A case of leptospirosis presenting with end-stage renal failure

Sir,

Leptospirosis is a zoonosis caused by spirochetes. Information concerning leptospirosis-induced end-stage renal failure (ESRF) is very scarce [1]. This report describes a young male patient presenting with leptospirosis who continued as a chronic haemodialysis patient despite treatment for leptospirosis.

A 21-year-old, previously healthy man, a farmer by profession, was brought to the Emergency Room on March 20, 2004 by relatives due to high fever, headache, nausea and vomiting, and constant sleepiness. At admission, he was lethargic and clinically dehydrated. At physical examination, conjunctival congestion and petechia in the soft palate region were observed. Pupils were symmetric and reactive. No meningeal signs were noted. Axillary temperature was 37.8°C, blood pressure was 130/90 mmHg and heart rate was 88 beats/min. Initial laboratory evaluation showed leukocyte levels of 4200/μl, haemoglobin at 9.2 g/dl, thrombocytes at 62 000/μl, serum urea at 474 mg/dl, creatinine at 17.8 mg/dl, albumin at 3.5 g/dl, uric acid at 10.2 mg/dl, serum sodium at 127 mmol/l, potassium at 8.8 mmol/l, calcium at 8.9 mg/dl, phosphorus at 6.1 mg/dl and proteinuria of 1750 mg/day. Liver function tests, and the levels of serum glucose, lactate dehydrogenase and creatine kinase were normal. Arterial blood gases were pH 7.25, HCO3^- 8.5 mmol/l, pO2 114 mmHg and pCO2 22 mmHg. Urinalysis showed microscopic haematuria, and leukocyturia. The daily urine volume was 3000–5000 ml. Blood and urine cultures were sterile. Serum antibodies for brucella, salmonella, toxoplasmosis, cytomegalovirus, Epstein–Barr virus and hantavirus were negative. At bone marrow biopsy, megakaryocytes were reduced. Haemodialysis treatment was immediately initiated. In addition, empirical antibiotic therapy was started using cefoperazone/sulbactam.

His relatives stated that he lived in the south of Turkey, which has a temperate climate, and that 20 days previously he had worked without gloves in an irrigation channel containing river water.

Leptospirosis was investigated 10 days after the patient’s admission due to high fever, haematological and renal abnormalities. No leptospira were seen in the blood under dark field microscopy. Leptospira icterohaemorrhagiae and Leptospira australis were positive by macrotube agglutination (Danke-Seien) test. Leptospira immunoglobulin M (Ig M) was positive by enzyme-linked immunosorbent assay (ELISA). Blood samples were screened for anti-leptospiiral IgM antibodies using a Leptospira

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**Fig. 1.** Interstitial mononuclear cell infiltration and mild fibrosis in the first kidney biopsy specimen [Trichrome staining (Gomori), ×10].
biflexa antigen in a commercial ELISA kit (Pan Bio®, Leptospira IgM ELISA test). Microscopic agglutination test (MAT) was also positive (titre 1:200) for L. australis serotype Bratislava. Twelve days after presentation, antibiotic therapy was switched to penicillin (12×10⁶ IU/day, intravenously).

Despite 3 weeks of haemodialysis treatment, renal function did not improve and a kidney biopsy was performed. Under light microscopy, mesangial hypercellularity, interstitial dense lymphocyte infiltration, tubular atrophy and interstitial fibrosis were determined (Figure 1). At immunofluorescent examination, C3 accumulation was observed in the glomerular capillary wall. No spirochetes were observed in the proximal tubular epithelium.

His general condition improved with regular haemodialysis and penicillin treatment. Kidney function did not improve, however. Ten weeks after beginning haemodialysis treatment, a kidney biopsy was repeated. At the second kidney biopsy, tubular atrophy and interstitial fibrosis were determined to have increased (Figure 2). In this period, MAT yielded a 1:50 titre to L. australis serotype Bratislava and leptospira IgM ELISA appeared to be negative. No icterus, respiratory insufficiency, rhabdomyolysis or multi haemorrhage developed in the patient, who was observed and treated in our clinic between March and September 2004. The patient was admitted to a regular haemodialysis programme and discharged.

