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Clinical Profile of patients with Ascitic Fluid Infection at Ain Shams University Hospitals

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ABSTRACT

Background: Ascites is a common problem in patients with chronic liver disease. About 60% of patients with cirrhosis will develop ascites. Patients with chronic liver disease and cirrhosis frequently develop infections of the ascitic fluid.

Aim: The aim of this study is to assess the clinical profile of patients with ascetic fluid infection admitted to Tropical Medicine department at Ain Shams University hospitals.

Patients and Methods: The current Cross-sectional study was conducted at The Tropical Medicine Department, Ain Shams University on 87 Egyptian patients with chronic liver disease and ascites during the 12-months period from June 2017 to May 2018 by collecting their clinical, laboratory and radiological data.

Results: The frequency of infected ascites among the studied patients with chronic liver disease and ascites was 31%. The main presenting symptom of infected ascites was abdominal pain (37%) and the most common clinical sign was lower limb edema (81%). The most frequently isolated micro-organism was E.coli that was detected in 7% of patients with infected ascites. Among the 27 patients with infected ascites, 12 patients responded to the third generation cephalosporins, nine patients responded to Meropenem.

Conclusion: Infection of the ascitic fluid is frequent among patients with chronic liver disease and cirrhosis admitted to Ain Shams University Hospitals. Almost one third of the ascitic patients developed at least one attack of spontaneous bacterial peritonitis or bacterascites. Monomicrobialbacterascites is more frequent than polymicrobialbacterascites and E coli is the most common isolated organism. Third-generation, broad-spectrum cephalosporins remain a good initial therapy for patients who do not have allergy to cephalosporins. Alternative antibiotics such as Meropenem and pipercillin-tazobactam should be considered for patients for patients who fail to improve on traditional antibiotic regimens.

Keywords: Ascites, Spontaneous bacterial peritonitis, Ascitic fluid infection.

INTRODUCTION

Ascites is a common problem in patients with chronic liver disease. About 60% of patients with cirrhosis will develop ascites [(1)]. The main pathophysiology of ascites is progressive increase in portal venous pressure as a result of increased intrahepatic resistance caused by cirrhosis [(2)]. Portal hypertension increases the hydrostatic pressure at the sinusoidal level and causes some hemodynamic changes including the splanchnic vasodilation, reduced systemic resistance, increased plasma volume and cardiac output. These alterations stimulate the renin-angiotensin-aldosterone system leading to renal sodium and water retention that result in ascites [(3)].

Patients with chronic liver disease and cirrhosis frequently develop infections of the ascitic fluid. Spontaneous bacterial peritonitis (SBP) is defined as an ascitic fluid infection without an evident intraabdominal surgically treatable source, it primarily occurs in patients with advanced cirrhosis [(4)]. The diagnosis is established by positive ascitic fluid bacterial culture and elevated ascitic fluid absolute polymorphonuclear leukocyte (PMN) count ($\geq$250 cells/mm^3). SBP occurs in one third of patients with cirrhosis and is associated with hospital mortality of 20% to 40% [(5)]. Patients who recover an attack of SBP have an increased risk of recurrence of 40% to 70% in one year and poorer survival on follow-up [(4)].

Other variants of ascitic fluid infections include culture-negative neutrocytic ascites, monomicrobial non-neutrocyticbacterascites and polymicrobialbacterascites. These variants of infected ascites are distinguished from classic SBP largely by ascitic fluid analysis. It is important to recognize these variants in at-risk patients who do not fulfil classical definitions of SBP [(6)].

The bacterial isolates in SBP may differ from the isolates detected in neutrocytic ascites, monomicrobial non-neutrocyticbacterascites and polymicrobial bacterascites. Gram-negative organisms are the most common organisms in SBP [(7)].

Third generation cephalosporins are commonly used as emperical treatment of infected ascites with cirrhosis as they cover both enterobactericae and non-enterococcal streptococci [(8)]. The development of multidrug resistant strains raise the need to investigate other antibiotic regimen based on the prevalence and the antimicrobial resistance pattern of the infection [(9)].

