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Study Of Role of Novel Biomarkers for Early Detection of SBP

Sharehan Abdelrahman Ibrahim*[1], Elham Ahmed[1], Mohamed Roshdy[1], Omar Abdelazim[2], Lamiaa Hamdy[3], Hatem Ahmed Hassan[1]

Departments of [1]Internal Medicine, [2]Tropical Medicine and [3]Clinical Pathology, Faculty of Medicine, Minia University, Egypt

***Corresponding author:** Sharehan Abdelrahman Ibrahim, **Mobile:** (+20)01142741126, **E-mail:** anjaz3036@gmail.com

ABSTRACT

Background: Spontaneous bacterial peritonitis (SBP) is a life-threatening complication in cases with liver cirrhosis and ascites. Diagnostic paracentesis is the gold standard for identifying SBP, but it is invasive and resource-intensive. There is a growing interest in finding reliable non-invasive biomarkers to predict SBP early and improve patient outcomes.

Objectives: This study aimed to evaluate the utility of the neutrophil-to-albumin ratio (NAR), mean platelet volume (MPV), and their ascitic fluid (AF) counterparts as predictive markers for SBP in cases with liver cirrhosis and ascites.

Methods: A prospective study that was conducted on cirrhotic cases with ascites at Minia University Hospital from October 2024 to April 2025. Patients were divided into three groups: culture positive SBP, culture negative SBP, and non-SBP. Clinical, biochemical, hematological analysis, and abdominal ultrasound were conducted. Neutrophil-to-albumin ratios and MPV were calculated from blood and ascitic fluid samples.

Results: MPV and NAR were significantly higher in SBP cases, particularly those with culture positive ascites. MPV demonstrated superior diagnostic performance (AUC = 0.80, cut-off >8.15 fL) compared to the blood NAR (AUC = 0.61, cut-off >25). In ascitic fluid, the neutrophil count-to-albumin ratio (AUC = 0.96, cut-off >135.17) outperformed the neutrophil percentage-to-albumin ratio (NPAR) (AUC = 0.60). Both MPV and NAR showed significant positive correlations with inflammatory and liver dysfunction markers, supporting their role as effective indicators of SBP.

Conclusion: MPV and neutrophil-to-albumin ratios are promising noninvasive biomarkers for the early prediction of SBP in cirrhotic cases. Their integration into clinical practice may reduce reliance on invasive procedures and enhance timely intervention.

Keywords: SBP, NAR, MPV, Neutrophil percentage, Ascitic fluid, Neutrophil count.

INTRODUCTION

Cirrhosis is a major health problem globally. In the past, most prominent cirrhotic cases were due to alcoholism (nearly 45%), hepatitis C infection (41%), and nonalcoholic fatty liver disease (NAFLD) (26%), with several cases having more than one cause [(1)]. On the other hand, today, hepatitis C can be cured and most recently diagnosed cirrhosis is due to NAFLD (representing 61.8% of incident cases) and alcoholism (representing 20%) [(2)].

Spontaneous bacterial peritonitis (SBP), which is an acute bacterial infection of ascitic fluid, is associated with a poor long-term prognosis with increased risk of mortality [(3)]. It is diagnosed when the neutrophil count is 250 cells/μL or more, together with a positive bacterial culture result with a single organism is usually identified on the culture in most cases and so these patients should take antimicrobial treatment [(4)].

Culture negative neutrocytic ascites (probable SBP), another entity of SBP, is noted when culturing the ascitic fluid revealed no growth, but the neutrophil count is 250 cells/μL or more. It represents 50% of patients with SBP due to inadequate culturing approaches or late-stage resolving infections. These cases have to be managed just as violently as those with positive culture results [(5)].

The NAR has been suggested as a useful marker for infection and inflammation. Neutrophils are key players in the body's immune response to bacterial infections, and albumin levels tend to decrease in chronic liver disease, partly due to liver dysfunction and chronic inflammation. An increased NAR could potentially indicate a heightened risk of infection, including SBP [(6)].

