

Role of Ascitic Fluid Polymorphic Nuclear Cell Count and Prostaglandin E2 Prognostic Outcome of Cirrhotic Diseased Individual's Mortality

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ABSTRACT

Background: One of the most common and dangerous consequences in individuals with decompensated cirrhosis is SBP. Early antibiotic treatment is crucial for effective treatment and lowering death rate. A modulator of the immune response and infection is PGE2. Aim of the Study: To analyze how Prostaglandin E2 and the ascites polymorphic nuclear cell count affect cirrhotic diseased individual mortality prognosis.

Material and Methods: Ninety diseased individuals were chosen from the wards of the Internal Medicine Department of Menoufia University Hospitals, the NLI of Menoufia University, and the El Helal Health Insurance Hospital in Shibin Al-Kum for this prospective diagnostic test study. Two cohorts of participants were formed. Cohort I: 45 diseased individuals with hepatic cirrhosis and SBP, as well as 45 diseased individuals without SBP. All diseased individuals had a history review, MELD and CTP scores, a pelvic-abdominal ultrasound, and laboratory investigations such as serum, CBC with differentials made by CRP and ESR, INR and prothrombin time, SAAG, serum creatinine and urea, and liver function tests (ALT and AST), GGT, and Alk.pho. PMN and serum ascitic fluid prostaglandin E2 are included in ascitic fluid analyses.

Results: While ascitic fluid PGE2 was elevated in the non-SBP cohort, PMN cell count was elevated in the SBP cohort. Regarding ascitic fluid PGE2, there is a substantial difference between the two cohorts. There are numerous advantages. The relationship between PMN cells and other factors, including ascitic fluid PGE2, Hb, WBC, GGT, alkalinity, and MELD score. The relationship between ascitic fluid PGE2 and other WBC is significantly inverse.

Conclusion: both blood and ascitic fluid for the detection and eradication of SBP, as well as for predicting death in cirrhotic diseased individuals, PGDE2 and PMN can be employed as diagnostic and prognostic markers.

Key words: ascitis polymorphic nuclear cell count, Prostaglandin E2, spontaneous bacterial peritonitis

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INTRODUCTION

Cirrhosis, which causes the development of fibrosis and parenchymal function loss as a result of chronic or repetitive liver injury, accounts for about 170,000 deaths annually in Europe. Critical occurrences in the normal course of cirrhosis, such as the development of fluid accumulation in the abdomen, renal failure, and bleeding of varices, all of which portend a poor outcome. Acute-on-CLF and hepatic decompensation may result from most of these problems, which are linked to portal hypertension (1).

Individuals with cirrhosis typically have bacterial infections more frequently than the general population, which quadruples mortality (2). SBP, the most common infection among people with cirrhosis and ascites, is associated with a poor outcome. SBP risk factors include low serum sodium levels, CTP stage C, and elevated A-PMN cell counts (greater than one hundred cells/l) (3).

To improve the prognosis of individuals with SBP, new biomarkers that can diagnose SBP or predict mortality must be developed. In diseased individuals with decompensated cirrhosis, ascitic prostaglandin E2 level is a highly likely valuable predictor marker of in-hospital mortality, and it is important in detection of SBP as an immunological informatory mediator (4).

The objective of the current study was to assess how the ascitic fluid polymorphic nuclear cell count and prostaglandin E2 prognosticated the death of cirrhotic diseased individuals.

MATERIAL AND METHODS

Ninety diseased individuals were chosen from the wards of the Internal Medicine Department of Menoufia University Hospitals, the NLI of Menoufia University, and the El Helal Health Insurance Hospital in Shibin Al-Kum for this prospective diagnostic test study. Each diseased individual and control gave their written agreement to share in the study, which was performed in compliance with the Menoufia University local ethical committee between April 2020 and May 2021.

The participants were divided into two cohorts Cohort I: forty five diseased individuals with SBP and liver cirrhosis (included forty five diseased individuals Non-SBP with liver cirrhosis).

All diseased individuals were enrolled in the study period if they were above eighteen years old, both genders were included and diseased individuals fulfilling the criteria for SBP.

