Spontaneous Bacterial Peritonitis and Renal Allograft Recipients

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Dear Sir,

Spontaneous bacterial peritonitis (SBP) is a well-known complication which occurs in up to 8% of cirrhotics with ascites [1]. Moreover, SBP has been reported in patients without hepatic disease but in whom ascites alone was present [2, 3].

In a report of peritonitis in renal transplant recipients Hau et al. [4] describe a case of 'primary peritonitis' in a patient shortly after transplantation. We think that this case of postoperative diffuse peritonitis with no clear intraabdominal source of infection might not be defined as an episode of SBP. Similarly, in a recent report published in your journal [5], Shaked and Samra report a case of a so-called 'spontaneous peritonitis' in a renal transplant recipient 48 h after a graft nephrectomy. Undoubtedly, this episode of fulminant peritonitis is an early postsurgical one and none of us can affirm that this patient died from a well-defined SBP episode. Probably, an inadvertent postoperative source of infection related to a previous immediate surgery was the mechanism of the peritonitis. In 1971, Hadjiyannakis et al. [6] reported 8 cases of peritonitis observed in 162 kidney transplants; 2 of them were 'primary' of undetermined causes.

Obviously, these are not SBP episodes.

We think it is necessary to define what SBP is. It is not the infection of the peritoneum shortly after laparotomy. It is the infection of preexisting ascites in the absence of any intraabdominal source, including pancreatitis, perforated viscus, immediate abdominal surgery or peritoneal dialysis. In the majority of cases, infection of ascites probably occurs via hematogenous seeding in a patient with global deficits in host defense [1, 2].

Runyon and Hoefs [7] have found serial ascitic fluid polymorphonuclear leukocyte counts and serial cultures to be useful in differentiating SBP from nonperforation secondary bacterial peritonitis, which is probably the cause of death in the patient reported by Shaked and Samra [5]. In SBP, there is a sharp decline in neutrophil count not observed in secondary bacterial peritonitis; in the latter, in fact, the follow-up neutrophil count is greater than the baseline value. Unfortunately, the patient reported died within the first 48 h and there was not enough time to evaluate these data.

In summary, SBP is a well-defined entity that occurs in the absence of any intraabdominal source of infection. To date, the episodes of peritonitis reported in renal transplant recipients with an

undetermined source of infection are not probable cases of SBP [4–6]. However, renal transplants are prone to intraperitoneal infection and impaired host defenses may predispose them to SBP. Nephrologists should be aware of this possibility in renal transplant recipients with ascites of any origin, and serial cultures of ascitic fluid must be drawn in this setting. Laparotomy should never be performed in any patient with SBP and high doses of parenteral antibiotics are the goal of therapy; mortality rates are declining [8].

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