MPV is a measure of platelet (PLT) size, and it has been suggested as a possible marker for systemic inflammation and platelet activation. In cirrhosis, platelet counts often decrease due to splenic sequestration and bone marrow (BM) suppression. Larger platelets (which are seen in increased MPV) may indicate a hyperreactive or activated platelet system, which could be reflective of inflammatory or infectious processes, such as SBP [(7)].

The NAR and MPV are emerging as potential non-invasive markers for predicting SBP. While both markers individually show promise, the combined use of NAR and MPV may offer enhanced predictive value for SBP. By assessing both the inflammatory status (via NAR) and platelet activity (via MPV), clinicians may be able to better detect cirrhotic patients being at a higher risk of developing SBP, allowing for earlier intervention and more targeted monitoring [(8)].

PATIENTS AND METHODS

This prospective study enrolled cirrhotic patients with ascites admitted to the Internal Medicine Department at Minia University Hospital. All patients (aged from 18 to 70 years with liver cirrhosis and ascites especially with fever and abdominal pain) came to Minia Hepatology Center in the period from October 2024 to April 2025.

Exclusion criteria: Non-cirrhotic ascites, patients were on prophylactic antibiotics for SBP or had taken antibiotics prior to hospitalization, patient with secondary SBP from surgical causes, patients with

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hepatocellular carcinoma, patients with hepatorenal syndrome and septic cases.

Patients were divided into three groups:

- Liver cirrhosis and ascites with culture positive SBP.
- Liver cirrhosis and ascites with culture negative SBP.
- Cirrhotic patients with ascites without SBP.

Entire cases were subjected to history taking with focusing on history of fever and abdominal pain, past history of any chronic illness, HCV, HBV, HIV infection, liver cell failure, hematemesis or melena, drug history of antibiotics and recent surgical history
Full clinical examination was done to all patients.

Sampling: At the patient's bedside, fifteen milliliters of ascitic fluid (AF) sample was withdrawn under complete aseptic conditions. The sample was immediately added to the bedside vials for both aerobic and anaerobic cultures (ten milliliters). The residual AF was investigated biochemically and cytologically by utilizing tubes comprising EDTA.

In addition, 2.5 milliliters of venous blood were withdrawn throughout paracentesis. Two milliliters were put in a polystyrene EDTA tube to assess CBC profile. The residual three milliliters were positioned in a clotting tube for more assessment. Centrifugation was used to separate non-hemolysed sera, utilized to test for urea, creatinine, and liver functions (ALT, AST, albumin, bilirubin, and prothrombin time). Imaging in the form of abdominal ultrasound was done.

Ethics approval: Minia Faculty of Medicine IRB approved the study, and all subjects provided written consents before the study. The study adhered to the Helsinki Declaration throughout its execution.

Statistical analysis

Data were detected using SPSS version 26 (Armonk, NY: IBM Corp). Qualitative data were analyzed using number and percentage. The Kolmogorov-Smirnov test & Shapiro-Wilk test were utilized to confirm the normality of distribution. Quantitative data were defined using mean ± SD for parametric data and median was included for non-parametric data. Statistical significance of the variables was interpreted when p equal to or less than 0.05. Chi-square test and Fisher exact test were utilized for categorical variables, to compare between different groups. Comparisons of the three groups were done with Student t test (parametric data) and Mann-Whitney test (nonparametric data). ANOVA test and Kruskal Wallis test were used to compare between more than two studied groups. Pearson and spearman correlation analyses were done between different variables for parametric and non-parametric variables correspondingly and Roc curve analysis for prediction of SBP.

As demonstrated in table (1), there were insignificant differences among the groups regarding age ($p = 0.08$) and gender distribution ($p = 0.27$). However, significant differences were observed in clinical presentation. Both abdominal pain and fever were reported exclusively in the SBP groups, with no cases in the non-SBP group ($p < 0.001$ for both symptoms), suggesting that these symptoms are strongly associated with SBP regardless of culture positivity. This highlighted the importance of clinical assessment in the early identification of SBP, even in the absence of culture confirmation.