Diseased individuals who declined to participate in

the trial, those who had any other cause of ascites outside liver cirrhosis (such as cardiac, renal, etc.), those who had portal vein thrombosis, and those who had HCC or other cancers were removed from the study.

Data on demographics, including age, gender, place of residence, disease onset, duration, and family history, were collected on all diseased individuals. SBP symptoms include: diseased individual classification based on MELD and CTP scores, imaging, which revealed results from an abdominal ultrasound.

Laboratory investigations were conducted using a CBC with differentials and a Beckman Counter AU680 chemistry analyzer (both from California, USA). CRP and ESR, two markers of inflammation. INR and thrombin time, SAAG, serum creatinine and urea, ALT and AST liver function tests, GGT, and alk. pho. Ascitic fluid prostaglandin E2 was measured using an ELISA, and ascitic fluid examination also involves detection of PMN.

RESULTS

PMN cell count and ascitic fluid PGE2 show a significant difference between the two cohorts, with the SBP cohort having a higher PMN cell count and the non-SBP cohort having a higher ascitic fluid PGE2 level (*table 1*).

In terms of PMN cells, there is a substantial difference between the two cohorts, indicating that the higher the ascitic PMN cell count, the higher the hospital mortality rate. Regarding Ascitic fluid PGE2, there is a substantial difference between the two cohorts, showing that the greater the hospital mortality rate for diseased individuals with hepatic cirrhosis, the lower the Ascitic fluid PGE2 (*table 2*).

Regarding other factors (such as ascitic fluid PGE2), there is a substantial negative correlation between PMN cells, but not with regard to other parameters (Age- Platelets-ESR- Prothrombin time-Sodium level INR). There is a substantial positive correlation between PMN cells and other measures such as hemoglobin, white blood cell count, GGT, alkaline phosphatase, and MELD score, but not with the following parameters: RBC, CRP, serum urea, serum creatinine, SGOT, SGPT, albumin, or serum total bilirubin (*table 3*).

There is no significant positive association with Hb and no significant negative correlation with the remaining parameters and ascitic fluid PGE2, however there is a substantial negative correlation between ascitic fluid PGE2 and other parameters with regard to WBC (*table 4*).

Table 1 - Comparison between studied cohorts as regard PMN cell count and ascitic fluid PGE2. (Diagnostic markers for SBP)

	SBP cohort (N=45)		Non-SBP cohort (N=45)		Test of significance	P value
	No.	%	No.	%		
PMN cell count (cell/ML)					U	< 0.001**
Mean $\pm$ SD	531.9 $\pm$ 171.1		74.2 $\pm$ 45.7		8.1	
Range	270-940		4.60-220			
PMN cell count (cell/ML)					FET	< 0.001**
> 125	0(0%)		43(95.6%)		90	
125-250	0(0%)		2(4.4%)			
250-500	20(44.4%)		0(0%)			
< 500	25(55.6%)		0(0%)			
Ascitic fluid PGE2 (pg/ml)					t	<0.001*
Mean $\pm$ SD	44.3 $\pm$ 9.3		58.4 $\pm$ 14.8		5.36	
Range	28-58		35.6-89.6			

Table 2 - Comparison between studied cohorts as regard Mortality rate with PMN cell and Ascitic fluid PGE2

	(Deceased) mortality rate		Test of significance	P value
	Yes (N=15)	No (N=75)		
PMN (cell/ml)			U	0.035*
Mean $\pm$ SD	402.5 $\pm$ 199.1	283.1 $\pm$ 269.1	2.10	
Range	46-900	4.60-940		
Ascitic fluid PGE2 (pg/ml)			t	< 0.001**
Mean $\pm$ SD	34.4 $\pm$ 3.2	54.7 $\pm$ 13.1	11.7	
Range	28.4-38.3	28-89.6		