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AIM OF THE WORK

The aim of this study is to assess the frequency, clinical profile, bacteriological patterns and outcome of spontaneous bacterial peritonitis and other variants of ascitic fluid infections in patients of liver cirrhosis admitted to Tropical Medicine department at Ain Shams University hospitals. The study will also aim to investigate the bacterial isolates and antibiotic sensitivity and resistance patterns in different variants of ascitic fluid infections.

PATIENTS AND METHODS

This cross-sectional study enrolled 87 patients with chronic liver disease and ascites above the age of 18 years old admitted at the Tropical Medicine Department of Ain Shams University hospitals during the period from June 2017 to May 2018. The study was approved by the Ethical Committee of Faculty of Medicine, Ain Shams University. An informed consent was obtained from each patient or his relatives prior to enrollment.

Patients with other causes of ascites other than chronic liver disease as tuberculous or malignant ascites and patients who received antibiotics one week prior to hospital admission were excluded.

Enrolled patients were subjected to the following:

A. Complete history taking with special consideration of the presence of abdominal pain, fever, history of ascitic fluid infection, history of recent tapping in last month, deteriorated level of consciousness and any previous endoscopic maneuvers for intervention for oesophageal varices.
B. Thorough clinical examination with special stress on signs of chronic liver disease and portal hypertension like hepatomegaly and splenomegaly, fever, jaundice, lower limb oedema, signs of hepatic encephalopathy, grade of ascites and, abdominal tenderness and rebound tenderness.
A. Laboratory investigations:
    1) Complete blood count, CRP.
    2) Liver profile (including coagulation profile, liver enzymes, serum albumin and serum bilirubin).
    3) Renal profile including serum creatinine, serum sodium and serum potassium levels.
    4) Ascitic fluid analysis:
        a. Ascitic cell count.
        b. Biochemical assay of albumin, LDH, glucose, total proteins levels.
        c. Culture and sensitivity.
B. Abdominal ultrasonography with special stress on internal echoes of ascitic fluid, hepatic focal lesion and patency of portal vein, hepatic veins and inferior vena cava.

Enrolled patients were classified into two groups:

1. **Group A:** includes patients who had infected ascites. The diagnosis was based on fever, abdominal tenderness, leukocytosis, elevated CRP, ascitic cell count and ascitic culture and sensitivity. Those patients received empirical antibiotics in the form of third generation cephalosporins till the result of the ascitic culture and sensitivity. Monitoring of the response was guided by improvement of clinical signs (fever and abdominal tenderness) and improvement of ascitic cell count taken after 48 hours from the start of antibiotic treatment.
2. **Group B:** includes patients did not have infection of ascites.

Statistical analysis:

Baseline demographic and clinical characteristics were analyzed descriptively using Student t-tests, ANOVA or Kruskall–Wallis test as appropriate for continuous variables and Chi-square or Fisher's exact tests were used for categorical variables. Bacterial counts were examined in box-plots as continuous variables. A Kruskal–Wallis one-way analysis of variance test tested for a significant overall shift in bacterial levels in cases and controls and the Mann–Whitney *U*-test examined identified sample pairs. Comparison of bacterial counts before and after antibiotic treatment will be assessed by Paired t-test. Pearson r correlation test was used to assess the relation between bacterial strain and ascetic fluid infection variant. Logistic regression was used to predict ascites infection in patients with liver disease. Results were expressed as mean values± S.D. Statistical analysis was performed using SPSS version 22 (SPSS, IBM Inc., NC, USA) and GraphPad Prism software (GraphPad Software Inc., CA, USA).

