

EDITORIAL

SPONTANEOUS BACTERIAL PERITONITIS

Spontaneous bacterial peritonitis (SBP) frequently occurs in patients with liver cirrhosis and ascites. It is defined as an infection of previously sterile ascitic fluid without any demonstrable intrabdominal source of infection. It is now internationally agreed that a polymorphonuclear (PMN) cell count in the ascitic fluid of over 250 cells/mm³ is diagnostic of this condition(1).

Colonisation of the ascitic fluid from an episode of bacteraemia is nowadays the most accepted hypothesis in pathogenesis of SBP. Several factors have been postulated as leading to bacterial migration from the alimentary tract via the blood stream into the ascitic fluid. The skin, urinary tract and upper respiratory tract can also be sources of primary infection. Once bacteria gain access to the blood stream (bacteraemia), they can then easily pass on to the ascites because of the common fluid exchange between these two compartments(2). Cirrhotic also tend to have bacterial overgrowth in the gut leading to an increase in aerobic gram-negative bacilli. These can easily gain access to ascitic fluid due to altered gut permeability resulting from portal hypertension or compromised circulation which causes decreased mucosal blood flow.

Patients with cirrhosis also have diminished reticuloendothelial system capacity to prevent microorganisms crossing over from the bowel lumen to the systemic circulation via the portal vein. There is also increased bacterial translocation via the mesenteric nodes into the blood stream. Once microorganisms have colonised the ascites, the development of peritonitis depends on the defensive capacity of the ascitic fluid which is greatly compromised in most cirrhotics.

Clinically SBP may be asymptomatic. Where symptomatic, abdominal pain and fever are the most characteristic symptoms. Generalised tenderness occasionally with rebound may be elicited. Vomiting, ileus and diarrhoea due to altered gastric motility, hepatic encephalopathy, GIT bleeding, renal impairment, septic shock and hypothermia may be present in a high number of patients but are rather non specific.

The diagnosis depends on a high index of suspicion and careful clinical evaluation together with a few confirmatory laboratory tests. Ascitic fluid analysis is of great importance, and a PMN count of greater than 250 cells per mm³ is diagnostic. Where the fluid is haemorrhagic allowance should be made using 1 PMN cell to 250 red blood cells; anything higher than this being significant.

New techniques that have led to more rapid diagnosis have been described. Castellote with others(3) described use of urine "dipsticks" to detect neutrophils in ascitic fluid, thereby reducing the time from paracentesis to presumptive diagnosis of SBP to seconds. Sensitivity was 96% with specificity of 89%.

Secondary bacterial peritonitis is suspected when ascitic lactic dehydrogenase levels are higher than serum levels, protein greater than 10g% and glucose levels are less than 50mg%(4).

Gram stain of centrifuged sediment normally yields gram negative bacteria although bacterial concentration is normally low with one organism per ml or less. Culture of ascitic fluid (aerobic and anaerobic) is positive between 50-70% of patients with SBP and blood cultures are positive in an equally significant number of patients.

Most common organisms encountered both locally and internationally are gram negative with *E. Coli* and *Klebsiella*. Gram positive organisms also occur and these include *Streptococci* and *Staphylococcal* species. In up to 30% of cases the culture might be negative for various reasons including prior use of antibiotics.

The prognosis of SBP has improved in recent years with the advent of effective antibiotics and quick intervention. Mortality remains high ; in some cases up to 30-50%. Known severe complications that often lead to fatal outcomes include renal impairment, gastrointestinal bleeding and hepatic encephalopathy.

Antibiotic therapy is the mainstay of treatment and this may be empirical while awaiting the results of culture. Third generation cephalosporins such as cefotaxime are the gold standard. In poor resource settings amoxicillin with clavulanic acid has been shown to be highly effective but quinolones and aminoglycosides such as gentamicin(5) are also equally effective. Oral antibiotic administration is effective and where possible, should be encouraged. Treatment with albumin infusions in addition to an antibiotic reduces the incidence of renal impairment and improves survival.

There is also room for antibiotic prophylaxis in those cirrhotic patients likely to develop SBP, for example, those with gastrointestinal haemorrhage, low ascitic total protein, or patients with established liver failure. However, evidence on the cost-effectiveness and efficacy of long term prophylaxis in patients with cirrhosis and ascites is still controversial and requires more study.

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