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MALARIA IN PREGNANCY: UPDATE ON EMERGENCY MANAGEMENT

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□ **Abstract—Background: Pregnancy complicates the diagnosis, treatment, and clinical course of malaria. This clinical problem may be encountered in emergency department patients due to international travel. Case Report: A primigravida woman at 20 weeks gestation presented to the Emergency Department with episodic fever, chills, headache, and nausea after travel to India and Asia. She had not taken malaria prophylaxis. After hospitalization, she developed acute respiratory distress syndrome and required intensive care management. Although she ultimately recovered from severe infection with *Plasmodium vivax,* she was not able to sustain her pregnancy and suffered a miscarriage. Conclusion: This case illustrates the serious nature of malaria in the pregnant patient. For this high-risk group, there is an increased incidence of severe anemia, as well as acute respiratory distress syndrome and pulmonary edema. A guideline is presented for the initial choice of anti-malarial drug treatment for the pregnant patient.** © 2011 Elsevier Inc.

□ **Keywords—malaria; pregnancy; quinine; Coartem®**

INTRODUCTION

Malaria is a serious, multi-organ disease. For some patients, malaria can be life-threatening. The pregnant patient with malaria is more likely to develop severe manifestations of the disease, with increased mortality and increased risk for fetal loss (1).

In the emergency department (ED), one should consider a diagnosis of malaria in a patient with fever and other non-specific symptoms such as myalgias, headache, vomiting, and cough, in conjunction with a history of travel to tropical or subtropical regions (2–4). Because no chemoprophylactic regimen has been found to be completely effective against malaria, the diagnosis should be considered even in travelers who have taken appropriate prophylactic therapy (5). Rarely, cases of malaria are reported in the United States in non-travelers from various geographic locales (6–8). Hyperpyrexia > 39 °C is present in more than 60% of patients on initial presentation with acute malaria; as many as 20% may not have a measurable fever at the time of their ED visit (9,10).

The diagnosis of malaria is initially confirmed with the identification of trophozoites in red cells on Giemsa-stained smears of peripheral blood (2,3,9). The sensitivity of detection by peripheral smear is approximately 90%; detection is improved when blood is sampled in the time interval between fever paroxysms (2,11). Numerous rapid antigen "dipstick" assays have been developed with improved sensitivity compared to microscopic analysis of stained blood smears, with less dependency on the expertise of the microscopist (12,13).

Pregnancy complicates the clinical course, diagnosis, and treatment of malaria. Pregnancy is associated with downregulation of maternal immune responses, to protect the fetus from rejection. This altered immunity explains, in part, the association of pregnancy with more severe malaria (14). Anemia is more pronounced in pregnant

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patients due to hemolysis from rupture of infected erythrocytes, exacerbated by erythrocyte sequestration in the spleen and liver, as well as iron deficiency (2,15–18). Thrombocytopenia, another key hematologic feature of malaria, is more common in the pregnant patient (2,19,20). The diagnosis of malaria thus may be confused with the HELLP syndrome (hemolytic anemia, elevated liver enzymes, and low platelet count) of pregnancy (1).

Hypoglycemia is more commonly seen in the pregnant patient with malaria, due to increased consumption by host and parasite, as well as glycogen depletion and impaired gluconeogenesis (21). This may be exacerbated by drug treatments for malaria (22). *Plasmodium* parasites uniquely sequester in the placenta in the case of *falciparum* malaria, contributing to miscarriage, premature labor, and low-birth-weight infants (2,14,22,23). Placental sequestration leads to an increased possibility of false-negative malaria smears, especially in primigravidas (1).

The physiologic changes associated with pregnancy make the patient more prone to congestive heart failure, which is exacerbated by anemia (20). Anti-malarial drug treatment requires consideration for the potential adverse effects on the fetus, balanced against the need for rapid and effective therapy to attack the parasites within the mother, to reduce complications for both. The following case illustrates the serious nature of malaria in the pregnant patient.

CASE REPORT

A 39-year-old primigravida woman, at 20 weeks gestation, presented to the ED with complaints of intermittent fevers associated with shaking chills, sweats, malaise, nausea, and mild frontal headache of 2 or 3 days' duration. She also reported mild dyspnea on exertion, and cough productive of clear sputum. In between episodes of fevers, she was notably asymptomatic. She denied chest pain, abdominal pain, dysuria, or vaginal discharge; she had noted dark-colored urine over the previous day. She had returned several days earlier from a 3-week trip to several Southeast Asian countries, including her native India, as well as Singapore and the Philippines. The patient had not taken malaria chemoprophylaxis, both because of her fear of medication effects on her fetus and her belief that many years of living in India had rendered her immune to malaria, although she had resided in the United States for more than a decade.

Medications on presentation were limited to prenatal vitamins and acetaminophen taken approximately 90 min before her arrival in the ED; she was otherwise in good health.

On physical examination, the patient was a pleasant, alert woman who was complaining of shaking chills and myalgias. Oral temperature was 38.2 °C (100.8°F), pulse was 110 beats/min, respiratory rate was 24 breaths/min and unlabored; blood pressure was 131/73 mm Hg. Oxygen saturation by pulse oximetry was 100% on room air. She was mildly diaphoretic. No scleral icterus or conjunctival pallor was noted. The lungs were clear, and the heart sounds were regular without murmurs or gallops. The abdomen was non-tender with a uterine fundus palpable at the level of the umbilicus; fetal heart tones were measured at 160 beats/min by bedside Doppler ultrasonography. Hepatosplenomegaly was not present. There was no peripheral edema and no rashes were observed.

Laboratory studies revealed a white blood cell count of 3600/mm^3, hematocrit was 37%, and platelet count was 90,000/mm^3. Peripheral blood smear was reported as positive for malaria trophozoites.

Electrolytes revealed sodium of 133 mEq/L; the other electrolytes were normal, as were glucose and renal function levels. Urinalysis was negative for white or red blood cells. An electrocardiogram showed a sinus tachycardia. A chest radiograph was recommended but refused by the patient.

After consultation with the Infectious Disease service, she was administered oral quinine as well as intravenous clindamycin. She was admitted to the Obstetrics floor; the laboratory subsequently identified schizont forms in the patient's blood smear consistent with *Plasmodium vivax.* Parasitemia was measured at 0.4%.

The patient's hospital course was complicated by the rapid development of pulmonary edema and acute respiratory distress syndrome (ARDS), with hypoxia and respiratory distress necessitating intubation and mechanical ventilation the day after admission. She became hypotensive after intubation, with a systolic blood pressure of 82 mm Hg; intravenous pressors were administered. Her hematocrit fell to 24% and platelet count fell to 63,000/mm^3. Transaminases rose to the level of 350 u/L. Anti-malarial regimen was changed to intravenous quinidine.

The following day, the patient suffered a spontaneous miscarriage, with an estimated 300 cc of blood loss. The placenta was examined with special staining techniques that were negative for malarial parasites.

Over the next few days in the hospital, the patient was able to be weaned from mechanical ventilatory support. She ultimately recovered and was discharged after a 3-week long hospital stay.

DISCUSSION

Five species of *Plasmodium* are associated with malaria in humans. *Plasmodium falciparum (P. falciparum),*