RESULTS

The present study enrolled 87 Egyptian patients with chronic liver disease and ascites divided into two groups (group A included 27 patients with infected ascites and group B included 60 patients with non-infected ascites). There was no significant difference in age between the two study groups (mean age in group A was 52.11±12.99 years old and in group B, it was 53±54.77 years). Among studied patients in group A, 17 patients were males (63%) and 10 patients were females (37%) and in group B 36 patients were males (60%) and 24 patients were females (40%). Chronic hepatitis C and HCV related cirrhosis was the primary liver disease etiology in the majority of enrolled patients 59 (67.82%) patients.

Table (1): History and clinical symptoms of the enrolled patients.

Parameter No./%	**Group A N: 27**	**Group B N:60**	**P value**
Previous history of SBP; n (%)	3 (11)	3 (5)	0.3690
History of recent tapping in last month; n (%)	9 (33)	25 (42)	0.4880
Fever; n (%)	8 (30)	2 (3)	0.0133**
Encephalopathy; n (%)	7 (26)	8 (13)	0.2185
Jaundice; n (%)	10 (37)	15 (25)	0.3081
Abdominal pain; n (%)	10 (37)	2 (3)	<0.0001**

**Highly significant: <0.01, 0.0001

As shown in Table 1, more patients in group A had previous history of SBP compared to patients in group B where the percentage of patients who had history of SBP in group A and group B were 11% and 5%, respectively but without statistical significance. Fever and abdominal pain were significantly detected in group A.

Table (2): Physical signs among the enrolled patients.

Parameter No./%	**Group A N: 27**	**Group B N:60**	**P value**
Fever	8 (30)	2 (3) ***	<0.0001**
Lower limb edema	22 (81)	14 (23)	<0.0001**
Enlarged liver	16 (59)	31 (52)	0.6428
Enlarged spleen	11 (41)	23 (38)	1.0000
Grade of ascites Moderate Tense	 6 (22) 21 (78)	 40 (67) 20 (33)	 0.0002** <0.001**
Abdominal tenderness and rebound tenderness	10 (37)	2 (3) ##	<0.0001**

** Highly significant difference

A highly significant difference was observed between the two groups in the occurrence of fever, abdominal pain and tenderness, lower limbs edema, grade of ascites. The percentage of patients who had fever was 30% in group A versus 3% in group B. Abdominal tenderness and rebound tenderness were reported in 37% and 3% of patients in groups A and groups B respectively. Lower limb edema was detected in 81% in group A and 23 % in group B. Also, the percentage of patients who had tense ascites was 78 % in group A and 33% in group B.

Table (3): Complete blood picture in the studied groups.

	Group	Mean	Std. Deviation	P value
White blood cells count (4-10 * 10^3/ul)	A (N=27)	9.1481	2.95832	.005*
	B (N=60)	6.7083	2.81680	
PML % (50-80 %)	A (N=27)	70.4308	10.26321	0.19
	B (N=60)	67.1368	10.64352	
Hemoglobin (12-15 g/dl)	A (N=27)	10.2556	2.36193	0.78
	B (N=60)	10.9517	2.05092	
Platelet (150-410 * 10^3/ul)	A (N=27)	166.2963	12.09709	0.81
	B (N=60)	159.0167	17.59383	

* Significant difference

Table 3 shows a statistically significant difference between the two groups in total white blood cell count.

Table (4): Liver functions in the enrolled patients.

Parameter	Group	Mean ±SD	P value (Sig: 2 tailed)
Total serum bilirubin (0.3 – 1 mg/dl)	Group A: N=27	4.7037±1.84456	.045*
	Group B N=60	2.7300±1.19631	
Direct bilirubin (up to 0.2 mg/dl)	Group A: N=27	3.2308±1.93891	.005*
	Group B N=60	1.3867±0.26671	
AST (13 – 39 U/L)	Group A: N=27	102.3333±12.90333	.289
	Group B N=60	78.2717± 8.81394	
ALT (7 – 52 U/L)	Group A: N=27	42.3704 ± 5.26677	.972
	Group B:N=60	41.9333± 5.35295	
Total serum Proteins (6 – 8.3 g/dl)	Group A: N=27	6.2571 ± 1.33288	.646
	Group B N=60	6.4026± 1.05151	
Serum Albumin (3.5 – 5.7 g/dl)	Group A: N=27	2.1852 ±0.42940	.016*
	Group B N=-60	2.4517±0.53851	

* Significant difference

As shown in Table 4, significant differences were observed between the two studied groups in total and direct serum bilirubin. Serum albumin was significantly lower in group A than in group B among the studied patients.

