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Ascetic Fluid Mannose Binding Lectin in Patients with Spontaneous Bacterial Peritonitis

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Abstract

Background: Spontaneous bacterial peritonitis is very common bacterial infection in patients with cirrhosis and ascites, when first described its mortality exceeded 90% but it has been reduced to approximately 20% with early diagnosis and treatment. Mannose-binding lectin (MBL) is an important protein of humoral innate immune system with multiple carbohydrate recognition domain , it is able to bind to sugar group displayed on the surface of wide range of micro organism and therepy provide first line defense, it is also occur activate complement MBL produced in liver in response to infection and is a part of many other factor termed acute phase protein. We aimed to study the level of ascitic fluid mannose binding lectin in patients with spontaneous bacterial peritonitis. Methods: This study was conducted on 90 patients with cirrhotic ascites with and without spontaneous bacterial peritonitis admitted to Benha University Hospital and Manshyet El Bakriy Hospital. They were classified into two groups, group (I) included 45 cirrhotic patients with spontaneous bacterial peritonitis (35 males and 10 females), aged between 36 - 69 with a mean age of 52.96 ± 7.90 years while group (II) included 45 cirrhotic patients without spontaneous bacterial peritonitis (29 males and 16 females), aged between 28 - 65 with a mean age of 49.49 ± 10.21 years. Results: Regarding the ascetic level of MBL in patients with SBP, it was found in this study that the mean value of MBL level was significantly lower in SBP group than non SBP group. There are statistically significant positive correlations between MBL and Bilirubin, prothrombin time, INR and u MELD values and significant negative correlations between MBL and Platelets and serum ascetic albumin gradient values among patients with SBP. In the present study, the ROC curve shows that the best cut off point for MBL to diagnose cases with ascites with SPB cases was found 1307 with sensitivity 75.56%, specificity of 77.78% and area under curve (AUC) of 77.0%. In this study, by multivariant analysis WBCs, RBCs, serum creatinine, ALT, serum albumin and MBL were significantly determining predictors for SBP positivity. Conclusion: The mean value of ascetic level of MBL was significantly lower in SBP group than non SBP group. Ascitic fluid MBL could be a good predictive and prognostic marker in patients with cirrhosis and spontaneous bacterial peritonitis.

Key words: Ascetic Fluid Mannose Binding Lectin, Spontaneous Bacterial Peritonitis.

1. Introduction

Spontaneous bacterial peritonitis is the most frequent bacterial infection in patients with cirrhosis, followed by urinary tract infection, pneumonia, skin and soft tissue infections, and spontaneous bactermia **[1].**

The occurrence of spontaneous bacterial peritonitis varies according to the studied population. It is estimated that the incidence reaches 3.5% at 1 year in outpatients with decompensated cirrhosis and varies between 7% and 30% in hospitalized patients with cirrhosis and ascites **[2].**

MBL belongs to the class of collectins in the C-type lectin superfamily, whose function appears to be pattern recognition in the first line of defense in the pre-immune host. MBL recognizes carbohydrate patterns, found on the surface of a large number of pathogenic micro-organisms, including bacteria, viruses, protozoa and fungi. Binding of MBL to a micro-organism results in activation of the lectin pathway of the complement system. Another important function of MBL is that this molecule binds senescent and apoptotic cells and enhances engulfment of whole, intact apoptotic cells, as well as cell debris by phagocytes **[3]**.

Mannose binding lectin deficiency has been implicated in the course of viral, bacterial, fungal, and protozoan infection. MBL binds to a range of clinically relevant pathogens, which were isolated from clinical patients. There is also great variation in the binding of MBL to various organisms; Candida albicans, b-hemolytic group A Streptococci and Staphylococcus aureus bind with high affinity, while Clostridium Pseudomonas aeruginosa, Staphylococcus, epidermidis, and Streptococcus pneumonia exhibit low or no binding. It is also observed that some organisms (e.g. Klebsiella sp. and Escherichia coli) show avariable pattern of binding **[4].**

Finally the MBL deficiency predisposes patient with liver cirrhosis to develop spontaneous Bacterial peritonitis **[5].**

The aim of this work is to study the level of Ascitic fluid mannose binding lectin in patients with spontaneous bacterial peritonitis and determine its role in diagnosis of spontaneous bacterial peritonitis.

