

Case Report

Pulmonary Edema in Malaria

Amar Safdar, MD;* Barry J. Hartman, MD;† Bradley A. Connor, MD;† and Henry W. Murray, MD†

Two patients, hospitalized in New York City with malaria caused by *Plasmodium falciparum*, developed pulmonary edema while responding to antimalarial therapy. These cases serve as a timely reminder of this serious pulmonary complication of malaria.

CASE REPORTS

Patient 1

A 33-year-old Australian woman, who had been stationed in Liberia for 4 months, flew to New York City for vacation. Several days later, she developed fever, rigors, lower back pain, and headache. On the third day of symptoms, she was acutely ill and was admitted to The New York Hospital where a peripheral blood smear showed characteristic ring forms of *P. falciparum* (Figure 1). She had discontinued chloroquine and proguanil prophylaxis due to gastrointestinal intolerance 10 weeks before admission. Initial laboratory studies included white blood cell (WBC) count of 4800/mm³; hemoglobin, 11.9 g/dL; hematocrit, 34%; platelet count, 43,000/mm³; lactic dehydrogenase (LDH), 399 IU; prothrombin time (PT), 14.7 seconds (control 12.0 s); and partial thromboplastin time (PTT), 35.1 seconds (control 38 s). Admission chest examination and roentgenogram were normal (Figure 2, A).

After 18 hours of treatment with oral quinine and doxycycline, parasitemia was reduced from 2.1 to 1.2%. However, 12 hours later, the patient developed acute respiratory distress with severe hypoxemia (arterial PO₂ 38 mmHg breathing room air). Bronchial breath sounds and rales were heard over both lower lung fields. Fever to 39°C persisted, although parasitemia was less than 0.1%. The patient developed evidence suggesting coagulopathy or disseminated intravascular coagulation (DIC) with a further increase in PT to 15.4 seconds and a positive D-dimer test. Repeat chest x-ray was consistent with

pulmonary edema (see Figure 2, B). In the 24 hours prior to the onset of respiratory distress, the patient had received normal saline intravenously at a rate of 150 cc per hour. Serial electrocardiograms (EKG), cardiac enzymes, transthoracic echocardiogram, and a ventilation-perfusion scan were all normal.

Although the patient had no clinical evidence of fluid overload, she received furosemide and responded with a diuresis of 2 liters of urine over 24 hours. Despite this, there was essentially no improvement in oxygenation (arterial PO₂, 63 mmHg, while receiving 100% oxygen). Two days later clinical and radiographic findings began to improve and evidence of coagulopathy began to resolve. Antimalarial therapy was stopped after 7 days, and the patient was discharged on day 8 with a normal chest roentgenogram.

Patient 2

A 35-year-old French-Haitian woman, who lived in New York City, presented to The New York Hospital with 2 days of high fever (40°C), rigors, drenching sweats, and headache. Her symptoms started 7 days after a 5-day trip

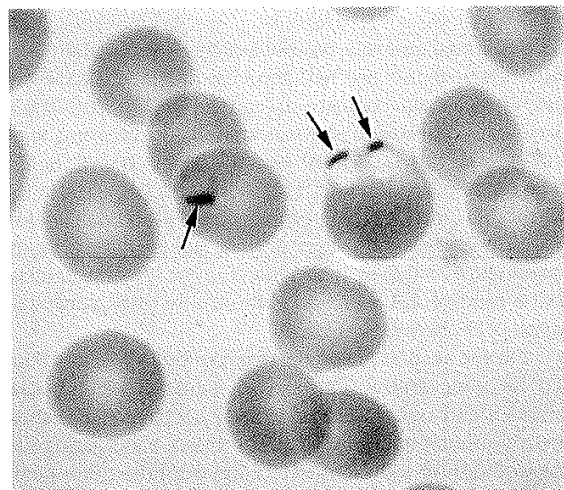


Figure 1. Giemsa-stained admission peripheral blood smear. Arrows indicate characteristic intra-erythrocytic ring and multiple appliqué forms (merozoites) of *P. falciparum*.

*Department of Medicine, Memorial Sloan-Kettering Cancer Center and
†Department of Medicine, New York Hospital-Cornell Medical Center, New York, New York.

Address correspondence to Dr. Amar Safdar, Fellow, Infectious Disease Service, Memorial Sloan-Kettering Cancer Center, 1275 York Avenue, New York, NY 10021. E-mail: safdara@mskcc.org.