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Original article

Combined spontaneous bacterial empyema and peritonitis in cirrhotic patients with ascites and hepatic hydrothorax

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ABSTRACT

Background and study aims: Spontaneous bacterial empyema (SBEM) is an underestimated condition in patients with ascites and hepatic hydrothorax with a high mortality. This study aimed to find whether spontaneous bacterial peritonitis (SBP) is a prerequisite for SBEM. *Patients and methods:* 3000 HCV-related cirrhotic patients with ascites and hydrothorax were screened for the presence of SBP (ascitic fluid neutrophils >250/mm^3) and SBEM (positive pleural fluid culture and neutrophils >250/mm^3 or negative pleural fluid culture and neutrophils >500/mm^3 with no evidence of pneumonia/parapneumonic effusion on chest radiograph or CT). *Results:* The prevalence of SBEM in cirrhotic patients was 1.2% (36/3000) unlike SBP (1.6%; 48/3000). SBEM was detected in 51.4% of the patients with hepatic hydrothorax (36/70). A total of 70 patients had concomitant ascites and hydrothorax, namely SBP (n = 17), SBEM (n = 5), and dual SBP and SBEM (n = 31), whereas 17 patients had sterile concomitant ascites and hydrothorax. Age, sex, liver function, kidney function tests, complete blood count, INR, MELD, MELD-Na, blood chemistry, and culture/sensitivity for ascitic and pleural fluid were statistically not different (p > 0.05) between SBP and dual SBP and SBEM patients. *Escherichia coli* and *Klebsiella pneumoniae* were detected in the culture. From univariate analysis, no predictors of dual SBP and SBEM were detected. *Conclusion:* SBEM is a part of SBP in cirrhotic patients with ascites and hydrothorax.

Introduction

Liver cirrhosis is usually associated with increased mortality and morbidity as it may be complicated by ascites, portal hypertension (PH) and fatal hepatocellular carcinoma [1]. There are various pulmonary diseases which develop as a consequence of cirrhosis and PH, namely hepatopulmonary syndrome, portopulmonary hypertension, and hepatic hydrothorax [2].

Hepatic hydrothorax is a transudative pleural effusion in patients with cirrhosis/PH without evidence of cardiopulmonary aetiology. It is usually seen in patients with ascites with an incidence of 6–15%. In 79.5% of the cases, it is usually right sided, however, may be left sided in 17.5% and as bilateral effusion in 3% of the cases. It may be asymptomatic or associated with dyspnoea and respiratory distress [3].

Spontaneous bacterial empyema (SBEM) is simply an infection of pre-existing hepatic hydrothorax. It usually occurs in association with spontaneous bacterial peritonitis (SBP), although it may also occur independent of it [4]. It is reported to be associated with 20% mortality [5]. In fact, the name empyema is a misnomer because it is completely different from pneumonia-related empyema in its aetiology and physical and chemical criteria of the effusion fluid. Moreover, indwelling chest tube should not be used in patients with SBEM [6].

This study aimed to find if SBP is a prerequisite for SBEM.

Patients and methods

After institutional review board approval, this study was conducted in National Liver Institute hospitals, Menoufia University, Egypt. Informed consent was obtained from all enrolled patients.

A total of 3000 patients with HCV-related liver cirrhosis who were admitted to the Hepatology department, National Liver Institute hospitals, Menoufia University, Egypt, were enrolled. Diagnosis of cirrhosis was based on clinical, laboratory, and ultrasonographic findings with or without liver biopsy [7]. The main targets were patients with concomitant ascites and hydrothorax.

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On admission, all the patients underwent thorough history taking, clinical examination, liver function tests, renal function tests, CBC, INR, abdominal ultrasonography, and chest X-ray (posteroanterior view).

In patients diagnosed with ascites, ascitic fluid sample was obtained on admission [8,9] and was guided by ultrasonography in cases with minimal to mild ascites. The sample was analyzed for leucocytic count with polymorphonuclear (PMN) cell count, blood chemistry (total proteins, albumin, LDH, and glucose), and bedside blood cultures by inoculating 10 mL of ascitic fluid into a blood culture bottle [5].

In patients diagnosed with hydrothorax, pleural effusion fluid sample was obtained on admission [10] and was ultrasonography guided in cases with minimal to mild effusion [11]. The sample was analyzed for leucocytic count with PMN count, blood chemistry (total proteins, albumin, LDH, and glucose), and bedside blood cultures by inoculating 10 mL of ascitic fluid into a blood culture bottle [5].

SBP was defined as PMN > 250/mm^3 in ascitic fluid [12]. SBEM was diagnosed by the following criteria [13,14]: positive pleural fluid culture and PMN count >250 cells/mm^3 or negative pleural fluid culture and PMN count >500 cells/mm^3. No evidence of pneumonia/parapneumonic effusion was observed on chest radiograph or CT.

Once SBP or SBEM was diagnosed, IV third generation cephalosporin (cefotaxime 2 g/12 h) was initiated. A follow-up ascitic and pleural fluid samples were obtained 48 h after therapy initiation to guide and tailor therapy. Finally, another sample was obtained on day 7 of therapy [9].

Statistical analysis

Data were statistically analyzed using IBM® SPSS® Statistics® version 21 for Windows. Data were expressed as mean ± standard deviation. All p-values are two-tailed, with values < 0.05 considered statistically significant. Comparisons between two groups were performed using the Student's *t*-test. Chi-squared test (χ^2) and Fisher's exact test were used for categorical data analysis. Univariate logistic regression analysis was used to detect the predictors of dual SBP and SBEM.

