Japanese Journal of Gastroenterology and Hepatology

Review Article

Metamorphosis (Transfiguration) of Spontaneous Bacterial Peritonitis

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Received: 19 June 2020

Accepted: 29 June 2020

Published: 01 July 2020

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1. Review Article

Nowadays between 30% and 50% of patients with liver cirrhosis die due to bacterial infections [1, 2]. Spontaneous Bacterial Peritonitis (SBP) is a big complication of the severe complication "ascites" in liver cirrhosis [3-5]. SBP is the main cause of death in patients with cirrhosis [2, 6].

SBP does not arisefrom intraabdominal source which requiressurgery [6, 7], but viabacterial translocation of intestinal (monomicrobial) flora in themesentericlymphnodes [8]. SBP is observed in 10-15% of all patients with cirrhosis and ascites, but its incidence increases to29-33% in patients with double increased serum bilirubin and/or creatinine (>88,4μmol/L) [9]. Renal dysfunction is important predict of mortality in patients with SBP [10].

The life duration of patients with liver cirrhosis and ascites is now significantly extended [11]. The prolonged survival of patients with cirrhosis and as cites leads to increased incidenc eof SBP. In 1/4 of the cases SBP occurs in the third year after manifestation of ascites (7-30% per year) [12].

Global trend in the etiology of SBP is the displacement from gram-negative to gram-positive microorganisms [13] (and in naïve patients) [14, 15]. At present, half of the episodes of SBP are caused by gram-positive bacteria [14]. Multidrug-resistant (MDR), extensively drug-resistant and pan drug resistant bacteria are an independent predictive factor of mortality in patients with SBP [16, 17]. The prevalence of antibiotic-resistant bacterial infections among patients with cirrhosis at a US Liver Center is high [18]. The quinolone prophylaxis leads to increasing the proportion of gram-positive microorganisms in the etiology of SBP and the resistance to the empirical treatment [15].

SBP israrelyobserved in outpatients, but 26-50% oftheepisodesof SBP areduring hospitalization. Nosocomial infections in SBP are not rare (38.8%) and half (52%) of them are caused by gram-positive bacteria [15, 19] (difficult for treatment).

Nowadays bleeding from oesophageal varices is not the leading predisposing factor for SBP, due to the early antibacterial prophylaxis [20].

Although SBP has been observed in 70-85% of patients with Child-Pugh class C liver cirrhosis (6), today SBP can be observed in 51-68.5% of the patients with cirrhosis in class B [21, 22]. Peritonitis in Child-Pugh class A cirrhosis is probably secondary [23]. MELD score is not related to SBP [24].

The classical (oligo ssymptomatic) clinical form of SBP and the culture-negative neutrophili cascites (CNNA) are the most common forms of contemporary ongoing SBP. Fatigue, low grade fever, impaired liver function, encephalopathy [10-50% (1, 25)] and renal dysfunction [in 1/3 of the patients with SBP [26] may be the only symptoms of SBP. The minimal and covered hepatic encephalopathy are rarely diagnosed [27]. The latent (10%) and oligo symptomatic forms of SBP have become more frequent, and fulminant forms are rare - 5% [23, 28]. In over 10L of ascites peritoneal irritation is absent not only in SBP, but in perforative peritonitis as well [29].

Peripheral blood leucocyte count ≥11x10^9/l and/or MELD ≥22 define poor prognosis of SBP [30]. Elevated CRP is however better diagnostic marker for SBP than leukocytosis [31].

In patients with Ascitic Fluid Total Protein (AFTP) <10g/L the risk of SBP increases tenfold (6). After diuretic treatment AFTP increases [32] and SBP may occur at higher values of AFTP (usually <15g/L) (33, 34). Leukocytes also increase in ascitic fluid after diuretic treatment, but not the polymorpho nuclears (PMNs) [32].

According to IAC (International Ascites Club) the SBP diagnosis is considered to be placed in PMNs>250/mm^3 [35], including the cases of CNNA (variant of SBP), as there is no significant difference in mortality between SBP and CNNA [36]. Delayed paracentesis is associated with increased in-hospital mortality in patients with SBP [37]. PMNs in ascitic fluid increase >250/mm^3 in gram-negative flora, but it is not clear whether this is the same in gram-positive microorganisms [38]. On the 48th hour from the beginning of a successful empirical treatment PMNs decrease with 25% [33]. The failure of empiric treatment of SBP may be due to antimicrobial resistance or secondary bacterial peritonitis [33]. Persistent SBP is a common complication in patients with SBP and high score in the model for end-stage liver disease [39]. Follow-up create nine level is an important predictive factor of in-hospital mortality in cirrhotic patients with SBP [40].

The ascitic bacterialculturesare rarelypositive (≈40%) [33]. Due to the low concentration of micro organisms in ascitic fluid (1 bacteria/1 ml) [23], even if collected in blood culture broth (≈70%) [33]. Peritoneal fluid cultures rarely alter management in patients with ascites [41].

In negative bacterial cultures and PMNs in ascitic fluid >250/mm^3, the isolated bacteria from blood cultures should be considered a causative agent of SBP [42].

Bacter Ascites (BA) (positive bacterialculturesfrom ascitic fluid without PMNs>250/mm^3) israrelyobserved- 5%. BA does not needtreatment, but monitor in, if there are no clinical symptoms [33]. At a more advanced stage of cirrhosis BA might progress to SBP.

First-line treatment with cephalosporins in SBP provides poor antibiotic coverage [43]. Third generation cephalosporin's are recommended as first-line treatment for community-acquired SBP in countries with low rates of bacterial resistance [34]. Treatment of nosocomial SBP: carbapenem alone or with daptomycin, vancomycin or linesolide if high prevalence of MDR gram /+/ bacteria or sepsis [34].

Albumin should be used in all patients with SBP [33, 34] [? [9, 44, 45]]. Renal dysfunction is the most important predict of mortality in cirrhotic patients with SBP [10].

Primary prophylaxis with norfloxacin in patients with Child-Pugh score ≥9 and serum bilirubin level ≥3mg/dL, with either impaired renal function or hyponatraemia, and AFTP <15g/L is recommended [34].

2. **Keywords:** Spontaneous bacterial peritonitis; Culture-negative neutrophilic ascites; Child-Pugh classification, Forms of spontaneous bacterial peritonitis

3. Conclusion

In the coming years, serious problems with the diagnosis and treatment of SBP are expected.

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