Table 3 - Multivariate correlation between ascitic PMN cell and other parameters

	PMN cell count (cell/ ML)	
	rho	P value
Age (yrs)	-0.004	0.970
Hb (g/dl)	0.689	<0.001**
RBC(cell/mm ³)	0.129	0.225
WBC(cell/mm ³)	0.205	0.052*
Platelets(cell/mm ³)	-0.072	0.500
CRP (mg/ml)	0.109	0.314
ESR (hr/mm)	-0.029	0.783
Serum urea	0.122	0.253
Serum creatinine	0.171	0.108
AST	0.170	0.110
ALT	0.118	0.268
GGT(U/L)	0.407	<0.001**
Alk-ph (U/L)	0.279	0.008*
Serum albumin (g/dl)	0.040	0.705
SAAG (g/dl)	-0.212	0.045*
Serum total bilirubin (g/dl)	0.098	0.358
Prothrombin time (sec.)	-0.127	0.235
Sodium level (Mmol/L)	-0.111	0.296
INR %	-0.014	0.898
MELD score	0.250	0.019*
Ascitic fluid PGE2 (pg/ml)	-0.306	0.003*

Table 4 - Multivariate correlation between Ascitic fluid PGE2 and other parameters

	PMN cell count (cell/ ML)	
	r	P value
Age (yrs)	-0.059	0.583
Hb (g/dl)	0.072	0.503
RBC(cell/mm ³)	-0.038	0.724
WBC(cell/mm ³)	-0.214	0.043*
Platelets(cell/mm ³)	0.044	0.683
CRP (mg/ml)	-0.096	0.380
ESR (mm/hr)	-0.048	0.653
Serum urea	-0.182	0.253
Serum creatinine	-0.097	0.362
AST	-0.131	0.217
ALT	-0.130	0.222
GGT(U/L)	-0.038	0.719
Alk-ph (U/l)	-0.055	0.606
Serum albumin (g/dl)	-0.052	0.629
SAAG	0.234	0.027*
Serum total bilirubin (g/dl)	-0.113	0.288
Prothrombin time (sec.)	-0.024	0.825
Sodium level (Mmol/L)	-0.111	0.296
INR %	-0.014	0.898
MELD score	-0.165	0.124

Table 5 - Validity of ascitic PMN cell count as a prognostic marker for mortality in cirrhotic diseased individuals

AUC	P value	Cutoff point	Sensitivity	Specificity	NPV	PPV	Accuracy
0.672	0.036	>210	80%	55.7%	93%	27%	60%

Table 6 - Validity of Ascitic fluid PGE2 as a prognostic marker for mortality in cirrhotic diseased individuals

AUC	P value	Cutoff point	Sensitivity	Specificity	NPV	PPV	Accuracy
0.965	0.00	<38.4	93.3%	96%	99%	82%	96%

The accuracy of ascitic PMN cell count as a prognostic marker for mortality in cirrhotic diseased individuals at a cutoff point (more than 210) is sixty percent, with sensitivity eighty percent and specificity fifty five percent, respectively (*table 5*).

Sensitivity (93%), specificity (96%), NPV (99%), PPV (82%) and accuracy (96%) of ascitic fluid PGE2 as a prognostic marker for mortality in cirrhotic diseased individuals at a cutoff point (38.4) (*table 6*).

The reliability of combined ascitic PMN cells and ascitic fluid PGE2 as a prognostic marker for death in cirrhotic diseased individuals at sensitivity (93%) and specificity (51%), with negative predictive value (97%) and positive predictive value (27%) and accuracy (58%) respectively (*table 7*).

The accuracy (100%), sensitivity (100%), specificity (100%), and negative predictive value (100%) of ascitic fluid PMN cell count was important in detection of SBP in cirrhotic diseased individuals at a cutoff point more than 245) (*table 8*).

The reliability of ascitic fluid PGE2 in detection of SBP in diseased individuals with cirrhosis at a cutoff

Table 7 - Validity of combined ascitic PMN cells and ascitic fluid PGE2 as a prognostic marker for mortality in cirrhotic diseased individuals

Sensitivity	Specificity	NPV	PPV	Accuracy
93%	51%	97%	27%	58%

point (54.1), with sensitivity (80%), specificity (60%), and, respectively, negative predictive value = (75%) positive predictive value = (67%) and accuracy (70%) (*table 9*).

Table 10 and *fig. 1* showed a significantly lower survival in patients with spontaneous bacterial peritonitis (SBP) as it showed mean survival 5.3 months versus 5.71 months for non SBP group with p value 0.04.