Hemoglobin levels and platelet count did not show significant difference among the three groups. However, total leukocyte count (TLC) was significantly different ($p=0.009$), with the highest value in culture positive SBP (12.9 ± 8.1 x10^3/mm^3), followed by culture negative SBP (11.35 ± 9.2 x10^3/ mm^3) and non-SBP (8.6 ± 5.6 x10^3/ mm^3). Post-hoc analysis displayed a significant difference between culture positive SBP and non-SBP ($p=0.002$) and between culture negative SBP and non-SBP ($p=0.05$). Neutrophil count and percentage were also significantly higher in culture positive SBP than in culture negative SBP and non-SBP with $p<0.001$. MPV was significantly increased in culture positive SBP (9.28 ± 2.2 fl) compared to culture negative SBP (8.62 ± 1.03 fl) and non-SBP (7.5 ± 1.1 fl, $p<0.001$), indicating increased platelet activation in infection. NPAR was highest in culture positive SBP (29.4 ± 9.7), followed by culture negative SBP (27.3 ± 9.5) and non-SBP (25.1 ± 8.3, $p=0.05$) with significant difference between culture positive SBP and non-SBP ($p=0.05$). Neutrophil count to albumin ratio was significantly higher in culture positive SBP (4028 ± 3440) compared to culture negative SBP (3189 ± 2860) and non-SBP (2216 ± 1619, $p=0.002$) with significant differences between culture positive and non-SBP ($p<0.001$), and between culture negative and non-SBP ($p=0.05$) and between culture positive and culture negative SBP ($p=0.05$).

These results propose that the NCAR may be more useful than the NPAR in distinguishing culture positive SBP from non-SBP. Serum urea levels were similar across the groups ($p=0.78$). However, serum creatinine was significantly increased in culture positive SBP ($p=0.001$), while the difference between culture negative and culture positive SBP was not significant ($p=0.69$). Albumin levels did not differ significantly ($p=0.37$), but INR was significantly higher in culture positive SBP (1.79 ± 0.56) compared to non-SBP (1.49 ± 0.47, $p=0.003$) reflecting worse coagulation status. AST levels were significantly elevated in culture positive SBP compared to culture negative SBP ($p=0.03$) and non-SBP ($p=0.03$) suggesting greater hepatic involvement in infection. ALT did not show significant differences among the groups.

RESULTS

Table (1): Comparison of baseline and laboratory parameters between SBP cases with positive culture, culture positive SBP, culture negative SBP and non-SBP cases

