Campylobacter fetus bacteraemia in a renal graft recipient

Sir,

We report here the first case of bacteraemia due to Campylobacter fetus subsp. fetus in a kidney transplant recipient.

Case. A 68-year-old woman presented with 38°C fever 23 days after kidney transplantation. She received cyclosporin, sirolimus and corticoids against organ rejection. During the post-operative course, she developed mild suppuration of the scar due to methicillin-resistant Staphylococcus aureus (MRSA) susceptible to ofloxacin, gentamicin and vancomycin treated by local antiseptic.

On the day of admission, physical examination revealed a slight suppuration of the scar. Chest radiographs were unremarkable. Laboratory findings revealed a plasma creatinine level of 115 μmol/l, haemoglobin level 7.9 g/dl, lymphopenia 0.394 Giga/l, C reactive protein (CRP) 225 mg/l, hypogammaglobulinaemia 6.3 g/l, platelet count 72 Giga/l and mild hepatic cytolysis. Cyclosporin and sirolimus plasma trough levels were high: 403 and 26.4 ng/ml, respectively. The dosage of cyclosporin and sirolimus was decreased. Specimens of blood and urine were obtained for culture, and vancomycin 1 g/day and gentamicin 80 mg/day were started. Fever disappeared within 3 days. Cultures performed on three blood samples inoculated in aerobic vials (BACTEC Plus/Aerobic F, Becton Dickinson, Meylan, France) yielded a motile Gram-negative rod after 3 days of incubation in BACTEC 9240 (Becton Dickinson). Anaerobic blood cultures remained sterile. The microorganism was oxydase- and catalase-positive, and hippurate- and urease-negative. C. fetus subsp. fetus was identified by the apiCampy gallery (BioMérieux, Marcy l’Etoile, France), and a susceptibility test was performed by the disc diffusion method with Mueller–Hinton agar [1] in a microaerophilic atmosphere (GENbag microaer, BioMérieux). The bacterium was susceptible to amoxicillin, gentamicin and ofloxacin.

The treatment was changed to i.v. amoxicillin (3 g/day) associated with ofloxacin (200 mg/day) given orally. On day 10 of treatment, ofloxacin was stopped and amoxicillin was given orally (3 g/day). A transthoracic echocardiogram showed no sign of endocarditis. The CRP decreased to 15 mg/l. Cyclosporin and sirolimus plasma trough levels were 310 and 14 ng/ml, respectively. The patient was discharged.

The patient was readmitted to the hospital 7 days later with abdominal pain, vomiting and diarrhoea. She had no fever. Laboratory finding revealed haemoglobin 8.8 g/dl, WBC 4.600 Giga/l, PMN 0.655 Giga/l, platelet count 172 Giga/l, CRP 175 mg/l, hepatic cytolysis (ASAT 117 U/l, ALAT 118 U/l, γGT 757 U/l) without cholestasis, and persistent profound hypogammaglobulinaemia (4.1 g/l). The plasma creatinine level was 228 μmol/l. Despite no increase in the dosage of cyclosporin and of sirolimus, the trough levels were high: 450 ng/ml and 27.5 ng/l, respectively. Abdominal ultrasonography showed a slight accumulation of liquid (2×2 cm) behind the graft. Blood cultures drawn on the day of admission were sterile. Amoxicillin was stopped 5 days later after a total antibiotic course of 22 days.

During the next 3 days the patient’s temperature rose to 39°C. Campylobacter fetus was isolated from three aerobic blood cultures and from stools. The sensitivity was the same as found previously. Amoxicillin 6 g/day and ofloxacin 400 mg/day were rein infused. Gastric fibroscopy revealed acute gastric lesions and although Cytomegalovirus was not confirmed, gancyclovir was added. A thoracic and abdominopelvic computed tomography (CT) scan did not show any abscess. A small homogeneous accumulation behind the graft was interpreted as lymphohela. The patient was severely immunocompromised with WBC 1.8 Giga/l, PMN 1.512 Giga/l and lymphocytes 0.162 Giga/l. Despite a decrease in the dosage of cyclosporin (100 mg/day) and of sirolimus (1 mg/2 days), the plasma trough level remained high: 250 and 26.3 ng/ml, respectively. After 5 days of antibiotic treatment she developed a septic shock with fatal multiple organ failure. Since the transplantation, the total amount of corticosteroids administered was 1820 mg. Other specimens cultured from blood and urine remained sterile. Cytomegalovirus infection was detected neither in blood samples nor in urine. The post-mortem kidney biopsy showed mild tubular anomalies with no sign of rejection or infection.
Comment. Only one previous case of C. fetus infection (meningitis) has been reported in a renal transplant recipient [2]. The diagnosis and treatment of these infections are usually delayed because of the slow growth of C. fetus from culture samples [3]. Relapsing bacteremia is associated with high mortality [4,5]. Our patient relapsed and developed severe sepsis. The C. fetus was still isolated from stools. Death occurred despite treatment with amoxicillin and ofloxacin, and sensitivity of C. fetus to amoxicillin and to fluoroquinolone. The adequacy of the daily dose and duration of antibiotic treatment for a complete eradication of C. fetus from the intestinal tract is thus questionable.

Campylobacter fetus infection occurs in immunocompromised hosts [2,4,5]. It is usually serum-resistant (S-protein) and needs to be opsonized by specific antibodies for uptake killing by phagocytic cells [6]. Humoral immunity plays a major role in preventing intestinal translocation [5]. In our case, the profound suppression of both humoral and cellular immunity was related to the patient’s age, corticosteroid therapy, and the inhibition of T lymphocyte activation induced by synergism between cyclosporine and sirolimus treatment. Therefore, C. fetus should be considered a possible cause of fatal bacteraemia in renal transplant recipients. In these circumstances, reduction or interruption of immunosuppressive drugs, and prolonged high dosage antibiotic treatment should be performed.

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