2. Patients and Methods

Patients

This cross sectional study was carried out on 90 patients with cirrhotic ascites with and without spontaneous bacterial peritonitis admitted to Benha University Hospital and Manshyet El Bakriy Hospital from March 2021 to October 2021. They were classified into two groups,

- **Group (I)** included 45 cirrhotic patients with spontaneous bacterial peritonitis while
- **Group (II)** included 45 cirrhotic patients without spontaneous bacterial peritonitis.

Inclusion criteria:

Adult patients with cirrhotic ascites

Exclusion criteria:

1. Evidence of gastrointestinal bleeding or bacterial infection in the preceding 6 weeks.
2. Treatment with non-absorbable antibiotic in the preceding 6 weeks.
3. Other non peritoneal infection (skin infection, chest infection, biliary tract infection, urinary tract

infection, gastroenteritis, dental infection and meningitis).

4. Secondary bacterial peritonitis
5. Rheumatic heart disease.
6. autoimmune disease.

All patients were subjected to the following:

I- Thorough history taking with particular attention to:

- Manifestations of liver cell failure e.g. Jaundice, lower limb oedema, deterioration of conscious level, bleeding tendency.
- Manifistations suggesting SBP e.g. fever and abdominal pain.
- History suggesting possible complications of SBP e.g. variceal bleeding, hepatic encephalopathy and hepato-renal syndrome.

II- Full general and local examination, looking for:

- Signs of chronic liver disease e.g jaundice, ascites, palmar erythema, spider naevi, liver size, spleen size, lower limb oedema and encephalopathy.
- Signs of SBP such as fever, hypotension, tachycardia, abdominal tenderness and rebound tenderness.

III- Full investigations:

1. **Complete blood picture .**
2. **serum creatinine .**
3. **Liver profile:**
   - Serum alanine transaminase (ALT).
   - Serum aspartate transaminase (AST).
   - Serum bilirubin (Total and direct).
   - Serum albumin.
   - Serum alkaline phosphatase(ALP)
   - Prothrombin time, concentration and INR.

4- Serological tests for viral markers:

- Hepatitis B surface antigen Hepatitis C virus anti body

By using enzyme – linked immunosorbent assay technique (ELISA).

Table (1) Modified Child's Pugh score: [6]
)

Points	1	2	3
Encephalopathy	None	Minimal	Advanced
Ascites	Absent	Controlled	Refractory
Bilirubin(mg/dl)	< 2	2-3	> 3
Albumin (g/l)	> 3.5	3.5 –2.8	< 2.8
Prothrombin(sec)	< 4	4-6	> 6

1. **Child A (5 - 6) points.**
2. Child B (7 - 9) points.
3. Child C (10 -15) points.

6. MELD score:

MELD score is calculated for every patient according to the following equation 9.6 X loge (creatinine mg/dl) + 3.8 x loge (total bilirubin mg/dl) +11.2 X loge (INR) +6.4 [(7).]

7. Updated MELD score:

Updated MELD is calculated for every patient according to the following equation:-

1.27 x In (1 + creatinine (mg/dl) + 0.94 x In (1 + bilirubin mg/dl) + 1.66 x In (1 + INR) [(8).]

8. Abdominal ultrasonography

Real time abdominal Ultrasonography was done for all patients included in the study by (Mindray dc 30) for evaluation of:

- Liver: size, tecture, border, reflectivity, homogenity, periportal thickening, hepatic veins and pattern.
- Focal lesion(s): number, site, size, shape, echogenecity, halo sign and vascularization by color Doppler assessment Portal vein: diameter, patency, direction of flow, respiratory variation and velocity by color Doppler assessment.
- Spleen: size, splenic vein diameter, collaterals.