	SBP cases with negative culture (n=54)	SBP cases with positive culture (n=46)	Non-SBP cases (n=50)	P value
Age (years) Mean ± SD	60.5±3.6	60.8±3.1	61.7±3.5	**0.08**
Gender Males Females	41 (75.9%) 13 (24.1%)	30 (65.2%) 16 (34.8%)	31 (62%) 19 (38%)	**0.27**
Symptoms Abdominal pain Fever	30 (55.6%) 31 (57.4%)	17 (37%) 26 (56.5%)	0 (0%) 0 (0%)	**<0.001*** **<0.001***
HB (gm/dl) Mean ± SD	10.6±2.1	10.5±2.2	9.8±2.04	0.15
TLC (x10^3/UL) Mean ± SD	11.35±2.78	12.9±2.98	8.6±1.94	0.009* , P1=0.05* P2=0.02*, P3=0.002*
Neutrophils count Mean ± SD	9069±54	10905±44	5862±44	<0.001*, P1=0.05* P2=0.02*, P3<0.001*
Neutrophils % Mean ± SD	76.48±17.6	81.4±8.1	67.5±4.5	<0.001*, P1=0.43 P2<0.001*, P3<0.001*
Platelet (x10^3/UL) Mean ± SD	191±6.8	177.3±44.11	153.6±8.1	0.22
MPV Mean ± SD	8.62±1.03	9.28±2.2	7.5±1.1	<0.001* P1=0.05* P2=0.001* P3<0.001*
Albumin (g/dl) Mean ± SD	2.95±0.62	2.93±0.60	2.8±0.52	**0.37**
Neutrophil % to albumin ratio (NPAR) Mean ± SD	27.3±6.7	29.4±7.1	25.1±5.3	0.05*, P1=0.72 P2=0.28, P3=0.05*
Neutrophil count to albumin ratio (NCAR) Mean ± SD	3189±260	4028±340	2216±119	0.002*, P1=0.05* P2=0.05*, P3<0.001*
Urea (mg/dl) Mean ± SD	70.5±7.4	72.2±8.2	68.1±6.7	0.78
Creatinine (mg/dl) Mean ± SD	1.24±0.30	1.30±0.31	1.04±0.24	0.001*, P1=0.69, P2=0.008, P3=0.001*
INR % Mean ± SD	1.71±0.41	1.79±0.43	1.49±0.36	0.01*, P1=0.18 P2=0.07, P3=0.003*
AST (IU/L) Mean ± SD	105.4±24.9	140±34.8	105.3±23.8	0.02*, P1=0.03* P2=0.99**,** P3=0.03*
ALT (IU/L) Mean ± SD	116.7±28.9	118±9.1	96.9±4.1	0.12

* Significant at p value ≤ 0.05, **p1:** p value between SBP negative and positive culture, **p2:** p value between SBP negative and non-SBP, **p3:** p value between SBP positive culture and non-SBP , p value was calculated by ANOVA test and Kruskal Wallis test for numerical data followed by post hoc analysis, Chi square and Fisher exact for categorical data

As shown in table (2), ascitic fluid WBC count was significantly higher in SBP cases (p<0.001), with culture positive cases showing the highest values (4008±1001.1 cells/UL), followed by culture negative SBP (3582±892.8 cells/UL) and non-SBP (246.5±60.9 cells/UL). Culture positive and culture negative SBP cases had significantly higher WBC counts than non-SBP (p<0.001), but there was insignificant difference between the SBP subgroups (p=0.88). Ascitic fluid neutrophil percentage was also significantly higher in SBP cases (p=0.02), with significant differences between culture positive and non-SBP (p=0.01) and between culture negative and non-SBP (p=0.03), but no statistical significance between the SBP subgroups (p=0.63). Also, ascitic fluid NPAR was significantly higher in SBP cases with culture positive than non-SBP cases (p value =0.01). While, non-significant difference was found between SBP with culture negative and non SBP and between SBP with culture negative and culture positive (p value >0.05). While, ascitic fluid neutrophil count to albumin ratio was significantly increased in culture positive SBP than in non-SBP cases (p value =0.006) and between SBP with culture negative and non SBP (p=0.05) and non-significant difference was found between SBP with culture negative and culture positive (p=0.55).

Table (2): Comparison of ascitic fluid analysis between SBP cases with positive culture, SBP cases with negative culture and non-SBP cases

	SBP cases with negative culture (n=54)	SBP cases with positive culture (n=46)	Non-SBP cases (n=50)	P value
Ascitic fluid WBCs (cell/ UL) Mean ± SD	3582± 92.8	4008±101.1	246.5±60.9	<0.001* P1=0.88 P2<0.001* P3<0.001*
Ascitic fluid neutrophils % Mean ± SD	36.5±5.3	40.5±9.6	25.08±2	0.02* P1=0.63 P2=0.03* P3=0.01*
Ascitic fluid albumin Mean ± SD	1.45±0.28	1.46±0.22	1.31±0.38	0.19
Ascitic fluid neutrophil % to albumin ratio Mean ± SD	28.5±3.2	33.8±5.9	19.8±1.1	0.01* P1=0.50 P2=0.14 P3=0.01*
Ascitic fluid neutrophil count to albumin ratio Mean ± SD	1114±239	1629±235	49.07±6.3	0.007* P1=0.55 P2=0.05* P3=0.006*
SAAG Mean ± SD	1.49±0.37	1.47±0.26	1.49±0.26	0.93

***** Significant at p value ≤0.05, **p1:** p value between SBP negative and positive culture, **p2:** p value between SBP negative culture and non-SBP, **p3:** p value between SBP positive culture and non-SBP, p value was calculated by Kruskal Wallis test for numerical data followed by post hoc analysis.