Low and high values were divided according to median of ascitic fluid PEG2 in SBP group which was 46.2. There was a significantly longer survival among patents of SBP with high ascitic fluid PEG2 (5.26 months) versus those with lower values (4.18 months) with p value <0.001 (*table 11, fig. 2*).

There was a non-significant difference in survival in

Table 8 - Validity of ascitic PMN cell count as diagnostic marker for SBP in cirrhotic diseased individuals

AUC	P value	Cutoff point	Sensitivity	Specificity	NPV	PPV	Accuracy
1.00	0.00	>245	100%	100%	100%	100%	100%

Table 9 - Validity of ascitic fluid PGE2 as a diagnostic marker for SBP in cirrhotic diseased individuals

AUC	P value	Cutoff point	Sensitivity	Specificity	NPV	PPV	Accuracy
0.768	0.00	<54.1	80%	60%	75%	67%	70%

Table 10 - Overall survival between the studied groups (SBP & Non SBP)

	Mean survival			Log Rank	P value
	SE	value	95% CI		
SBP	0.20	5.30	4.91 – 5.69	4.05	0.04
Non SBP	0.14	5.71	5.43 – 5.99		

SE = standard error, CI = confidence interval

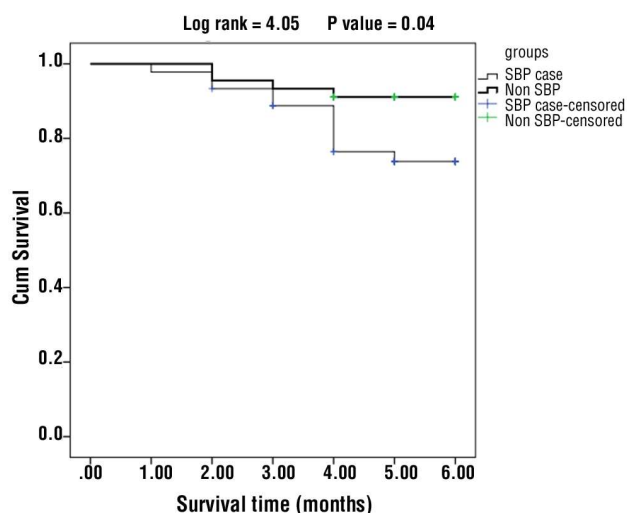


Figure 1

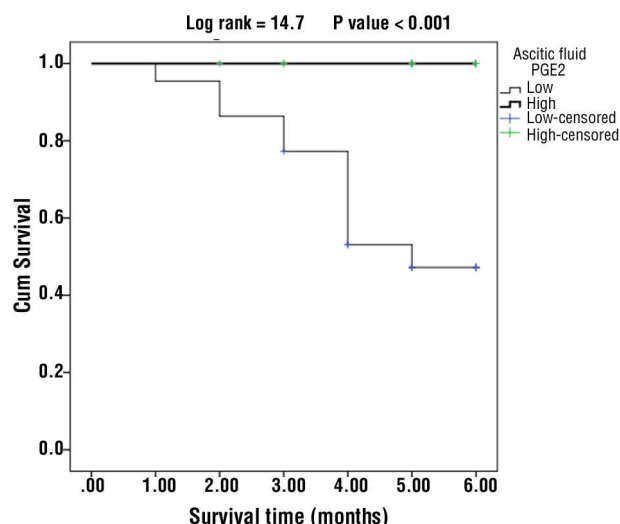


Figure 2

Table 11 - Overall survival in relation to ascitic fluid value in SBP group

Ascitic fluid PGE2	Mean survival			Log Rank	P value
	SE	value	95% CI		
Low	-----	4.18	-----	14.70	<0.001
High	0.19	5.26	4.65 – 5.99		

SE = standard error, CI = confidence interval

Table 12 - Overall survival in relation to PMN cell count value in SBP group

PMN cell	Mean survival			Log Rank	P value
	SE	value	95% CI		
Low	0.35	5.06	4.38 – 5.74	0.40	0.53
High	0.19	5.53	5.16 – 5.91		

SE = standard error, CI = confidence interval

Low and high values were divided according to median of PMN cell count in SBP group which was 520 (cell/ML)

relation to values of PMN cells with p value 0.53 (table 12, fig. 3).