9. Diagnostic abdominal paracentesis:

It was done for all patients included in this study including Patients with cirrhosis and ascites at admission and Patients who develop symptoms or signs of SBP during hospitalization i.e. fever, abdominal pain or changes in gastrointestinal motility (vomiting, diarrhea or ileus).

The technique of paracentesis was explained to the patient and it was done under aseptic precautions using a wide bore needle. The needle was introduced in the right lower quadrant while the patient lies in supine position.

This technique is accomplished by displacing (with one gloved hand) the skin approximately 2 cm downward and then slowly inserting the paracentesis needle mounted on the syringe held in the other hand.The skin is not released until the needle has penetrated the peritoneum and fluid flows [(9).]

The ascitic fluids were aspirated from each patient and they were cheked for

1. **Biochemical tests including:**
   A. Total protein content.
   B. Albumin.
   C. Glucose.
2. **WBCs (total and differential):**

SBP is diagnosed when PMN count in ascitic fluid ≥ 250 cell/mm in the absence of data compatible with secondary peritonitis (i.e. gastro-intestinal perforation).

3. Serum - ascites albumin gradient (SAAG):

SAAG: The serum ascites albumin gradient, which is based on the difference between the albumin level of serum and of ascitic fluid, may be used to assess the extent of ascites [10]

4. Ascitic fluid mannos biniding lectin by

A.Sampling [11]:

1. Blood samples:

- Blood samples (about 10 ml) were collected under complete aseptic technique from all individuals using a serum separator tube (SST) and allow samples to clot for 30 minutes before centrifugation for 15 minutes at 1000 x g, serum removed divided into small aliquots one was stored at - 20° C till time of use for LFTs.
- A second blood sample was added to EDTA for complete blood count and tested immediately

2. Ascetic fluid sample:

15 ml of ascetic fluid was obtained under complete aseptic condition and divided into:

- 5-10 ml was introduced into blood culture broth bottles at bedside and incubated at 37 c.
- 3 ml was introduced into purple top tube for cell count; if TLC> 500 cells/ mm3 [12] another 3ml was introduced into red top tube for routine chemistries (LDH, glucose, albumin and protein).

B.Ascitic fluid examination [11]:

- Direct microscopic examination of ascetic fluid was done for each Sample, each was concentrated by centrifugation and the supernatant aseptically aspirated with a sterile pipette, leaving approximately 1ml liquid in which to mix the sediment thoroughly and stained with Gram stain to visualize rods, cocci, white blood cells, red blood cells, or squamous epithelial cells.
- 10 ml of each sample cultured on blood broth biphasic culture bottles from oxoid and incubated at 37c and subcultured every 48 hrs on culture media as Blood agar and MacConky agar to reveal occurrence of growth. Specimen discarded after 21 days if no growth occurred.
- Identification of bacterial growth was carried out by colonal morphology, Gram stained films, and biochemical tests [13].

C. Assessment of ascitic ascitic fluid mannose binding lectin:

Human Mannose Binding Lectin (MBL) ELISA Kit is used to assay the Mannose Binding Lectin (MBL) in the sample of human's serum, blood plasma, and other related tissue Liquid.

3. Results

Table (1) Demographic features of the studied patients.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
Gender	**Male**	35 (77.8%)	29 (64.4%)	0.163
	Female	10 (22.2%)	16 (35.6%)	
Age	**Mean ± SD**	52.96 ± 7.90	49.49 ± 10.21	0.075
	Range	36 – 69	28 – 65	

***= Significant SBP = spontaneous bacterial peritonitis SD = standard deviation**

SBP tends to be more common in males than females. However of no significant value.

The mean age of patients with SPB was higher than patients without SBP without significant value.

Table (2) Clinical presentation of the studied patients.