ROC analysis was performed to evaluate the diagnostic accuracy of NPAR and MPV in predicting SBP. MPV had a significantly higher AUC of 0.80 (95% CI: 0.72-0.87, p<0.001) compared to NPAR, which had an AUC of 0.61 (95% CI: 0.51-0.70, p=0.02). This indicated that MPV was a more reliable predictor of SBP than NPAR. The optimal cutoff value for MPV was determined to be >8.15 fL, with a sensitivity (Sn) of 75%, specificity (Sp) of 72%, PPV of 84.3%, NPV of 59%, and accuracy of 75%. In contrast, NPAR had a cutoff value of >25, which yielded 62% sensitivity, 50% specificity, PPV of 71.3%, NPV of 39.7%, and accuracy of 58% (table 3). These results suggested that MPV has a stronger predictive ability for SBP compared to NPAR, making it a more effective biomarker.

Table (3): ROC analysis for Neutrophils percentage to albumin ratio and MPV for prediction of SBP

	Neutrophils percentage to albumin ratio	MPV
AUC	0.61	0.80
95% CI	0.51-0.70	0.72-0.87
P value	0.02*	<0.001*
Cut off value	>25	>8.15
Sensitivity	62%	75%
Specificity	50%	72%
PPV	71.3%	84.3%
NPV	39.7%	59%
Total accuracy	58%	75%

ROC curve analysis was conducted to assess the diagnostic accuracy of ascetic neutrophil Percentage and count to Albumin Ratio in predicting SBP. Ascitic neutrophil count to Albumin Ratio had a significantly higher AUC of 0.96 (p<0.001) compared with ascitic NPAR, which had an AUC of 0.60 (p=0.03).

This indicates that ascitic NPAR is a more reliable predictor of SBP than ascitic NCAR. The optimal cutoff value for ascitic neutrophil count to Albumin Ratio was determined to be >135.1750, with a Sn of 88%, Sp of 92%, PPV of 88%, NPV of 96%, and accuracy of 89.3%.

In contrast, ascitic NPAR had a cutoff value of >15.2778, yielding 63% Sn, 46% Sp, PPV of 70%, NPV of 38%, and accuracy of 57.3%. These results suggest that ascitic NPAR has a stronger predictive ability for SBP compared to ascitic NPAR, making it a more effective biomarker. (Figures 1 and 2)

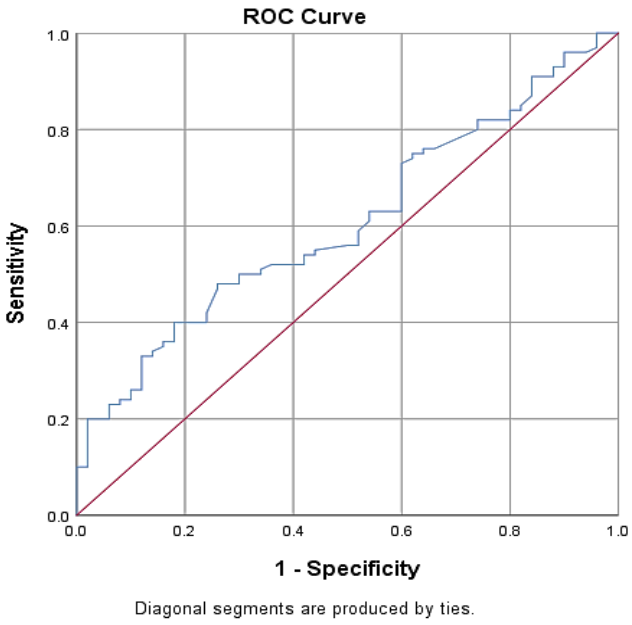

Fig. (1): ROC curve analysis for ascitic neutrophils percentage to albumin ratio for prediction of SBP.