DISCUSSION

SBP is the most prevalent bacterial infection in people with cirrhosis (5).

To enhance the diagnosis of SBP, numerous studies have been conducted in recent years, including the nonculture approach and the search for novel biomarkers (6,7). Some of these non-culture methods were expensive and time-consuming, while some of these biomarkers had low stated sensitivity or specificity (8).

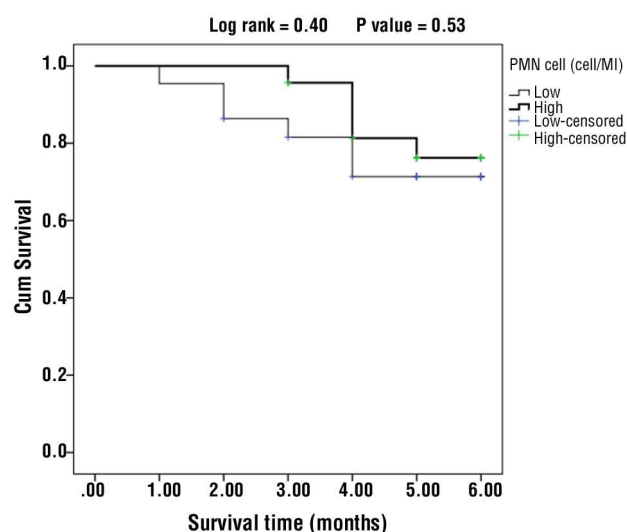


Figure 3

No information was actually released on the comparison of the ascitic PGE2 levels in the decompensated cirrhotic illness patients with or without SBP. The aim of the present study was to investigate the effects of prostaglandin E2 and ascitic fluid polymorphic nuclear cell count on cirrhotic disease-related mortality.

Our findings indicate that there are no appreciable differences in age, gender, or residency between the two cohorts (SBP cohort and Non-SBP cohort). These findings concur with those of Luo et al., who found no significant differences in the median onset age or gender between the two cohorts (9). Additionally, Aboghiaty et al. found no statistically significant differences in age, gender, smoking, or the prevalence of concomitant diabetes or hypertension between the study cohorts (10). According to earlier research, there was no statistically significant difference in age between the infected and non-infected cohorts (11, 12). These results largely rule out demographic variables as confounding variables.

Additionally, we discovered that there is no discernible difference between the two cohorts in terms of the cause of cirrhosis (HBV- HCC- Biliary cirrhosis- Bilharaziz) co-morbid conditions, types of co-morbid conditions (HTN-CKD-Cardiac). The prevalence of HCV infection was higher in the non-SBP cohort, though, and there were notable differences between the two cohorts in terms of the outcome (mortality), which was higher in the SBP cohorts. Additionally, there is a notable difference in terms of DM, which was higher in the non-SBP cohort. In line with our findings, Aboghiaty et al. (10) found no statistically significant difference in the presence of hypertension between the examined cohorts. However, they disagreed with our findings because they discovered that DM was not related to SBP.

Additionally, Mehta et al. (13) observed that diseased individuals with diabetes had a greater adjusted risk of decompensated cirrhosis than diseased individuals without diabetes. According to Tergast et al. (14), diseased individuals with DM were more likely to acquire an SBP over the course of their follow-up. The increased SBP incidence among DM diseased individuals could be due to a few different factors. The human immune system is significantly changed by DM, including leukocyte function, which exacerbates liver cirrhosis-2-induced immunological dysfunction (15, 16). Additionally, DM-induced polyneuropathy may result in dyskinesia of the bowel and gastric muscles, which lengthens the time it takes for food to pass through the intestines (16). As a result, there may be a higher chance of bacterial translocation from the gut,

which plays a crucial role in SBP pathogenesis (17).

