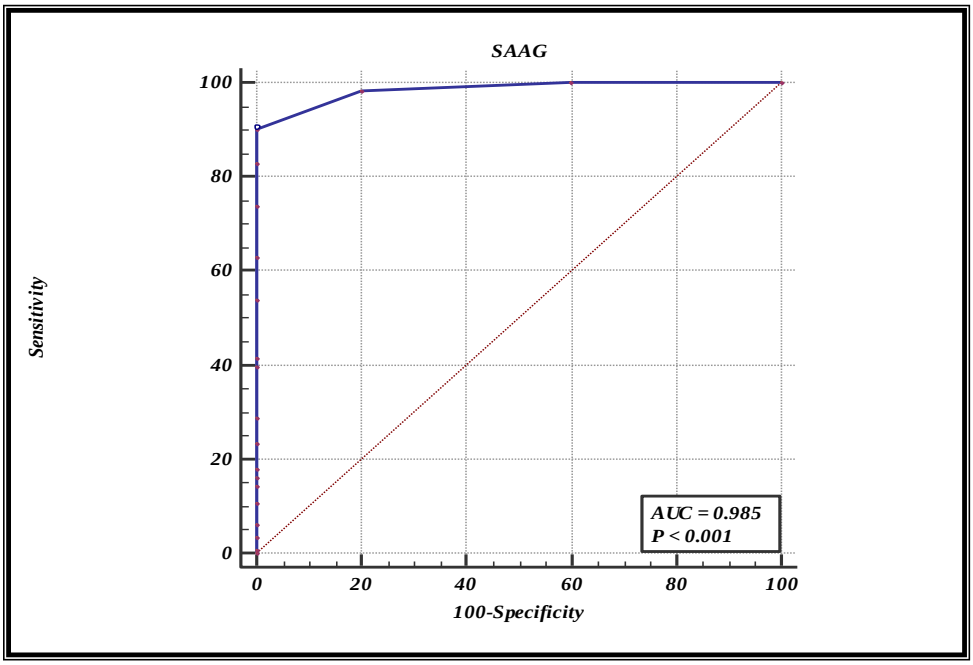

Fig. 1. Receiver operating characteristic (ROC) curve of SAAG value as a predictor for the presence of O.V in Cirrhotic patients.

DISCUSSION

One of the potentially dreadful complications in patients with liver cirrhosis and portal hypertension is esophageal varices. Since effective preventive modalities for variceal hemorrhage have been established, early detection of esophageal varices is critical for primary prevention of bleeding. The gold standard test for screening is still upper gastrointestinal tract endoscopy even with its limitations [4, 16]. However, many studies were developed to predict portal hypertension and OV with diverse rate of accuracy.

The SAAG can define if portal hypertension is present or not with 96.7% accuracy [17]. Indeed, SAAG is now considered a useful physiological and clinical tool in the work-up of ascites. High-albumin gradient (>1.1 gm/dl- due to portal hypertension) or low-albumin gradient (<1.1 gm/dl- not due to portal hypertension) have replaced the terms transudative or exudative in the description of ascites [18].

The aim of the present study is to evaluate the role of serum ascites albumin gradient for prediction of esophageal varices in HCV cirrhotic patients.

In the current study**,** the mean SAAG level was 1.77±0.49 gm/dl. This is in consistent with the findings of **AL-Knawy** and **EL-Sharqawy et al.,** who reported that SAAG level among cirrhotic patients was 1.71±0.61 gm/dl. and 1.46±0.27 (0.9-2.1) gm/dl respectively [19, 20].

This study revealed that SAAG was significantly higher in patients with OV than that of patients without OV. This result is consistent with many previous results including that of **Kumar et al, Rahman et al.**, **Begum et al. and Prabakaran and Gowri** [21- 24] who reported that high SAAG reflects the presence of esophageal varices. Moreover, the presence and size of OV is directly related to the degree of SAAG. On the other hand, these results disagree with that reported by **Demirel et al.** who concluded that no significant statistical difference was detected as regard the SAAG level between patients with esophageal varices and those without [25]. This difference can be attributed to the difference in the population samples. In their study most of the patients with high SAAG were alcoholic cirrhosis, and there was inclusion of some non- cirrhotic patients as acute fulminant hepatitis and congestive heart failure, while in the present study, all patients had ascites due to HCV cirrhosis. Furthermore, this difference may be due to the difference with sample collection. In this study the samples of serum and ascitic fluid were obtained simultaneously or within 30 minutes, while in the other study serum and ascitic fluid samples were not collected simultaneously.

The present study revealed that at cut-off value of ≥1.3 gm/dl SAAG could predict the presence of OV, with specificity and a positive predictive value of 100% and 90.09 %sensitivity and negative predictive value 64.5%. Our results are nearly similar to the results of **El-Sharqawy et al. and Masroor et al.** who reported that SAAG at a cut off value ≥1.435 gm/dl, ≥1.55gm/dl respectively could predict the presence of OV [20, 25]. While these results disagree with that of **Gurubacharya et al.** who reported that OV is present in all patients with SAAG value above 2.0 gm/dl [15]. This difference in the results as regard cut-off value of SAAG level as an accurate indicator for the presence of OV can be attributed to the difference in the population samples. In our study all patients had ascites due to HCV cirrhosis while in other studies; most of the patients with high SAAG had alcoholic liver cirrhosis.

In the present study, SAAG value increased significantly with the grade of OV and this denotes that SAAG value increases in ascites due to portal hypertension. More over our study revealed significant negative correlation between SAAG and albumin in ascetic fluid this result agree with that of **Demirel et al.** [25]

Conclusion: Serum ascites albumin gradient (SAAG) could be used as a non-invasive predictor for the presence of OV in cirrhotic patients with ascites. Moreover, SAAG value increased significantly with the progression of OV.

Ethical approval: all procedures were approved by the institutional Review Board (IRB)**,** the ethical committee of Zagazig University Hospitals.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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