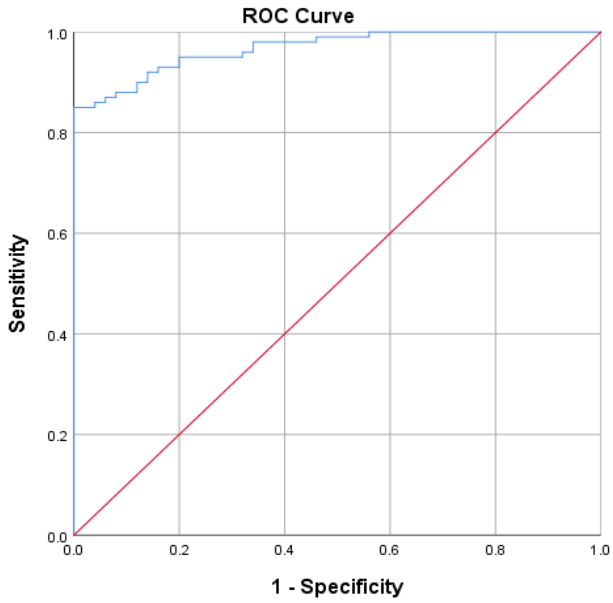

Fig. (2): ROC curve analysis for ascitic neutrophils count to albumin ratio for prediction of SBP.

Table (4) evaluated the correlation (Pearson's r) between MPV, NPAR, and various hematological, renal, liver, and ascitic fluid parameters to identify significant relationships. MPV showed significantly +ve correlations with Neutrophil count (r=0.17, p=0.03), creatinine (r=0.21, p=0.009) suggesting that MPV may be linked to declining renal function & Neutrophil percentage (r=0.16, p=0.04), reinforcing its association with systemic inflammation and AF WBC count (r=0.41, p=0.001). NPAR displayed stronger correlations with Neutrophil % (r=0.68, p<0.001), INR (r=0.40, p<0.001), TLC (r=0.24, p=0.002), Neutrophil count (r=0.20, p=0.01), AST (r=0.20, p=0.01) and ALT (r=0.21, p=0.01).

These correlation findings indicated that MPV is more closely linked to inflammatory and renal dysfunction markers, while NPAR is strongly accompanied by neutrophil response and coagulation abnormalities. The strong correlation between NPAR and INR (r=0.40, p<0.001) may suggest its potential role in identifying severe infections that are impacting coagulation.

Table (4): Correlation between MPV, Neutrophils to albumin ratio and other variables among all cases

	MPV		Neutrophils % to albumin ratio (NPAR)	
	R	**P value**	**R**	**P value**
HB (gm/dl)	0.11	0.15	-0.23	0.005*
TLC (x10^3/UL)	0.13	0.09	0.24	0.002*
Neutrophils count	0.17	0.03*	0.20	0.01*
Neutrophils %	0.16	0.04*	0.68	<0.001*
Platelet (x10^3/UL)	0.005	0.94	0.10	0.19
MPV	1		0.006	0.95
Urea (mg/dl)	0.12	0.13	0.08	0.32
Creatinine (mg/dl)	0.21	0.009*	0.09	0.26
Albumin (g/dl)	0.15	0.06	0.67	<0.001*
INR %	0.06	0.55	0.40	<0.001*
AST (IU/L)	0.11	0.16	0.20	0.01*
ALT (IU/L)	-0.03	0.71	0.21	0.01*
Ascitic fluid WBCs (cell/ UL)	0.41	0.001*	0.15	0.05*
Ascitic fluid neutrophils %	0.14	0.07	0.05	0.51
Ascitic NPAR	0.03	0.65	0.30	<0.001*
Ascitic NCAR	0.08	0.30	0.04	0.59
SAAG	0.12	0.14	-0.42	<0.001*

* significant at p value ≤0.05.

