

Case Report

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Severe *falciparum* malaria associated with massive pulmonary embolism

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Abstract

Falciparum malaria is known to cause alterations in the coagulation cascade, including disseminated intravascular coagulation. Microthrombotic complications are the best described; however, a number of cases of thrombosis involving larger vessels have been published in the literature. Herein, we describe the case of a woman with malaria associated with massive pulmonary embolism.

Keywords: Falciparum, malaria, pulmonary embolism

Résumé

Le paludisme à *falciparum* est connu pour causer des altérations dans la cascade de coagulation, y compris une coagulation intravasculaire disséminée. Microthrombotic complications sont mieux décrites ; Toutefois, un certain nombre de cas de thrombose impliquant des navires plus grands ont été publié dans la littérature. Ici, nous décrivons le cas d'une femme souffrant de paludisme, associé à une embolie pulmonaire massive.

Mots-clés : Falciparum, le paludisme, l'embolie pulmonaire

Case Report

A 61 year-old market vendor was admitted to Mulago Hospital in January 2011 with a four-day history of weakness and progressive depressed mental status. Labored breathing and rigors then developed, though no fevers were recorded. She smoked cigarettes and was known to have ischemic heart disease for which she had previously been treated with aspirin, carvedilol and captopril. On examination, her axillary temperature was 35.5°C, blood pressure was 70/40 mm Hg, pulse rate was 90/minute, and respiratory rate was 20/minute. Her oxygen saturation was 66% while breathing ambient air and increased to 95% on three liters of supplemental oxygen via nasal cannula. She appeared severely ill, was conscious yet confused,

and had conjunctival pallor. Her jugular venous pulsation was elevated to 10 cm and her superficial neck veins were engorged. First and second heart sounds were normal and the pulmonic component of the second heart sound was prominent. There was a right infra-axillary pleural rub. Her abdomen was tender with hepatosplenomegaly. Extremities were cool with bilateral pitting edema. Her neck was supple without signs of meningismus. There were no focal neurologic deficits.

An electrocardiogram [Figure 1] was notable for signs of right-sided heart strain and a bedside transthoracic echocardiogram revealed bulging of the inter-ventricular septum into the left ventricle, gross dilatation of the right ventricle, an estimated pulmonary artery pressure greater than 40 mm Hg,

and absence of a significant pericardial effusion. Blood tests revealed the following: White blood cell count 9.2×10^9 cells/L (63% were neutrophils and 37% were lymphocytes), hemoglobin 10.4 g/dL and platelets 73×10^9 cells/L without clumping. Blood slide for malaria revealed 4+ *Plasmodium falciparum* trophozoites by semi-quantitation. Partial thromboplastin time was 67.7 seconds, prothrombin time was 55.3 seconds and international normalized ratio was 4.7. Fibrinogen and D-dimer were not obtained due to logistical constraints.

We clinically suspected pulmonary embolism and initiated treatment with tinzaparin (low molecular weight heparin). We also initiated treatment for severe malaria with intramuscular artemether. By the following day, the patient's attendants had gathered the funds needed to obtain a contrast-enhanced CT scan of the chest. This was notable for a large pulmonary embolism in the right pulmonary artery [Figure 2] and gross dilatation of the right ventricle [Figure 3].

The patient's clinical appearance and vital signs gradually improved and supplemental oxygen was removed. Antimalarial treatment was changed to artemether/lumefantrine. Laboratory parameters also improved: Blood slide was negative by day six, platelets had increased to 174×10^9 cells/L by day 11, and coagulation studies improved (INR 1.8, partial thromboplastin time 38 seconds) by day 12. Abdominal and pelvic ultrasound scans did not reveal any evidence of underlying malignancy. On hospital day 13, she developed signs concerning for spinal cord compression. We suspected a spinal hematoma and discontinued anticoagulation. Unfortunately, over the subsequent days, signs of right-sided heart failure reappeared, she became progressively obtunded, and ultimately expired. Post-mortem examination was not performed.

Malaria is common throughout the developing world, with approximately 200 million cases annually. Ninety-one percent of the 655,000 deaths from malaria in 2010 occurred in Africa and these were largely due to infection with *Plasmodium falciparum*.^[1] Malaria has long been known to cause activation of the coagulation cascade and to result in disseminated intravascular coagulation.^[2] The hypercoagulable complications of malaria typically manifest in the microvasculature. However, several cases of malaria resulting in symmetric peripheral gangrene^[3,4] and intracranial venous thrombosis^[5] have been reported. This case is the first to document an association of malaria and pulmonary embolism. We suspect that infestation with *Plasmodium falciparum* in this patient without hypercoagulable risk factors other than tobacco use, resulted in disseminated intravascular coagulation manifesting



Figure 1: Patient's electrocardiogram was notable for evidence of right ventricular strain, including a right axis deviation, large S waves in lead I, and inverted T waves in the anterior leads

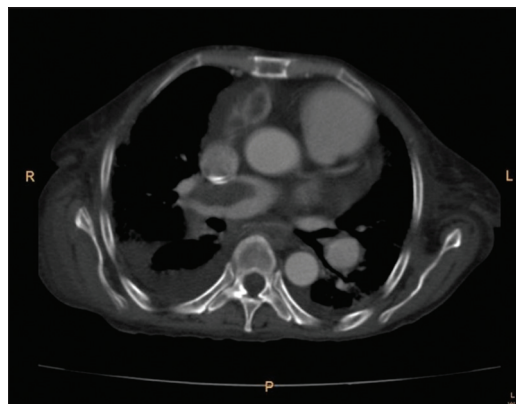


Figure 2: CT scan with intravenous contrast revealing a large intraluminal filling defect in the right pulmonary artery consistent with pulmonary embolus

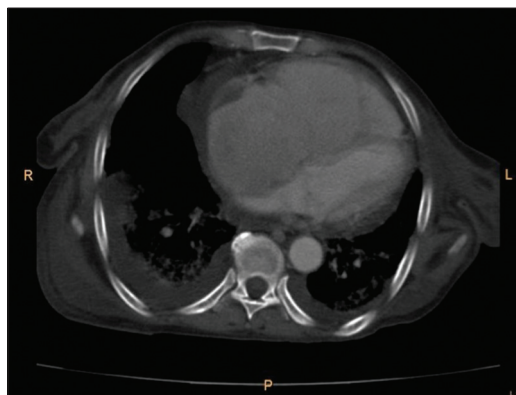


Figure 3: CT scan with intravenous contrast revealing gross dilatation of the right ventricle

as massive (indicating the presence of hemodynamic compromise) venous thromboembolism. Clinicians caring for patients with malaria should be aware of this life-threatening complication and initiate prompt appropriate treatment once suspected.

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