Salmonella enteritidis Infection in Renal Transplant Recipients

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Dear Sir,

In a recent article, Berk et al. [1] reported 8 cases of nontyphoid salmonella infections after renal transplantation. A few cases of salmonellosis in renal transplant recipients have been reported [2,3]. There is evidence that in 10 of 20 patients described salmonella infection occurred in close proximity to rejection episodes or to administration of high doses of immunosuppressive drugs [1–3]. It has been suggested that impaired cell-mediated immunity and inhibition of macrophage function, which are important in the elimination of intracellular bacterias, may be predisposing factors for this kind of infection. A remark to be made is the tendence of these patients to become chronic carriers or to present a relapse of the infection, and treatment with antibiotics has to be continued during several weeks or months to sterilize their infection sources or to prevent relapse [3].

We recently had the opportunity to observe 2 cases of Salmonella enteritidis septicemia. In both cases salmonella infections occurred in close relation to the administration of high doses of corticosteroids. Renal function at the time of infection was good in both patients.

Clinical data of the patients are shown in table I. Patient 1 acquired salmonella infection 3 weeks after transplantation, when the steroid dose was high. The clinical features included septicemia, lymphocele infection, bowel perforation, peritonitis, pneumonia with ca-vitation, reactive arthritis, and urinary infection. The duration of active disease was 3 months; however, the lesions resolved with vigorous and prolonged therapy. Follow-up bacteriologic controls remained negative, and renal function remained normal.

In patient 2 salmonella infection occurred shortly after the treatment of a rejection episode, 1 month after transplantation. Clinical features included gastroenteritis, septicemia, perinephric abscess of the graft, and per-

Table I. Clinical data of patients with S. enteritidis infection

PCK = Polycystic kidneys; LN = lupus nephropathy; RT = re-nal transplantation. 1 By Elisa (Enzygnost, Behring).

sistent stool and urinary carriage during months. At the same time of the salmonella infection this patient developed a primary cytomegalovirus (CMV) infection, as defined by seroconversion

with the presence of CMV-specific IgM and IgG antibodies. With appropriate therapy the patient recovered and could be discharged with a normal renal function. However, despite a prolonged treatment with ampicillin and subsequent courses with co-trimoxazole, excretion of bacteria in stool and urine persisted. Only after 11 months and an additional period of co-trimoxazole did the salmonella excretion subside. No persistent focus of infection could be found, and because her infection ultimately resolved, gastrointestinal or biliary tract carriage seems most likely. The resolution of the salmonella infection corresponded with the

Salmonella enteritidis Infection in Renal Transplant Recipients

123

delayed disappearance of specific IgM CVM antibody. These data suggest that a deficiency in the host's immune system rather than bacterial virulence factors permitted this prolonged infection and that CMV infection may have predisposed the patient to chronic gastrointestinal and urinary carriage.

There is agreement that chloramphenicol is the drug of choice for enteric fever. In renal transplant recipients Berk et al. [1] suggest that chloramphenicol is the drug of first line of treatment, remaining ampicillin and co-tri-moxazole of second choice. However, it has been suggested that a course of several weeks of chloramphenicol therapy, in addition to full-dose azathioprine, has a high likelihood of producing marrow aplasia [4]. Because of this, and because in our patients ampicillin and co-tri-moxazole were effective, we believe that in renal transplant recipients with nontyphoid salmonella infection an initial high-dose ampicillin regimen followed by a prolonged course of either ampicillin or co-trimoxazole instead of the potentially more toxic chloramphenicol regimen has excellent results [5].

High doses of immunosuppressive drugs may be a predisposing factor for this kind of severe infection. An impairment of the mononuclear phagocyte function by concomitant CMV infection [6] may be an aggravating factor which can make it difficult to eradicate the bacteria.

References

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