Table (5): Renal functions and electrolytes among the enrolled patients.

Parameter	Group	Mean ±SD	P value (Sig: 2 tailed)
BUN (5 – 23 mg/dl)	Group A: N=27	26.307±8.340	0.834
	Group B: N=60	27.59±8.269	
Creatinine (0.6 – 1.2 mg/dl)	Group A: N=27	1.4444±0.01955	0.536
	Group B: N=60	1.8767±0.16522	
Sodium (136 – 145 mmol/l)	Group A: N=27	132.3333±5.51222	0.211
	Group B: N=60	129.1983±17.41558	
Potassium (3.5 – 5.1 mmol/l)	Group A: N=27	5.5852± 1.71406	0.778
	Group B N=60	6.3317±1.90372	
Serum Calcium (8.6 – 10.3 mg/dl)	Group A: N=27	2.4517±0.53851	0.770
	Group B N=60	7.8994±1.08836	
Serum PO4 (2.5 – 5 mg/dl)	Group A: N=27	3.8286±1.50570	0.353
	Group B N=60	3.4390±1.62540	
Uric acid (4.4 – 7.6 mg/dl)	Group A: N=27	7.3960±1.45814	0.835
	Group B N=60	7.1976±2.61892	
Serum Magnesium (1.8 – 2.6 mg/dl)	Group A: N=27	2.0571±0.44449	0.033*
	Group B N=60	1.8265±0.33603	

*Statistically significant difference

Comparing various biochemical data, a statistical significant difference between the two groups was observed only in the levels of serum magnesium

Table (6): INR and C-reactive protein in the studied groups.

Parameter	Group	Mean ±SD	P value (Sig: 2 tailed)
INR (0.8 – 1.2)	Group A: N=27	1.8822±0.11124	.019*
	Group B N=60	1.5118±0.2030	
C-reactive Protein (<6)	Group A: N=27	59.0200±4.87468	.036*
	Group B N=60	24.1943±9.3091	

*Statistically significant difference

A statistical significant difference between the two groups was observed in both INR and C reactive protein which were significantly higher in group A than in group B.

The portal vein, hepatic vein and inferior vena cava were assessed in the two groups, however, no statistically significant differences were found in any of the three parameters.

Table (7): Ascitic Fluid analysis.

Parameter	Group	Mean ±SD	P value (Sig: 2 tailed)
Ascitic fluid cell count (<250 cells/mm3)	Group A: N=27	549.8519±64.67823	0.007**
	Group B: N=60	58.35596 ± 4.10088	
Follow-up ascitic fluid cell count (<250 cells/mm3)	Group A: N=27	95.73919 ± 8.77809	0.004*
	Group B: N=60	58.35596 ± 4.10088	
Ascitic fluid protein (Transudate <3 g/dl Exudate >3 g/dl)	Group A: N=27	1.540±0.81	0.213
	Group B: N=60	13.858±1.01	
Ascitic fluid albumin (g/dl)	Group A: N=27	1.54±0.48	.127
	Group B N=60	0.7800±0.2864	
Ascitic fluid LDH (IU/L)	Group A: N=27	179.5304±41.65863	.0490*
	Group B N=60	79.8610 ±5.29765	
Ascitic fluid glucose (mg/dl)	Group A: N=27	118.4870±16.08609	.013*
	Group B N=60	144.6883±20.58725	

*Statistically significant difference

A statistical significant difference between the two groups was observed in both ascitic fluid LDH and glucose.