	Ascites with SBP No. (%)	Ascites without SBP No. (%)	P-value
Abdominal pain	25 (55.6%)	0 (0.0%)	***0.000**
GIT Bleeding	20 (44.4%)	9 (20.0%)	***0.013**
Encephalopathy	16 (35.6%)	7 (15.6%)	***0.030**
Fever	26 (57.8%)	0 (0.0%)	***0.000**
Jaundice	27 (60.0%)	17 (37.8%)	***0.035**

*** = Significant GIT = gastro intestinal tract**

SBP = spontaneous bacterial peritonitis

Cirrhotic patients with SBP complained mostly from abdominal pain , GIT bleeding , hepatic encephalopathy , fever and Jaundice with a high statistical significance.

Table (3) The blood picture in studied patients.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
Hemoglobin (12-17g/dl)	**Mean ± SD**	10.43 ± 1.95	11.34 ± 1.65	* **0.019**
White blood cells (4-11 Thousands/ cmm)	**Mean ± SD**	9822.22 ± 4183.57	6793.33 ± 1946.72	* **0.000**
Red blood cells (4 -6 Millions/ cmm)	**Mean ± SD**	3.58 ± 0.73	4.01 ± 0.50	* **0.002**
Platelet (150-450 Thousands/ cmm)	**Mean ± SD**	121.82 ± 60.11	124.42 ± 26.04	0.791

***= Significant SBP=spontaneous bacterial peritonitis SD = standred deviation**

White blood cells were significantly higher in patients with spontaneous bacterial peritonitis. While hemoglobin and red blood cell are statistically significant lower in patient with spontaneous bacterial peritonitis. There is no significant difference between two groups as regard platelets count.

Table (4): Liver function and serum creatinine of the studied patients.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
Creatinine (0.5-1mg/dl)	**Mean ± SD**	1.54 ± 0.58	1.03 ± 0.37	*
	Range	0.7 – 2.8	0.38 – 1.88	**0.000**
Bilirubin (0.2-1.3mg/dl)	**Median (IQR)**	2.6 (1.0 – 3.46)	1.5 (0.99 – 2.1)	*
	Range	0.4 – 10.9	0.59 – 2.4	**0.010**
ALT (9-52U/L)	**Mean ± SD**	46.58 ± 19.30	38.16 ± 14.81	*
	Range	10 – 88	17 – 78	**0.023**
AST (14-36U/L)	**Mean ± SD**	39.42 ± 15.15	34.56 ± 12.93	0.105
	Range	18 – 88	16 – 58	
ALP (175-420U/L)	**Mean ± SD**	95.36 ± 31.18	86.07 ± 30.34	0.156
	Range	55 – 197	38 – 182	
Serum Albumin (3.5-5g/dl)	**Mean ± SD**	2.60 ± 0.73	2.98 ± 0.55	*
	Range	1.6 – 4.4	2 – 4.8	**0.006**
Prothrombin time(12.5)	**Mean ± SD**	20.01 ± 4.90	17.9 ± 3.62	*
	Range	11.2 – 29	12.4 – 27.02	**0.022**
INR(0.9-1.1)	**Mean ± SD**	1.69 ± 0.43	1.44 ± 0.31	*
	Range	1 – 2.6	1.01 – 2.23	**0.003**

***=Significant ALT = alanine amino transferase,**
INR = international normalized ratio SBP = spontaneous bacterial peritonitis
SD = standard deviation .ALP = alkaline phosphates

The mean value of creatinine, ALT , Prothrombin and INR were significantly higher in cirrhotic patients with SBP group (I) than in the cirrhotic patients without SBP group (II) . the mean value of serum albumin was significant lower in patient with SPB. no statistical significant difference between the two groups as regards, AST and ALP.

Table (5) Serologic hepatitis viral markers in the studied patients.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
HCV Ab	**Positive**	36 (80.0%)	42 (93.3%)	0.063
HBs Ag	**Positive**	9 (20.0%)	3 (6.7%)	0.063

***= Significant SBP = spontaneous bacterial peritonitis**
HCVAb = hepatitis C virus anti bodies HBs Ag = hepatitis B surface antigen

No statistically significant difference between the studied groups as regards to HCV Ab and HBsAg.