The current study showed that there is no difference between the two cohorts in terms of hemoglobin, red blood cells, or platelets, but there is a difference between the two cohorts in terms of temperature and white blood cells, which were greater in the SBP cohort than in the non-SBP cohort. Similar to our findings, other research has shown that the ascitic total WBC count rises in SBP cases (18,19). Additionally, Elsadek et al. (20) evaluated demographic, clinical, and laboratory data between the SBP cohort and the sterile ascites cohort and found that the SBP cohort had significantly higher blood ESR, CRP, and PMNL count.

Additionally, we discovered that there is no discernible difference between the two cohorts in terms of (Serum urea - Serum creatinine - SGOT- SGPT- Serum albumin - Serum total bilirubin - Prothrombin time – blood Sodium level - INR) GGT and Alk-ph, however, significantly differed between the two cohorts and were higher in the SBP cohort than the non-SBP cohort.

In agreement with our findings, Gameel et al. (21) showed that SBP with a negative culture and one with a positive culture for total bilirubin, direct bilirubin, albumin, PT, WBCs, BUN, and creatinine did not differ significantly. Furthermore, our results essentially concurred with those of Pelletier et al. (22), who found that the sole distinguishing factor was the elevated blood creatinine in the cohort with a positive ascitic fluid culture. Furthermore, Popoiag et al. (23) found that there were no significant differences between the mean levels of Hb, AST, and ALT.

Compared to the non-SBP cohort, the SBP cohort had a higher prevalence of high alkaline phosphatase and total leukocyte count, but there was no statistically significant difference between the two cohorts for hemoglobin, platelets, AST, ALT, total bilirubin, creatinine, or fasting blood sugar in the studied cases. On the other hand, Elkafoury et al. (25) found that SBP diseased individuals had significantly higher levels of transaminases, total and direct serum bilirubin, and white blood cell counts than ascitic diseased individuals did.

According to the current study, CRP and ESR were greater in the SBP cohort than in the non-SBP cohort. Similar to how CRP levels and WBC may represent therapeutically helpful parameters to "non-invasively" evaluate risk of SBP development, Schwabl et al. (26) demonstrated these metrics. Additionally, Cekin et al. (27) demonstrated that hepatic function may affect CRP generation and levels. Additionally, while having a larger effect size than the WBC, the ability to predict

SBP appears to have clinical utility.

Additionally, the current study demonstrated that there is no discernible difference between the two cohorts with reference to Child Pugh score- MELD score. On the other hand, Mellinger et al. (28) demonstrated that diseased individuals with decompensated cirrhosis who had higher MELD scores and those who had developed hepatic encephalopathy were independently more likely to die in hospitals. Additionally, Obstein et al. (29) showed that MELD and Child Pugh stage C were conventional risk factors for the development of SBP. Seventy percent of the diseased individuals that developed SBP were in Child class C, according to Bandy and Tuttle (32), with the remaining diseased individuals falling into class B (30). Child-Pugh stage C, according to Schwabl et al. (26), implies a high chance of developing SBP.

In the present investigation, the PMN cell count of the two cohorts was significantly different, with the SBP cohort having a higher PMN cell count and the non-SBP cohort having a higher ascitic fluid PGE2 level. It was in line with the results of Luo et al. (9) who looked at how ascitic PGDE2 affected the diagnosis of spontaneous bacterial peritonitis and the prognosis of in-hospital mortality in cirrhotic patients with decompensated cirrhosis. Following antibiotic therapy, they found a statistically significant increase in the mean PGDE2 levels in the blood and ascitic fluid. Weiler's study (31) revealed that when portal hypertension was present, PGE2 tissue levels were considerably lower in the inflammatory gastric antral mucosa of cirrhotic patients. Moreover, these tissue levels were decreased in the inflamed gastric antral mucosa of diseased individuals with ulcers.

The ascitic PMN cell count was higher in the SBP group than in the non-SBP cohort, according to the current study. Our results are in agreement with those of Schwabl et al. (26), who discovered that ascitic fluid PMN count emerged as an independent risk factor for SBP development. As a result of bacterial translocation into the ascitic fluid, a recent study by Lutz et al. (32) found that the relative A-PMN count may operate as an indication of subclinical infection or inflammation, and that this function may be predictive of the emergence of SBP. Additionally, an absolute PMN level of one hundred cells/l has previously been associated with a noticeably increased risk for the emergence of SBP. (26) In addition, it has recently been demonstrated that the absolute A-PMN cell count may also be a predictor of SBP development (33).