As shown in the table, ascitic cell count and ascitic fluid LDH were significantly higher in group A than in group B, while ascitic fluid glucose was significantly lower in group A than in group B.

Figure 1

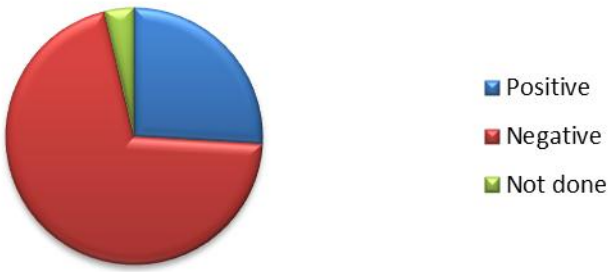

Figure (1): Pie chart showing results of ascetic culture and sensitivity among patients of group A

Figure (1) summarizes the results of the ascitic fluid culture and sensitivity. Among group A patients, seven patients (26%) showed positive ascitic fluid culture and sensitivity and nineteen patients (70%) showed negative ascitic fluid culture and sensitivity.

The most common isolated organism was E-coli which was isolated in two patients (7%), while each of the other organisms (Staph CoagNeg, Actinobacter spp., Strept. Viridans, Non-hemolytic strept and MRSA / Enterococci) was isolated in one patient (4%).

DISCUSSION

Ascitic fluid infection (AFI) is a common complication in that occurs in patients with chronic liver disease and ascites as a result of increased bacterial translocation, portosystemic shunting, gut dysbiosis, failure of liver functions and impaired immune response caused by cirrhosis [(10)].

In recent years, the rate of ascitic fluid infections caused by multidrug-resistant bacteria has become very evident. Patients with nosocomial SBP exhibited a greater resistance to antibiotics than those with community-acquired SBP [(11)].

The current study was designed to assess the frequency, clinical profile, bacteriological patterns and outcome of spontaneous bacterial peritonitis and other variants of ascitic fluid infections in patients with liver cirrhosis admitted to Tropical Medicine department at Ain Shams University hospitals. The study also investigated the bacterial isolates and antibiotic sensitivity and resistance patterns in patients with chronic liver disease and ascitic fluid infections.

The study included 87 patients with chronic liver disease and ascites, 27 patients (31%) of them had infected ascites which was either symptomatic e.g. abdominal pain and fever, or asymptomatic. seventeen patients (63%) were males and 10 (37%) were females. The mean and SD of age of the patients with infected ascites was 52.11±12.99 years old. This is consistent with ***Schwabl et al.*** [(13)] who reported that 73.8% of patients with ascitic fluid infection (AFI) were males and 26.2% were females and the mean age among the studied patients was 56.67 ± 11.28 years old.

In addition, HCV was the most common etiology of chronic liver disease among these patients as it was detected in 20 patients (74%) with infected ascites. Although Schwabl et al. [(13)] reported that the majority of patients in their study had alcoholic cirrhosis (52%) and 22% only of their patients had viral hepatitis, our results are consistent with **Badran** [(12)] who reported that the most common etiological cause among the Egyptian patients admitted at Ain Shams University hospitals with AFI was HCV infection that was detected in 83.5% of the studied patients.

The present study showed that the main presenting symptom of infected ascites was abdominal pain which was presented in 37%. This is less evident than Badran [(12)] who reported that abdominal pain was the main presenting symptom in 83.5%. In addition, the most common clinical sign in the current study was lower limb edema in 81% of patients on general examination. This is consistent with **Badran** [(12)] who reported that lower limb edema was the most common clinical sign which was presented in 84%.

As regard the other symptoms and signs of infected ascites jaundice was noted in 10 of 27 cases with AFI (37%) but jaundice was, also, noted in 25% of patients with non-infected ascites. Fever was found in 8 cases (30%) and signs of hepatic encephalopathy were noted in 7 cases (26%) only. This is consistent with **Badran** [(12)] who reported that fever and hepatic encephalopathy were noted in 31.8% and 43.5% of cases respectively.

