Acute renal failure in leishmaniasis

Sir,

Visceral leishmaniasis (VL) has become a frequent complication of AIDS [1]. When Leishmania and HIV interact, a new broad spectrum of leishmaniasis occurs. Leishmanias can spread to unexpected sites other than the reticulo-endothelial system, such as peripheral blood, normal skin, gastrointestinal tract and respiratory tract [2]. Acute renal failure (ARF) has rarely been reported [3]. A 28-year-old Nigerian woman, who had been in Italy for 8 months, attended the hospital because of epistaxis, confusion and anuria. Her temperature was 36.3°C, O₂ saturation 98%, blood pressure 120/70 mmHg and cardiac pulse 125/min. Her physical examination was unremarkable, except for abdominal tenderness. Laboratory investigations disclosed: HIV positivity, serum creatinine 20.7 mg/dl, Na 140 mEq/l, K 7.5 mEq/l, Hb 7.9 g/dl, MCV 74 fl, WBC 4050/mm³, platelets 557000/mm³, AST 424 U/l, ALT 41 U/l, bilirubin 3.6 mg/dl (3.4 mg/dl indirect), CPK 55 U/l, LDH 896 U/l, INR 2.4, PTT ratio 1.95 and fibrinogen 175 mg/dl. ANCA, HCV, HBsAg, Mycoplasma pneumoniae IgG and IgM titres, parvovirus IgM and blood cultures were negative. Parvovirus IgG titre was positive 1/256.

The patient received nasal packing and blood transfusions, and dialysis was initiated. She died 2 days later, with unremitting epistaxis. Post-mortem examination showed diffuse alveolar haemorrhage, splenomegaly, lomboaortic lymphoadenopathy and haemorrhagic necrosis of nasopharynx. Light microscopy examination evidenced striking diffusion of Leishmanias in phagocytic cells and extracellular presence in vascular lumina and connective tissues (kidney, liver, nasopharynx, lung, skin, uterus, ovary, lymph nodes, bone marrow, heart and spleen). Renal changes consisted of the following. (i) Glomerular lesions: as segmental collapse of capillary loops, congestion of capillaries filled by Leishmanias (Figure 1), capsular synechiae, focal and segmental areas of glomerular sclerosis, focal thickening of basement membranes, parasite invasion of mesangial axes (cells and matrix) and focal mesangial and epithelial cell hyperplasia with initial crescent formation. Immunohistochemical examination was negative for immunoglobulins and light chains. (ii) Tubulo-interstitial damage: as acute necrotizing tubulitis, tubular necrosis and hyaline casts, focal infiltration of CD68+ and CD3+ cells, and Leishmanias inside of histiocytes in peritubular areas. (iii) Blood vessel changes as focal leukocytoclastic vasculitis of vasa recta and free Leishmanias in the capillary lumina.

Acute glomerulonephritis (GN) [4], proliferative GN [5], collapsing focal segmental glomerulosclerosis [6], acute interstitial nephritis [5] and tubular cell necrosis and tubulitis [7] have all been described in patients with Leishmaniasis. ARF [3], nephrotic syndrome [4] and proteinuria [5] have been reported. The renal damage in our patient was mediated through different mechanisms. Tubular necrosis was secondary to ischaemia, due to small vessel obliteration by Leishmanias, and to haemolysis. Necrotizing tubulitis was due to direct invasion by the parasites and by inflammatory reaction. There was a diffuse invasion of renal structures by Leishmanias. Glomerular involvement secondary to Leishmaniasis may have been superimposed on prior HIV

Fig. 1. Renal glomerulus (40×, H&E). Leishmanias free in a capillary lumen; focal capillary congestion; segmental mesangial sclerosis; segmental mesangial expansion; flocculo-capsular synechiae.
nephropathy, as suggested by focally collapsed tufts and focal and segmental areas of glomerular sclerosis.

In conclusion, Leishmania behaves as an opportunistic infection in HIV-infected individuals. It should be suspected in African people in a case of acute renal failure, when no other causes can be advocated. ARF can be due to direct invasion of parenchyma and to tubulo-interstitial and glomerular structures involvement.

Conflict of interest statement. None declared.


DOI: 10.1093/ndt/gfg267

Icodextrin-associated peritonitis among CAPD patients

Sir,

Icodextrin-associated sterile peritonitis is well-recognized. An incidence of 10–30% amongst CAPD Extranal® users as we relied on organized check-ups rather than reporting by patients. Baxter has since had a voluntary recall of batches with known high peptidoglycan levels. This has resulted in a dramatic decline in incidence of Extranal®-associated sterile peritonitis. This makes peptidoglycan the likely cause of the icodextrin-associated sterile peritonitis observed recently. It would seem logical that only a <10 ng/ml peptidoglycan level fluid should be used. However, the concern is that presence of any peptidoglycan, even low levels <10 ng/ml, may lower the bacterial load required to trigger off peritonitis. Likewise, previously ‘sensitized’ patients may only be able to tolerate fluid containing no peptidoglycan. This, however, may not be technically feasible.

Conflict of interest statement. None declared.

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