Rhabdomyolysis and acute renal failure in Plasmodium falciparum malaria

Sir,

Acute renal failure is a common complication in malaria infection. This can be the result of multiple mechanisms [1]: hypovolaemia, excessive haemolysis, disseminated intravascular coagulation or impaired microcirculation due to a high level of parasitized erythrocytes. Rhabdomyolysis is another uncommon way of inducing renal failure in malaria infection. The diagnosis is based on high serum level of muscular enzymes; Creatine Phosphokinase (CPK) and clinical symptoms like myalgias. To our knowledge, only six cases of rhabdomyolysis complicated by acute renal failure during malaria infection have been described [2,3].

Case. A 20-year-old woman was admitted in our institution with a 5 day history of fever, nausea and muscle pain. She was a Malagasy woman and had been living in France for many years. Six days before, she returned to France after a prolonged stay in Madagascar. During this stay, she did not use any chemoprophylaxis against malaria.

On the day of admission, vigilance was impaired (Glasgow coma score: 12) and central temperature was 36°C. On examination, there was a cutaneous lesion on her leg, probably induced by a mosquito bite, without any infectious symptom. Blood pressure was low (88/44 mmHg). Cardiac pulse rate was 64 beats/min. The patient was anuric. Abdominal examination revealed a palpable 1 cm hepatomegaly, but no splenomegaly. Blood samples revealed serum haemoglobin level 12.2 g/dl, total white blood cell count 14000/mm³ and platelet count 19000/mm³. Blood smear showed ring-form Plasmodium falciparum in 7% of erythrocytes. C-reactive protein level was 171 mg/l. Serum potassium concentration was 3.9 mmol/l, sodium 124 mmol/l, creatinine 724 μmol/l and blood urea nitrogen 37.8 mmol/l. Serum creatinine kinase and myoglobinaemia levels were very high (71940 UI/l and > 20 000 UI/l, respectively). Her liver function tests showed hyperbilirubinemia (41 μmol/l) and elevation of liver enzymes [TGO level: 373 U/l (normal: <35 U/l) and TGP level: 1129 U/l (normal: <30 U/l)]. Urine analysis revealed pigment on dipstick examination and the absence of red blood cells in urine. Proteinuria was low at 0.25 g/l. Because of high CPK level, viral and bacterial serologies were prescribed: Legionella, Mycoplasma, Chlamydia, Leptospiira, influenzae A and B and para-influenzae viruses. Results were all negative.

The patient was treated with intravenous chloroquine (24 mg/kg/day) and intravenous hydration. Creatinine level increased initially in spite of a high daily urinary excretion, but returned to normal values after 10 days. No haemodialysis was performed. CPK level decreased, too. Rapidly, neurological status became normal. After complete recovery, the patient was lost of sight.

Comment. Ischaemic acute tubular necrosis is by far the most common cause of acute renal failure in P. falciparum malaria. It is the result of hypovolaemia, peripheral pooling of blood and blockage of microcirculation by parasitized red cells and non-specific effects of infection. In this case, none of these mechanisms may explain the renal failure. This patient had a severe rhabdomyolysis that may be the actual reason of this acute renal failure. Many mechanisms may induce these muscle damages [4]. In this case, rhabdomyolysis could not be explained by usual causes (hyperthermia, crush syndrome, metabolic abnormality, drugs or other infectious diseases). Thereafter, the responsibility of P. falciparum as the physiopathological mechanism of the rhabdomyolysis was supposed.

Only a few publications are available about rhabdomyolysis and P. falciparum infection [2,3]. The mechanism postulated to explain rhabdomyolysis is the sequestration of parasitized erythrocytes in striated muscle capillaries, inducing microcirculatory obstruction. Plasmodium falciparum may also induce myositis with myoglobinuria [5]. This mechanism may explain the muscle pain experienced by our patient and the high level of CPK.

We suggest that rhabdomyolysis has to be researched in patients with acute renal failure and P. falciparum malaria infection, especially if muscle pain is present.

Conflict of interest statement. None declared.

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Rhabdomyolysis due to hypothyroidism

Sir,

Muscular disorders are usual in hypothyroidism. Hypothyroid myopathy is most often limited to myalgias, muscle stiffness and cramps, with sometimes moderately elevated levels of muscle enzymes [1]. On the other hand, rhabdomyolysis due to hypothyroidism is very rare and only a few cases have been reported. We describe a patient with rhabdomyolysis due to hypothyroidism.

Case. A 31-year-old man presented with generalized body swelling and increasing stiffness. He had experienced severe aches and pains, lower limb weakness, polyuria, nocturia and polydipsia. He had no precipitating factor for