Table (6) The severity of liver cirrhosis assessed by Child score and MELD scores among the studied patients.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
Child score	**Mean ± SD**	9.53 ± 2.26	7.95 ± 1.61	*
	Range	6 – 13	7 – 12	**<0.001**
Child grade	**B**	18(40.0%)	31(68.9%)	*
	C	27(60.0%)	14(31.1%)	**0.005**
MELD	**Mean ± SD**	15.98 ± 5.28	13.09 ± 3.62	*
	Range	6 – 28	7 – 20	**0.003**
u MELD	**Mean ± SD**	3.58 ± 0.74	3.19 ± 0.43	*
	Range	2.3 – 5.4	2.3 – 3.8	**0.003**

*** = Significant SBP = spontaneous bacterial peritonitis**
MELD = Model for end stage liver disease
u MELD = update model for end stage liver disease SD = standred deviation

Child C were significantly higher in cirrhotic patients with SBP than in cirrhotic patients without SBP. The mean values of MELD, u MELD were significantly higher in group (I) than in group (II) with statistical significance

Table (7) Ultrasound findings of studied groups.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
PVD(mm)	**Mean ± SD**	11.59 ± 2.18	11.44 ± 1.98	0.741
	Range	8 – 16	8 – 15	
Splenomegaly >13cm		42 (93.3%)	33 (73.3%)	* **0.010**
HCC		0(0.0%)	0 (0.0%)	**NA**
Ascites		45 (100.0%)	45(100.0%)	

*** = Significant**
PVD = Portal vein diameter HCC=Hepatocellular carcinoma
SBP = spontaneous bacterial peritonitis SD = standred deviation.
Splenomegaly was statistically significant in SBP group**.**

Table (8) Ascitic fluid analysis in studied groups.

		Ascites with SPB No. = 45	Ascites without SPB No. = 45	P-value
Polymorph nuclear leucocytes (ul)	**Mean ± SD**	576.67 ± 167.82	91.69 ± 24.13	*
	Range	300 – 1030	38 – 160	**0.000**
Albumin (g/dl)	**Mean ± SD**	0.65 ± 0.32	0.92 ± 0.25	*
	Range	0.2 – 1.3	0.2 – 1.5	**0.000**
Protein (g/dl)	**Mean ± SD**	0.60 ± 0.28	2.07 ± 0.45	*
	Range	0.3 – 1.2	1.2 – 3	**0.000**
Glucose (g/dl)	**Median (IQR)**	48 (44 – 54)	55 (45 – 101)	*
	Range	38 – 65	34 – 350	**0.001**
SAAG(g/dl)	**Mean ± SD**	2.24 ± 0.84	2.2 ± 0.44	0.796
	Range	1.1 – 3.8	1.3 – 3	

SAAG = Serum ascetic albumin gradient
*** = Significant SBP = spontaneous bacterial peritonitis.**
SD = Standard deviation.

As regard to ascetic fluid analysis of studied groups, the mean value of polymorph nuclear leucocytes were significantly higher in SBP group than non SBP group. While the mean values of ascetic glucose ,ascetic albumin and ascetic protein were significantly lower in SBP group than in non SBP group. no statistical significant difference between the two groups as regards serum ascetic albumin gradient.

Table (9) Ascitic fluid MBL level in studied groups.

		Ascites with SBP No. = 45	Ascites without SBP No. = 45	P-value
MBL(ng/ml)	**Mean ± SD**	1303.80 ± 313.98	1702.64 ± 512.81	*
	Range	1000 – 2700	1000 – 3100	**<0.001**

*** = Significant**
MBL =mannose binding lectin SBP = spontaneous bacterial perotinitis
SD = standard deviation.itis
The mean value of MBL level was significantly lower in SBP group than non SBP group.

Table (10) Correlation of MBL with some variables in SBP group.

