Acute Glomerulonephritis without Fever: An Unusual Presentation of Malaria on Mefloquine Prophylaxis

J.C. Martinez-Ocaña
A. Serra
J. Bonet
P.F. Fernández-Crespo
A. Caralps

Department of Nephrology, Hospital Universitari Germans Trias i Pujol, Badalona, Barcelona, Spain

Dear Sir,

The delayed presentation of malaria due to Plasmodium falciparum has recently been suggested in travellers on prophylaxis with mefloquine [1]. We report on a traveller, returning from Togo, using this drug who presented with acute glomerulonephritis as the initial and delayed manifestation of malaria due to Plasmodium malariae.

A previously healthy 24-year-old white male, who was born and raised in Barcelona (Spain), was admitted on October 6, 1994, with a 2-day history of palpebral and ankle edema after returning from Togo 35 days before. He had received prophylaxis with mefloquine for 1 week before the trip and 4 weeks afterwards. No fever was noted. On admission, blood pressure was 170/120 mm Hg, but the physical examination was unremarkable. Laboratory tests showed: ESR, 9 mm/h; hemoglobin, 127 g/l (12.7 g/dl); white blood count, 6.9 × 10^9/l (6,900/mm³); platelet count, 147 × 10^9/l (147,000/mm³); blood urea, 14 mmol/l (84 mg/dl); serum creatinine, 140 µmol/l (1.6 mg/dl); total proteins, 58 g/l (5.8 g/dl); albumin, 33 g/l (3.3 g/dl); CK, 126 U/l; AST, 53 U/l; ALT, 126 U/l; alkaline phosphatase, 91 U/l; GGT, 28 U/l; and total bilirubin, 11 µmol/l (0.6 mg/dl). Urinalysis revealed 15-20 red blood cells (RBC) field. Proteinuria was 0.5 g/24h. Chest film was normal. Normal kidneys were evident on abdominal ultrasonography. Serologies for hepatitis A, B, and C, HIV, syphilis, cytomegalovirus, Toxoplasma, Brucella and Salmonella were negative. Immunology revealed: C3, 19 g/dl (normal range 52-120); C4, 8.7 mg/dl (18-49); CH50, < 10 U/ml (80-140); positive C3 nephritic factor; IgG, 1,285 mg/dl (700-1,500); IgA, 265 mg/dl (88-410); IgM, 129 mg/dl (50-240); circulating immunocomplexes, 2.3 ng/ml (normal range < 1.5), and negative antinuclear antibodies, ANCA, rheumatoid factor and antistreptolysins.

Frusemide and nifedipine were given and renal function improved. He remained asymptomatic until November 29, 54 days after the initial presentation, when malaise, fever and macroscopic hematuria appeared. The patient was readmitted. Physical examination was normal except for intermittent fever of up to 40°C and chills every 48 h. Analysis showed: blood urea, 6.8 mmol/l (41 mg/dl); serum creatinine, 138 µmol/l (1.6 mg/dl); proteinuria,
0.5g/24h; C3, 63 g/dl; C4, 30 mg/dl, and circulating immunocomplexes, 2.2 ng/ml. Macroscopic hematuria resolved in 24 h. Blood and urine cultures, search for Schistosoma in the urine and blood parasites were repeatedly negative. On December 7, 1994, a thick blood smear demonstrated P. malariae. Quinine was administered for 7 days. Over the next 48 h his fever abated, and 4 months after readmission he remains asymptomatic and normotensive without treatment, the creatinine level being 116 µmol/l (1.3 mg/dl), with normal complement and 5-10 RBC/field on urinalysis with no proteinuria.

Malaria is an important cause of glomerular disease in the tropics [2-4]. P. malariae infection has been associated with nephrotic syndrome usually leading to progressive renal failure. In the present report, acute glomerulonephritis without fever 35 days after the patient’s return from Togo constituted the initial, delayed manifestation of malaria. The appearance of fever 89 days after the patient’s return from the tropics enabled the diagnosis. This provides further evidence of the delayed and unusual presentation of malaria in patients on mefloquine prophylaxis [1].

In conclusion, malaria should be considered in the differential diagnosis of glomerular diseases appearing in travellers from the tropics, even in the absence of fever.

References