On the other hand, fever was reported in two patients with non-infected ascites due to UTI and chest infection, and abdominal tenderness and rebound tenderness were reported in two patients with non-infected ascites due to subacute IVC thrombosis and surgical abdomen with abdominal wall cellulitis.

As regard the CBC, the total leukocytic count was significantly higher in patients with infected ascites (9.15 10^3/ul) than in patients with non-infected ascites (6.71 10^3/ul). This is consistent with ***Schwabl et al.*** [(13)] who reported that the total leukocytic count was 7.88 among the patients with infected ascites and 7.13 among the patients with non-infected ascites.

Our study revealed that total bilirubin and direct bilirubin were significantly higher in patients with AFI (T.bil 4.7±5.84, D.bil 3.23±3.94) than in patients with non-infected ascites (T.bil2.73±3.2, D.bil 1.39±1.97) and serum albumin was significantly lower in patients with AFI (2.19±0.43) than in patients with non-infected ascites (2.45±0.54). These results are similar to ***Schwabl et al.*** [(13)] who found that total bilirubin and serum albumin were about 3.75 and 2.73 ± 6.06 respectively among the patients with AFI but, in contrast to our results, this total bilirubin and serum albumin levels were not significantly different from the total bilirubin and serum albumin levels in patients with non-infected ascites which were about 3.24 and 2.7 ± 5.46 respectively. Moreover, our results are consistent with ***Badawy et al.*** [(15)] who conducted a study on one hundred

Egyptian patients with AFI and found that their total bilirubin, direct bilirubin and serum albumin were about 5.22, 3.26 and 2.17 respectively.

In addition, Serum magnesium, INR and CRP were significantly higher in patients with AFI (2.06±0.44, 1.88±1.11 and 59.02±2.75 respectively) than in patients with non-infected ascites (1.83±0.34, 1.51±0.32 and24.19±39.31 respectively). Schwabl et al. [13] showed that the CRP was significantly higher in patients with AFI (5.84) than in patients with non-infected ascites (2.72). Moreover, the INR was reported to be 1.41 by Schwabl et al. [13] and 1.87 by ***Badawy et al.*** [15].

All cases of CLD were diagnosed according to clinical, biochemical, and/or imaging findings. The severity of liver disease was categorized by Child-Pugh's classification: 11 patients (41%) were in class B and 16 patients (59%) were in class C. The majority of patients had MELD score between 10 and 19 (59%). These results are consistent with **Badran** [12] who reported that 55% of patients were Child-Pugh stage C and 55.3% had MELD score between 10 and 19 and Schwabl et al. [13] who reported that 60.7% of their patients were in Child–Pugh stage C and their MELD score was 21.2 ± 9.29.

The high frequency of Child score class C among the patients with infected ascites which was 59% in the current study, 55% in **Badran** [12] and 60.7% in Schwabl et al. [13] can be referred to deteriorated liver functions, coagulopathy, elevated bilirubin level and hypoalbuminemia which are common risk factors for AFI in patients with chronic liver disease and ascites[1].

As regards abdominal ultrasonography, liver was coarse in all patients, 20 patient (74%) had hepatomegaly in comparison to 43% of patients with non-infected ascites, 12 patients (44%) with AFI had HFL in comparison to 27% of patients with non-infected ascites, 6 patients (22%) with AFI had portal vein thrombosis in comparison to 12% of patients with non-infected ascites, three patients (11%) with AFI had occluded hepatic veins in comparison to 23% of patients with non-infected ascites, one patient (4%) with AFI had inferior vena caval thrombosis in comparison to 10% of patients with non-infected ascites, 17 patient (63%) with AFI had splenomegaly in comparison to 57% of patients with non-infected ascites.