	MBL Ascites with SBP	
	r	**P-value**
White blood cell	-0.184	0.226
Platelets	**-0.401****	**0.006**
Serum Creatinine	-0.179	0.239
Serum Bilirubin	**0.405****	**0.006**
Serum Albumin	-0.232	0.125
Prothrombin time	**0.374***	**0.011**
INR	**0.354***	**0.017**
Child score	0.074	0.627
MELD	0.236	0.118
u MELD	**0.341***	**0.022**
Polymorph nuclear leucocyte	0.214	0.157
Ascitic Albumin	0.074	0.629
Ascitic protein	-0.290	0.053
Ascitic glucose	0.232	0.125
SAAG	**-0.349***	**0.019**

***= significant INR= international normalized ratio**
MELD = model for end stage liver disease
SAAG = Serum ascetic albumin gradient
u MELD = update model for end stage liver disease
SBP=spontaneous bacterial peritonitis INR=international normalized ratio.

There is a statistically significant positive correlation between MBL and Bilirubin, Prothrombin time, INR ,u MELD.
There is a statistically significant negative correlation between MBL and Platelets and SAAG.

Table (11) ROC curve analysis for MBL as a marker for SBP.

Cut off point	AUC	Sensitivity	Specificity	PPV	NPV
1307(ng/ml)	0.770	75.56	77.78	77.3	76.1

AUC: Area under curve; **PPV:** positive predictive value; **NPV:** negative predictive value

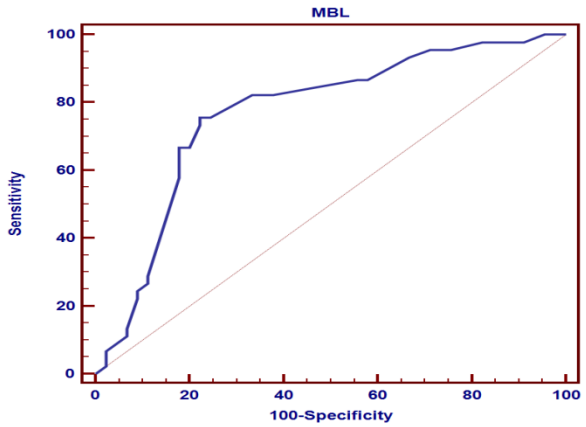

Fig.(1) Receiver operating characteristic curve (ROC) for MBL as a diagnostic marker for SPB.

The previous ROC curve shows that the best cut off point for MBL to diagnose cases with ascites with SPB cases was found 1307 with sensitivity 75.56%, specificity of 77.78% and area under curve (AUC) of 77.0%, PPV 77.3, NPV 76.1.

Table (12) Multivariate logistic regression analysis for predictors of ascites with SBP (using Forward:Wald method).

	B	S.E.	Wald	P-value	Odds ratio (OR)	95% C.I. for OR	
						Lower	Upper
WBCs	0.001	0.000	9.154	**0.002**	1.001	1.001	1.001
RBCs	-1.259	0.584	4.651	**0.031**	0.284	0.09	0.892
Creatinine	1.884	0.768	6.023	**0.014**	6.582	1.462	29.642
ALT	0.053	0.022	5.962	**0.015**	1.055	1.011	1.101
Serum Albumin	-1.633	0.649	6.336	**0.012**	0.195	0.055	0.697
MBL	-0.002	0.001	6.562	**0.010**	0.998	0.996	0.999

***= significant , MBL = mannose binding lectin,**
WBC=white blood cell
RBC= red blood cell
ALT=alanine amino transferase

By multivariate analysis WBCs, RBCs, serum creatinine, ALT, serum albumin and Ascitic fluid MBL were significant predictors of SBP.

4. Discussion

In this study, SBP tends to be more common in males than females, however of no significant value. **Mahmoud et al. [14]** evaluated the role of mannose-binding lectin (MBL) in cirrhotic patients with and without spontaneous bacterial peritonitis; they found that there was no significant statistical difference between the studied groups with regards to gender.