DISCUSSION

Cirrhosis is an important cause of death globally. Portal hypertension causes about seventy five percent of cases of ascites, with the remaining cases emerging from infections, and infiltrative diseases [(7)]. SBP is a common complication in cirrhotic cases with ascites (10-30 %) with a 20% incidence and an estimated 20% in-hospital death rate [(9)].

Based on international guidelines, the best traditional approach for diagnosis of SBP is diagnostic paracentesis [(10)]. On the other hand, this method is invasive and carries risks. A novel inflammation-based predictor, the NPAR, has been developed. Traditional blood tests are used by the NPAR to determine albumin and neutrophil %. There is limited data regarding the role of the NPAR in SBP diagnosis.

So, we hoped to identify other non-invasive, cheap and easily achieved markers for early detection of SBP to avoid the high mortality rate associated with SBP.

In the present study, culture positive SBP patients had higher serum creatinine, INR, AST, TLC, neutrophil count and percentage, MPV, NPAR and neutrophil count to albumin ratio than other groups. This is consistent with **Metwally *et al.*** [(11)] who found that creatinine, INR, and serum WBC were significantly higher in 59 SBP patients. Also, **Popoiag *et al.*** [(12)] found significant difference between the mean values of creatinine, INR, TLC, but inconsistent with our results about serum albumin and platelets that were significantly increased in the SBP cases compared to non-SBP. In the same line, **Abdel Rahman *et al.*** [(13)] conducted their study on 80 cases, 40 cases with liver cirrhosis and SBP and 40 cases with liver cirrhosis without SBP, the mean levels of Hb and ALT didn't vary significantly between both groups but neutrophils were significantly different. On the other hand, creatinine values were comparable in the two groups. **Abudeif *et al.*** [(6)] displayed that neutrophils were significantly higher in cases with SBP.

Our study displayed that ascitic fluid WBCs count and neutrophil percentage were significantly higher in SBP either with culture +ve or culture -ve than with non SBP patients and this is in agreement with **Mousa *et al.*** [(14)] and **Khalil *et al.*** [(15)] who found that PMN in ascetic fluid showed higher levels in the SBP group.

In our study, we found that NPAR was elevated in SBP +ve culture than in -ve culture SBP or non SBP cirrhotic patients. Also, neutrophil count to albumin (NCAR) ratio was higher in SBP positive group than other groups. This is in agreement with **Mousa *et al.*** [(14)] who found significantly high NPAR in SBP patients but yet there are no studies as regards neutrophil count to albumin ratio.

Cases with liver cirrhosis and ascites had higher intestinal permeability, increasing risk of translocation of bacteria and SBP. Neutrophils play an important role in infections and defending against microbial invasion [(16)]. Measuring neutrophils in the blood is a cheap and easily obtainable investigation for determining bacterial infections. A high WBCs count may point to a microbial infection, whereas an increasing PMNL count could indicate that the infection hasn't been completely eradicated [(17)]. This resistance to infection and partial eradication could be accompanied by more formation of PMNLs by the BM. Neutrophils through phagocytosis

process and releasing ROS can protect patients from infection [18].

Albumin plays role in bacterial infections. Low serum albumin values are indicators for infection as they have a direct role in defending against infections. Albumin plays a role in opsonization by binding to bacteria and making them easier for immune cells to engulf and destroy. In patients with low serum albumin, the opsonization process is compromised, meaning bacteria in the ascitic fluid are less likely to be cleared effectively [19]. Both increased neutrophils and reduced albumin are linked to higher risk of infection so the combination of NAR can be used to assess the risk of SBP.

We found that NPAR > 25 can predict SBP with a Sn of 62%, specificity of 50%, NPV and PPV of 39.7 % and 71.3% correspondingly.