Katz et al. reported that serum creatinine levels were >1.5 mg/dl in 54% of patients with leptospirosis [2]. Covic et al. determined that renal functions normalized in 64% of leptospirosis patients presenting with acute renal failure (ARF) and followed-up for 90 days, and that there was persisting mild renal insufficiency in 10% [3].

Acute tubulointerstitial nephritis/post-infectious nephritis is reported to be the most characteristic renal lesion observed in leptospirosis [4,5]. In our patient, there was a gap of ~6 weeks between likely onset of illness and the first kidney biopsy being performed, showing mild interstitial fibrosis. Moreover, in a second kidney biopsy 7 weeks later, these lesions were shown to be still progressive, and it was thought that irreversible histopathological findings of interstitial fibrosis with tubular atrophy could develop (tubular atrophy, interstitial fibrosis).

Thrombocytopenia linked to increased peripheral platelet consumption frequently occurs in severe leptospirosis. In addition to an increase in megakaryocyte series in bone marrow being expected in this case, Nicodemo et al. reported that the static microscopic examination of a bone marrow aspirate cannot accurately depict the dynamic mechanisms of platelet production when these cells are being consumed in peripheral blood [6].

As well as a leukocyte count of 4200/mm³ being remarkably low for a severe case of leptospirosis, the size of the neutrophil fraction is noteworthy. Furthermore, leptospirosis patients with a low or normal leukocyte count together with ARF have been reported [4,7].

A case of leptospirosis resulting in irreversible dialysis-dependent ESRF is a very rare condition.

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True Aneurysm rupture of omental artery leading to hemoperitoneum and shock in a CAPD patient

Sir,

Haemoperitoneum is a rare complication in continuous ambulatory peritoneal dialysis (CAPD) patients. In most cases, haemoperitoneum subsides by itself or by flushing the peritoneal cavity several times with the peritoneal dialysis solution [1,2]. Haemoperitoneum by rupture of visceral aneurysms in CAPD patients is rare [3]. We report a case of true aneurysmal rupture of the omental artery in a patient undergoing CAPD.

A 71-year-old male on CAPD was transferred to our hospital, due to continuous bloody peritoneal effluents and decrease of consciousness. After he was transfused with 200 ml of packed red blood cells at a hospital nearby, the patient was immediately transferred to our hospital because of unstable vital signs and bloody peritoneal effluents. Bloody peritoneal fluid was persistently drained through the CAPD catheter at our hospital. He was in a confused mental state. His blood pressure was 80/60 mmHg, body temperature was 36.7°C, pulse rate was 126/min and respiratory rate was 20/min. Laboratory data showed a leukocytosis (white blood cells 16 300/mm³, segmented neutrophils 70%) and his haemoglobin level was 8.2 g/dl.

Free air was not detected on the simple erect chest film. Computed tomographic angiography revealed aneurysmal dilatation of the paracolic gut accompanied by haematoma (Figure 1). We preferred an exploratory laparotomy for the purpose of making a definite diagnosis and determining treatment. An aneurysm of 16 mm in diameter was detected at the margin of the greater omentum covered with blood clots. Omentectomy and aneurysmal resection were performed. The pathology showed a dilated true vessel lumen with all layers. The patient was discharged at 18 days after the operation and was judged to be without sequelae. He is doing well and continues on peritoneal dialysis.

In this case, true aneurysmal rupture of the omental artery was the cause of a massive haemoperitoneum in our patient undergoing CAPD. Several cases of massive haemoperitoneum in CAPD patients have been reported [4–6]. However, to our knowledge, haemoperitoneum caused by rupture of the true omental artery in a CAPD patient has not been reported. Even though this case is extremely rare, we should always suspect this critical condition in a patient undergoing peritoneal dialysis if bloody peritoneal effluent is observed.

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