The present study revealed that the mean age was higher in patients with SBP than in patients without SBP , however of no siginificant value. these results were in agreement with the study done by **Lutz et al., [15**] which documented that SBP patients were elder than non SBP patients

Analysis of the results showed that patients with SBP commonly presented by abdominal pain with significant difference compared to non SBP group and this goes in agreement with. **Paul et al. [16]** who detected that most patients of SBP have signs clearly suggestive of peritoneal infection, especially abdominal pain.

in the present study, 44.4% of SBP cases had gastrointestinal bleeding and this was close to that reported by **Lutz et al., (15) and El- Toukhy and Emam [17]** who stated that (27% , 22.2% , 30%) of SBP cases respectively had gastrointestinal bleeding.

In our study, hepatic encephalopathy was detected in 35.6% of SBP and 15.6% of non-SBP cases with high significant difference, these results were close to that reported by **Paul et al. [16[** who stated that hepatic encephalopathy was detected in 24.5% of SBP cases.

In clinical examination of studied patients, fever was predominant in cases with SBP. This goes in agreement with **Paul et al. [16]** who detected that most patients of SBP have signs clearly suggestive of peritoneal infection, especially fever; so, fever is considered one of the characteristic clinical sign of SBP.

Jaundice was detected in 60% of SBP group, with high statistically significant difference between groups I and II. These results were close to that reported by **Paul et al. [16]** detected that jaundice was in 39% in SBP patients.

Laboratory investigations in this study revealed that hemoglobin value was significantly lower in patients with SBP and this goes in agreement with that elicited by **Mohamed et al, [18] and Wojtacha et al., [19]** who reported decreased level of hemoglobin in SBP patients, this can be explained that patients with more severe liver disease have anemia. White blood cells were significant higher in patients with spontaneous bacterial peritonitis. this result goes in agreement with that reported by **Cholongitas et al., [20]** as leucocytosis was higher in SBP patients.

There is no statistical significant difference between groups I and II as regards platelets, This result was in agreement with **Paul et al. [16]**, but this difference may not related to SBP, rather to the severity of the liver disease.

In this study, the mean value of serum creatinine was significantly higher in cirrhotic patients with SBP group (I) than in the cirrhotic group (II) without SBP, and this goes in agreement with **Tsung et al. [21]** who stated that renal dysfunction occurs in patients with SBP and it is independent predictor of mortality**.**

In this study the level of serum bilirubin was siginificant higher in cirrhotic patients with SBP, these results goes in agreement with **Ruiz et al. [22]** who found that patients with SBP frequently develop a rapidly progressive impairment in systemic haemodynamics, leading to severe hepatic failure.

Also, in this study, the serum ALT level was siginificant high in patient with SBP this result agree with **Coral et al. [23]** who suggesting that liver function may be worsened by bacterial infection and severity of liver disease.

As regard serum albumin level, in this study it was significantly lower in SBP patients than in non SBP patients; this result goes in agreement with **Ruiz et al. [22]**, who found that patients with SBP frequently develop a rapidly progressive impairment in systemic haemodynamics, leading to severe hepatic failure.

Regarding to the prothrombin time in this study, the prothrombin time was significantly high in patients with SBP, this result was in agreement with **Umgelter et al. [24]**, who found that high prevalence of disturbance in prothrombin time among patients suffering from SBP.

As regards the cause of liver cirrhosis in this study, the results found that chronic hepatitis C is the cause in most of patients and that was in agreement with study done by **Nabiel et al., [25]** in which most of patients were chronic hepatitis C virus.

Regarding to the severity of liver disease, this study showed that Child C were significantly higher in cirrhotic patients with SBP than in cirrhotic patients without SBP, this result matched with that reported by **Nabiel et al., [25], El-Toukhy and Emam [17] and Cirera et al, [26]** who showed that most of patients with SBP were classified child C.

In the present study, it was found that the mean values of MELD and uMELD score were significantly higher in group (I) than in group (II) with statistical significance, This goes in agreement with **Obstein et al. [27]** who suggested that MELD score was associated with SBP risk.and with **Lutz et al., [15]** who suggested that MELD score was higher in patients with SBP.

