Case Report

Acute cortical necrosis in acquired immunodeficiency syndrome (AIDS)

A. Hertig, R. Couprie, J.-Ph. Haymann, B. Mougenot, N. Farres, M.-N. Peraldi, E. Rondeau and J.-D. Sraer

1Service de Nephrologie A et Association Claude Bernard, 2Service d’Anatomo-Pathologie, 3Service de Radiologie, Hôpital Tenon, Paris, France

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Introduction

Cortical necrosis has become a very infrequent type of acute renal failure in adults [1]. We report two cases of acute cortical necrosis in patients infected with the human immunodeficiency virus.

Case report 1

A 44-year-old-man, suffering from AIDS (CD4 count was 12/mm³), and receiving no treatment, was admitted for impaired consciousness and diarrhoea. His temperature was 40°C. He complained of abdominal pain. On physical examination he was cachectic, confused, and agitated. Blood pressure was 100/60 mmHg. He was anuric. The results of the laboratory examinations showed a haemolytic anaemia: haemoglobin level was 10.2 g/dl, lactate dehydrogenase serum level was 4501 IU/l and haptoglobin level was 0.66 g/l. The results of the laboratory examinations showed a haemolytic anaemia: haemoglobin level was 10.2 g/dl, lactate dehydrogenase serum level was 4501 IU/l and haptoglobin level was 0.66 g/l. Examination of the blood smear revealed schistocytes and a reticulocyte count of 97 000/mm³. A Coombs’ test was negative. The serum urea nitrogen concentration was 25 mg/l and the serum creatinine concentration was 692 μmol/l. Hepatic cytolysis (ASAT × 5N, ALAT × 3N) was evident and blood cultures were negative. Disseminated intravascular coagulation was not evident (prothrombin time was 98%, fibrinogen level was 6 g/l). Stool cultures were negative. The combination of renal failure, thrombocytopenia, and microangiopathic haemolytic anaemia was consistent with haemolytic–uraemic syndrome. CT scan showed a bilateral lack of enhancement of the renal cortex after contrast infusion, enhancement of renal medulla, and absence of renal excretion (Figure 1). Renal arteriogram showed a bilateral lack of vascularization of the renal cortex (Figure 2). Renal biopsy showed an almost complete cortical necrosis, with enlarged acellular glomeruli containing microthrombi, necrosis of arterioles, and extensive tubular lesions. The medulla was normal. The retinae appeared normal but histological examination of ulcerated areas biopsied during

Correspondence and offprint requests to: Pr J. D. Sraer, Service de Néphrologie A, Hôpital Tenon, 4 rue de la Chine, 75020 Paris, France.

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Discussion

Both of these patients presented with similar symptoms, namely neurological disturbance, fever, and acute renal failure due to haemolytic–uraemic syndrome. In both cases, the renal biopsy confirmed thrombotic microangiopathy with extensive cortical necrosis. Acute bilateral cortical necrosis (ACN) is a rare and catastrophic type of renal failure, characterized by the destruction of all elements of the renal cortex. As compared to the older literature [2], its incidence has dramatically decreased in the last 15 years in western countries to less than 1% of all the causes of renal failure [1]. In contrast, it still accounts for 3.8 to 6.3% of acute renal failure in eastern and northern India [3,4]. Acute cortical necrosis mostly occurs in association with obstetric complications such as abruptio placentae (50%), postpartum haemorrhage, puerperal sepsis, and eclampsia. Among the non-obstetric cases, snake bite, haemolytic–uraemic syndrome, acute pancreatitis, allograft rejection, and septicaemia are the most common causes associated with the development of acute cortical necrosis [1,2].

Haemolytic–uraemic syndrome damages the kidney through a characteristic vascular injury, thrombotic microangiopathy [5]. The haemolytic–uraemic syndrome mainly affects infants and small children who had a verotoxin producing Escherichia coli infection. However, acute cortical necrosis rarely accompanies this syndrome. Recently haemolytic–uraemic syndrome in adult patients with AIDS has been described, but is a rare cause of nephropathy in AIDS patients [6–8].

Case report 2

A 32-year-old man was admitted for confusion. He was homosexual, a drug-addict, and suffered from AIDS. The CD4 count was 10/mm³. He had been treated with bleomycin for 1 year for a Kaposi sarcoma, and with ganciclovir for CMV retinitis in the last 4 months. He had suffered from fever, disorientation and memory problems in the last 10 days.

Cutaneous necrosis of the fourth right finger and necrosis of the tongue were observed. Examination of the cerebrospinal fluid was normal. MRI of the brain showed ventricular dilatation and the absence of any abnormal cerebral mass. Laboratory examination showed a haemolytic anaemia: haemoglobin level was 5 g/dl; haptoglobin level was <0.06 mg/l; platelet count was 40 000/mm³ and schistocytes were observed on the blood film. He had renal failure with a serum creatinine level of 180 mmol/l. Renal biopsy showed a severe arterial form of thrombotic microangiopathy, with large areas of cortical necrosis. By light-microscopy, intranuclear inclusion was seen in some enlarged cells in peritubular capillaries. The patient was treated with 14 plasma exchanges and ganciclovir. In the following days, renal function worsened and dialysis was needed. Haemolytic anaemia and thrombocytopenia were still observed, and the patient died within 3 weeks.

References


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