The ascitic cell count was significantly higher in patients with AFI (550±865) than in patients with non-infected ascites (58±4).***Mostafa et al.*** [16] reported that the mean polymorphonuclear count was 211 in patients with AFI and Badran [12] found that the mean ascitic cell count was193±185.

Also, ascitic fluid LDH was significantly higher in patients with AFI (180±442 IU/L) than in patients with non-infected ascites (80±55 IU/L). While ascitic fluid glucose was significantly lower in patients with AFI (118±46 mg/dl) than in patients with non-infected ascites (145±41mg/dl). Whereas the mean ascitic fluid protein and ascitic fluid albumin in patients with AFI were 1.54±0.81 g/dl and 1.54±0.96g/dl, respectively. Badran [21] found that the mean LDH level was 153±80 IU/L, total protein was 1.37±0.52 g/dl, albumin was 0.63±0.41 g/dl and glucose was 115.5±30.94 mg/dl and ***Mostafa et al.*** [16] found that the mean AF protein was 1.3 ± 0.5 g/dl and albumin was 0.5 ± 0.2 g/dl in patients with AFI.

The ascitic culture and sensitivity taken from the studied patients showed that 20 patients had culture negative neutrocytic ascites (74% of patients with AFI), 6 patients had monomicrobialbacterascites (22% of patients with AFI) and one patients had polymicrobialbacterascites (4% of patients with AFI). This high frequency of negative cultures can be referred to many factors such as slow growth of the causative organism, low number of organisms, the use of antiseptic dressing before collecting the specimen and the delay in the transport of the specimen. However, these results are consistent with ***Enomoto et al.*** [17] who reported that more than 50% of patients with infected ascites will have negative ascitic culture and sensitivity despite of the high polymorphonuclear count (>250 cells/mm3). These results are also consistent with ***Mostafa et al.*** [16] who reported that more than 50% of the patients had negative ascitic culture and sensitivity and 35% of the patients had bacterascites.

The most frequently isolated micro-organism was E.coli that was detected in two patients (7%) followed by Staph CoagNeg, Actinobacter spp., Strept. Viridans, Non-hemolytic strept and MRSA / Enterococci where each of them was isolated in one patient (4%). This is consistent with **Badran** [12], ***Koulaouzidis et al.*** [14], ***Shi et al.*** [18] where all of them reported that E-coli was the most common organism isolated in patients with AFI.

Our study on the 27 patients with AFI showed significant improvement in ascitic fluid cell count 48 hours after starting antibiotic treatment where 12 patients responded to the third generation cephalosporins and 9 patients responded to Meropenem (4 patients of them were resistant to third generation cephalosporins, 2 patients of them received Meropenem based on results of ascitic culture and sensitivity and 3 patients received Meropenem empirically because they had nosocomial AFI), 3 patients responded to culture-based Linezolide and one patient responded to culture-based Ciprofloxacin, one patient was resistant to third generation cephalosporin and responded to Piperacillin/Tazobactam and one patient was asymptomatic Non-neutrociticbacterascites who did not receive antibiotic treatment.

CONCLUSION

Infection of the ascitic fluid is frequent among patients with chronic liver disease and cirrhosis admitted to Ain Shams University Hospitals. Almost one third of the ascitic patients developed at least one attack of spontaneous bacterial peritonitis or bacterascites. Abdominal pain is the dominant presentation; however, some patients have mild symptoms that maybe overlooked. Bacteriologic examination of the ascitic fluid is the hallmark for diagnosis of ascetic fluid infection in addition to identification of the type of bacteria. Monomicrobialbacterascites is more frequent than polymicrobialbacterascites and E coli is the most common isolated organism. Third-generation, broad-spectrum cephalosporins remain a good initial therapy for patients who do not have allergy to cephalosporins. Alternative antibiotics such as Meropenem and pipercillin-tazobactam should be considered for patients with nosocomial SBP or for patients who fail to improve on traditional antibiotic regimens.

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