The results of the current investigation revealed a substantial difference in PMN cell counts between the

two cohorts, indicating that the higher the ascitic PMN cell count, the higher the hospital mortality rate. Regarding ascitic fluid PGE2, there is a substantial difference between the two cohorts, indicating that the greater the hospital mortality rate for diseased individuals with hepatic cirrhosis, the lower the ascitic fluid PGE2. In agreement with our findings, Aboghiaty et al. (10) found no association between case cohort mortality, ascitic fluid, and serum PGDE2 before and after treatment. Additionally, MELD score and advanced age were independent predictors of death in multivariate analysis (34).

The results of the current investigation revealed that there is no significant negative correlation as reference other measures (such as ascitic fluid PGE2) and PMN cells (Age- Platelets-ESR- Prothrombin time-Sodium level INR). Additionally, there is a strong positive correlation between PMN cells and other measures such as hemoglobin, white blood cells, GGT, alkaline phosphatase, and MELD score, but not between RBC, CRP, serum urea, serum creatinine, SGOT, SGPT, albumin, or serum total bilirubin.

In the SBP cohort, there was a substantial positive association for NLR with diseased individual age, WBC count, neutrophils, lymphocytes, platelets, total and direct bilirubin, PT, and INR, and a negative correlation with albumin, Cr, HB, ALT, and AST, according to a study by Awad et al. (35).

The results of the current study showed that there is no significant positive correlation for Hb and no significant negative correlation for the remaining parameters and ascitic fluid PGE2, but there is a significant negative correlation between ascitic fluid PGE2 and other parameters with regard to WBC.

With sensitivity of 80% and specificity of 55%, negative predictive value of 93%, positive predictive value of 27%, and accuracy of 60%, the current study demonstrated the efficacy of ascitic PMN cell count as a prognostic marker for death in cirrhotic diseased individuals at a cutoff point (more than 210).

Additionally, Garcia-Tsao et al. (36) showed that cirrhotic diseased individuals may exhibit bacteriogenic peritoneal irritation as a prodromal phase of SBP and that the diagnostic cut-off for SBP at 250 PMN cells/L has been selected experimentally. Diseased individuals with an A-PMN cell count of 125 to 250 /l have a higher death rate than diseased individuals with counts below 125/l (35).

A previous study showed that primary SBP prevention significantly reduces the incidence of SBP, the onset of hepatorenal syndrome, and mortality in diseased individuals with certain risk factors (37).

The results of the current study demonstrated the efficacy of ascitic fluid PGE2 as a prognostic marker for death in cirrhotic diseased individuals at a cutoff point (38.4), with sensitivity (93%) and specificity (96%), respectively.

An earlier study showed that the ascitic PGE2 concentration of 32.8 pg/mL, with a sensitivity of 83% and a specificity of 53%, was the best threshold for predicting in-hospital death. Ascites PGE2 had an AUC of 0.7 for predicting in-hospital mortality (38).

The results of the current study demonstrated the efficacy of combined ascitic PMN cells and ascitic fluid PGE2 as a prognostic marker for death in cirrhotic diseased individuals at sensitivity (93%) and specificity (51%), respectively.

The greatest strength of the study is its thorough documentation of the study group, which contains details on the most important clinical, demographic, and laboratory parameters. This afforded us the chance to analyze a big cohort of ascites-positive cirrhotic patients over an appropriate follow-up period. Then, we could do precise comparisons and identify the elements that raise the risk of both getting SBP and passing away.

CONCLUSION

Both blood and ascitic fluid SBP can be identified and eliminated using the diagnostic markers PGDE2 and PMN. In cirrhotic individuals, the combination of ascitic PMN cells and ascitic fluid PGE2 can be employed as a predictive predictor for death. PGE2 at a cutoff point (38.4) as a predictive predictor for mortality in cirrhotic diseased individuals. At a cutoff point (more than 210), ascitic PMN cell count can be utilized as a prognostic predictor for mortality in cirrhotic individuals.

Conflict of interest

All author declare that they have no conflict of interest.

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