Results

In total, 3000 HCV-related liver cirrhosis patients were screened for concomitant ascites and hydrothorax. Only 70 patients had concomitant ascites and hydrothorax: SBP (n = 17; 24.3%), SBEM (n = 5; 7.1%), and dual SBP and SBEM (n = 31; 44.3%), whereas 17 (24.3%) patients had sterile concomitant ascites and hydrothorax. The prevalence of SBEM in cirrhotic patients was 1.2% (36/3000) unlike SBP (1.6%; 48/3000). SBEM was detected in 51.4% (36/70) of the patients with hepatic hydrothorax as shown in Fig. 1.

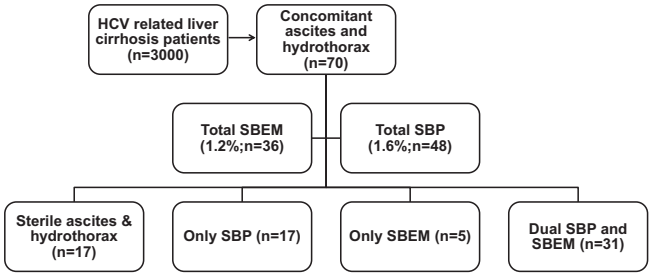

Fig. 1. Flow chart of the study.

Table 1
Comparison of baseline characteristics of patients with SBP and patients with dual SBP and SBEM.

	SBP N = 17 M ± SD	Dual SBP and SBEM N = 31 M ± SD	P
Age (years)	56.00 ± 7.34	56.03 ± 9.83	0.991
Female (N/%)	5 (31.3%)	11 (68.8%)	0.757
Diabetes Mellitus (N/%)	9 (37.50%)	15 (62.50%)	0.500
MAP (mmHg)	105.29 ± 12.56	103.29 ± 9.59	0.539
Bilirubin (mg/dL)	5.64 ± 4.70	7.74 ± 8.63	0.358
Albumin (g/dL)	2.24 ± 0.25	2.16 ± 0.40	0.460
AST (U/L)	168.82 ± 208.86	97.81 ± 162.41	0.197
ALT (U/L)	91.44 ± 123.46	50.94 ± 95.65	0.212
Urea (mg/dL)	74.53 ± 46.11	103.94 ± 85.63	0.196
Creatinine (mg/dL)	1.42 ± 1.10	1.75 ± 1.50	0.433
Na (mmol/L)	123.47 ± 5.84	122.45 ± 6.72	0.602
K (mmol/L)	4.50 ± 0.91	4.42 ± 0.85	0.751
INR	1.52 ± 0.27	1.66 ± 0.51	0.302
Hemoglobin (g/L)	9.86 ± 1.26	10.43 ± 1.51	0.193
WBCs (× 10^3/μL)	8.15 ± 5.62	10.55 ± 7.98	0.278
Platelets (× 10^3/μL)	104.88 ± 67.43	101.41 ± 83.31	0.884
MELD	19.65 ± 4.64	21.35 ± 7.38	0.393
MELD-Na	26.41 ± 3.41	27.48 ± 5.30	0.456

MAP: mean arterial blood pressure.

As shown in Table 1, both groups were matched for age (56 ± 7.34 vs. 56.03 ± 9.83 years; p = 0.991), sex [females 5 (31.3%) vs. 11 (68.8%); p = 0.757], and diabetes mellitus [9 (37.5%) vs. 15 (62.5%); p = 0.5].

There was no statistically significant difference (p > 0.05) between patients with SBP and those with dual SBP and SBEM for mean arterial pressure, serum bilirubin, serum albumin, AST, ALT, blood urea, serum creatinine, sodium, potassium, INR, haemoglobin, WBCs, and platelets.

Both MELD and MELD-Na score were comparable in patients with SBP and those with dual SBP and SBEM: 19.65 ± 4.64 vs. 21.35 ± 7.38 (p = 0.393) and 26.41 ± 3.41 vs. 27.48 ± 5.30 (p = 0.456), respectively.

As shown in Table 2, the chemical analyses of the ascitic and pleural fluid were comparable (p > 0.05) between patients with SBP and those with dual SBP and SBEM for ascitic total protein, ascitic albumin, ascitic LDH, ascitic glucose, pleural total protein, pleural albumin, pleural LDH, and pleural glucose. Pleural LDH was elevated in dual SBP and SBEM compared to SBP only (214.81 ± 227.63 vs. 165.24 ± 78.40), and ascitic LDH was elevated in dual SBP and SBEM compared to SBP only (239.61 ± 474.32 vs. 199.76 ± 168.96 IU/L); however, the difference did not reach statistical significance (p > 0.05) for both.

There was no statistically significant difference between patients with SBP and those with dual SBP and SBEM concerning

Table 2
Comparison of chemical characteristics of ascitic and pleural fluids in patients with SBP and patients with dual SBP and SBEM.

	SBP N = 17 M ± SD	Dual SBP and SBEM N = 31 M ± SD	P
Ascitic total protein (g/dL)	1.49 ± 0.74	1.42 ± 0.72	0.746
Ascitic albumin (g/dL)	0.55 ± 0.31	0.61 ± 0.35	0.543
Ascitic LDH (IU/L)	199.76 ± 168.96	239.61 ± 474.32	0.740
Ascitic glucose (mg/dL)	121.41 ± 58.40	136.58 ± 55.13	0.377
Pleural total protein (g/dL)	1.37 ± 0.59	1.63 ± 0.81	0.259
Pleural albumin (g/dL)	0.55 ± 0.35	0.71 ± 0.39	0.184
Pleural LDH (IU/L)	165.24 ± 78.40	214.81 ± 227.63	0.391
Pleural glucose (mg/dL)	165.29 ± 71.73	159.85 ± 68.64	0.797

LDH: lactate dehydrogenase

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