MPV was significantly increased among SBP cases either culture +ve or -ve than in non SBP with a cut off > 8.15 fl, which can predict SBP with a Sn of 70%, specificity of 50%, NPV and PPV of 66 % and 54% respectively. Similar findings are observed by **Suvak *et al.*** [20] who found that the MPV cutoff point for cirrhotic SBP was 8.4, with Sn, Sp, NPV, and PPV of 70.7%, 67.5%, 75.4% and 62.1% correspondingly. Also similar to that recorded by **Abdel-Razik *et al.*** [21] in which MPV had 8.77% as a cutoff value and 95.9% Sn and 91.7% Sp for detection of SBP. In addition, **Gálvez-Martínez *et al.*** [22] displayed that the best was the cutoff value of 8.3fl, with Sn, Sp, PPV, NPV and accuracy of 84%, 82%, 83%, 84% and 83% correspondingly. Also, **Lashin *et al.*** [23] found MPV level cutoff point for cirrhotic cases with SBP was 8.71FL, with a Sn, Sp, NPV and PPV of 68.75%, 80%, 87% and 54.8 % correspondingly. These results suggest that MPV has a stronger predictive ability for SBP compared to NPAR, making it a more effective biomarker.

The use of Ascitic neutrophils count to albumin ratio and ascitic NPAR as indicator for spontaneous bacterial peritonitis did not previously discussed in literature, therefore no studies to compare with.

Ascitic neutrophils count to albumin ratio had a cut off value of 135.17 for cirrhotic cases with SBP with a Sn, Sp, NPV and PPV of 88 %, 92%, 96% and 88 % respectively, which was superior to Ascitic NPAR which has a cut off value of >15.27 for cirrhotic cases with SBP with a Sn, Sp, NPV and PPV of 63%, 46%, 38% and 70% respectively suggesting that ascitic neutrophil count to albumin ratio has a stronger predictive ability for SBP compared to ascitic NPAR, making it a more efficient biomarker.

In this study we demonstrated that MPV showed significant correlations with neutrophil count, creatinine, neutrophil percentage and AF WBC count (r=0.41, p=0.001). **Khalil *et al.*** [15] found that there was a significant positive association between MPV and neutrophil count (r = .3720, p<0.0001) & ascitic fluid WBC count (r=0.27, P=0.0001), and a negative association with PLT (r=-0.14, P=0.03) among SBP group. **Gálvez-Martínez *et al.*** [22] studied the relation between systemic inflammatory response parameters and MPV in cases with liver cirrhosis in presence or absence of SBP and revealed statistically significant correlations between MPV with neutrophil count and ascitic fluid WBC count.

In this study we illustrated that NPAR showed stronger correlations with inflammatory and coagulation markers (neutrophil percentage, INR, TLC, neutrophil count, AST and ALT). **Mousa *et al.*** [14] found a significant negative association between NPAR, serum albumin, platelet, and eGFR and a significant positive association between NPAR and WBC count, neutrophil percentage, ALT, and creatinine. **He *et al.*** [24] displayed a significant association between NPAR, albumin, platelet, WBC and neutrophils.

LIMITATIONS

The main limitation was the relatively small sample size, so much research on a large number of cases is necessitated to evaluate this test in different situations.

CONCLUSION

In conclusion, ascitic neutrophil count to albumin ratio had the highest diagnostic accuracy, sensitivity and specificity for early detection of SBP followed by MPV then NAR. Ascitic fluid test being invasive and not always available, the NAR and MPV are a promising noninvasive biomarker for SBP prediction in cirrhotic cases with ascites. Its ability to reflect both systemic inflammation and liver synthetic function makes it a comprehensive and reliable tool for early diagnosis. The integration of NAR and MPV clinically could improve patient outcomes by enabling timely intervention and reducing the need for invasive procedures. Much research is necessitated to confirm the current findings and explore the full potential of NAR and MPV in the management of SBP.

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