The ultrasonographic examination in the present study detected that splenomegaly was significantly higher in SBP group ,this goes in agreement with **El-Toukhy and Emam [17]** in which splenomegaly was significantly higher in SBP patients

As regard Ascitic fluid analysis the mean value of polymorph nuclear leucocytes was higher in SBP group than non SBP group, however of significant value. This result goes in agreement with **Giron- Simbrunner et al. [28]** who reported higher ascetic fluid Polymorph nuclear leucocytes in SBP patients than non SBP patients.

As regards to ascetic fluid albumin level it was significantly lower in patients with SBP, this agrees with **Paul et al., [16],** this could be explained by the reduced functional activity of hepatocyte and innate immune system exhaustion causing what was called cirrhosis associated immune dysfunction (CAID) **[29].**

In this study, the mean value of ascitic protein was siginificantly lower in SBP group than in non SBP group, this goes in with **Paul et al. [16]** who denoted that patients with poor synthetic function have diminished level of protein in ascitic fluid that correlate with low level of opsonization and this play a role in SBP susceptibility and denoted also AF total protein < 1 g/dl is important predictor for SBP.

In this study, we found the mean value of ascitic glucose was siginificantly lower in SBP group than in non-SBP group, This goes in agreement with **Tsung et al. [21]**, who reported that lower level of ascitic glucose level in SBP patients and also considered that lower ascitic glucose level is independent predictive factor of overall survival rates in cirrhotic patients with SBP. Also**, Mahmoud et al. [14]** found that the glucose level in the ascetic fluid was significantly lower SBP.

The present study revealed that the mean value of SAAG was > 1.1 g/dl in both SBP and non-SBP groups but without significant difference, which confirms that etiology of ascites was portal hypertension in these patients. This finding is in concordance with **Agarwal et al., [30]** study which suggested that SAAG levels are > 1.1g/dl in all ascites due to portal hypertension irrespective of infection

Regarding the ascetic level of MBL in patients with SBP, it was found in this study that the mean value of MBL level was significantly lower in SBP group than non SBP group, this was agreed by results of **Esmat et al**. **[31]** .

Altorjay et al. [32] found that in absolute MBL deficiency (MBL level <100 ng/ml), the time to first infection was shorter, the impressive fact was the considerable variations between individuals in the studied groups regarding their MBL and this may be explained by the high genetic heterogenecity of MBL expressing genes.

Mohamed et al. [18] assessed ascitic fluid MBL in liver cirrhosis and spontaneous bacterial peritonitis and showed that ascitic fluid MBL level was significantly lower in patients with SBP.

In this study, there are statistically significant positive correlations between MBL and bilirubin, prothrombin time, INR and uMELD values and significant negative correlations between MBL and platelet and serum ascetic albumin gradient values among patients with SBP.

Mohamed et al. [18] found that MBL had a significant negative correlation with ascitic total leukocytic count (TLC), also with serum creatinine, bilirubin, PT, INR and MELD score among SBP patients.

In the present study, the ROC curve shows that the best cutoff point for MBL to diagnose cases with ascites with SPB cases was found 1307 with sensitivity 75.56%, specificity of 77.78% and area under curve (AUC) of 77%.

Mahmoud et al. [14] showed that the measurement of th sr MBL plays a significant role in the differential diagnosis of SBP from non SBP cirrhotic patients with sensitivity 71.4%, and specificity 69.4%, at cutoff point of 1202.5 ng/ml.

In this study, we found that serum creatinine serum ALT, serum Albumin,WBC, MBL and RBC are important predictors for development of SBP and are considered as risk factos for development for SBP. This goes in agreement with **Navasa et al. [33]** who stated that high serum bilirubin, deranged renal functions, high MELD score are important predictors for development of SBP. **Mohamed et al. [18]** found that fever, TLC, platelets, creatinine, MBL, glucose and polymorphs were independent predictors for SBP development.

5. Conclusion

The mean value of ascetic level of MBL was significantly lower in SBP group than non SBP group. Ascitic fluid MBL could be a good predictive and prognostic marker in patients with cirrhosis and spontaneous bacterial peritonitis.

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