Hepatic hydrothorax is defined as a significant pleural effusion, usually greater than 500 ml, in a cirrhotic patient without any underlying pulmonary or cardiac diseases. It appears to be a relatively uncommon complication of portal hypertension with an estimated prevalence of 5-12% in patients with the cirrhosis of the liver. Hepatic hydrothorax is usually right-sided (65-87% of reported cases) but may be left-sided or bilateral.[1]

Spontaneous bacterial empyema, defined as the spontaneous infection of the pleural fluid, represents a distinct complication of hepatic hydrothorax. This term may be confusing because in most cases there is no evidence of pus or abscess in the thoracic cavity and indeed, the pathogenesis, clinical course and treatment strategy of spontaneous bacterial empyema (SBEM) are different from those of empyema secondary to pneumonia. Nearly 40% of episodes of spontaneous empyema are not associated with spontaneous bacterial peritonitis (SBP) or even ascites. The condition portends a poor prognosis, and is frequently under-diagnosed. This article reviews the pathogenesis, diagnosis and management of spontaneous bacterial empyema.

**Key Words:** Cirrhosis, hepatic hydrothorax, spontaneous empyema

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Table 1: Risk factors for development of spontaneous bacterial empyema

| Risk Factor                          |
|-------------------------------------|
| High Child-Pugh score               |
| Low serum albumin                   |
| Low pleural fluid protein           |
| Low pleural fluid C3                |
| Spontaneous bacterial peritonitis   |

Fluid total protein concentration in clinical practice to detect those patients at a risk for developing SBEM. [5]

DIAGNOSIS OF SBEM

The infection of the pleural fluid is frequently associated with few localizing signs. Therefore, a high index of suspicion is essential for the diagnosis of SBEM. Diagnostic criteria are demonstrated in Table 2. Any patient with hydrothorax who develops fever, pleuritic pain, encephalopathy or unexplained deterioration in renal function should undergo diagnostic thoracocentesis. [6] SBEM is defined as pleural fluid with a polymorphonuclear (PMN) cell count > 500 cells/mm³ or positive culture with PMN cell count > 250 cells/mm³ with the exclusion of a parapneumonic effusion. In fact, SBEM is rarely diagnosed, because thoracocentesis is not routinely performed in cirrhotic patients with hepatic hydrothorax. Furthermore, since SBEM is probably an infection that involves a low concentration of bacteria as is SBP, conventional cultures are not sufficiently sensitive to diagnose the condition. Pleural fluid culture should be performed by inoculating 10 ml pleural fluid into a TSB blood culture bottle at bedside since it contains an opsonin inhibitor that protects bacteria from further complement- or phagocyte-mediated killing. [2]

Routine pleural fluid analysis showed limited diagnostic efficacy in the diagnosis of SBEM since lactate dehydrogenase, total protein and glucose were not reported to differ significantly between the patients with SBEM and those with noninfected effusion and it did not correlate with PMN cell count. Hence, the diagnosis of SBE should not be overlooked when these parameters are found within the expected levels found in the transudate.

Castellote et al. showed that the analysis of pleural fluid with a reagent strip for leukocyte esterase might represent a rapid, easy-to-use and inexpensive tool for the diagnosis of SBEM in cirrhotic patients. However, more studies are required to confirm these results. [7]

MANAGEMENT

In patients who develop SBEM, therapy with an intravenous third generation cephalosporin antibiotic such as ceftriaxone (1 g every 24 h for 7–10 days) should be commenced immediately after the diagnosis is made. Given that the patients who develop SBEM have approximately a 20% mortality during therapy and that a beneficial effect on mortality has been demonstrated with albumin infusion in the setting of SBP, some authors also use albumin therapy at 1.5 g/kg on day 1 and 1.0 g/kg on day 3 in the setting of SBEM, although albumin infusion has not been specifically studied in the setting of hepatic hydrothorax and SBEM. In cases where there is slow clinical recovery, a repeat thoracocentesis is recommended to document that the patient is responding to treatment. Because its insertion in cirrhotic patients can be harmful, a chest tube should not be used in the treatment of SBEM. [8]

The main goal of treatment is relief of symptoms and control of infection until liver transplantation can be performed. Recently, Xiol et al. studied the outcome of liver transplantation in patients with hepatic hydrothorax and showed that long-term evolution was similar between patients with refractory hepatic hydrothorax or spontaneous bacterial empyema and those with uncomplicated hepatic hydrothorax. Therefore, liver transplantation might be an excellent therapeutic option for patients with hepatic hydrothorax even when complicated by empyema. [9]

In conclusion, SBEM is a frequent but underdiagnosed complication of hepatic hydrothorax and portends a poor prognosis. More studies are required to elucidate the underlying pathogenetic mechanism and the natural course of SBEM. Meanwhile, its possible occurrence should be borne in mind in cases of hepatic hydrothorax who develop fever, encephalopathy or unexplained deterioration of renal functions, particularly if they have high Child-Pugh score with or even without SBP. A diagnostic thoracentesis with subsequent culture of pleural fluid should be performed in these patients.

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