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Case Report Open Access

Recurrent Fever in a Healthy 30 Year Old Pregnant Female

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Abstract

A 30-year-old female who is 34 weeks pregnant was admitted with a 3 day history of fever associated with rigors, chills, nausea, and vomiting. Her blood pressure was normal; there was no tachycardia with fever. On admission, her white blood cell count was 4.4×10^3/μL with 3% bands and 74% neutrophils, hemoglobin was 11.0 g/dL, and platelet count was 63×10^9/L Human immunodeficiency virus testing was negative. On day 2 of admission, she developed a fever of 38.8°C resulting in fetal bradycardia, thus prompting an emergent cesarean section. A healthy male infant was delivered. The hematoxylin and eosin stained preparation of the placenta is shown in Figure 1. What is your diagnosis?

Keywords: Malaria; Malaria + Pregnancy; *P. falciparum*; Congenital + Malaria

Discussion

The woman has *Plasmodium falciparum* malaria. Figure 2 shows *P. falciparum* parasites in the placenta section, hematoxylin and eosin stain, 1000X. The arrows depict parasitized red blood cells (RBCs) in the maternal circulation. Figure 3 is a peripheral smear of the cord blood that demonstrates <0.01% parasitemia with a single ring trophozoite. Figure 4 is a Wright stain peripheral smear from the mother's blood, with the arrows depicting multiple ring trophozoites and 5.1% parasitemia.

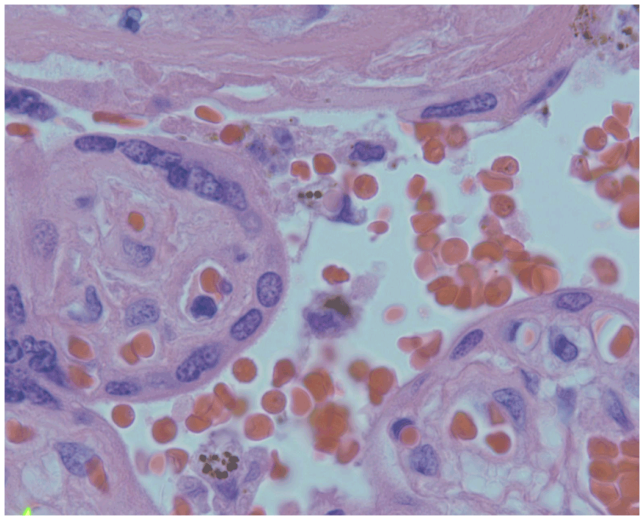

Figure 1: Placenta section, H&E stain, 1000X

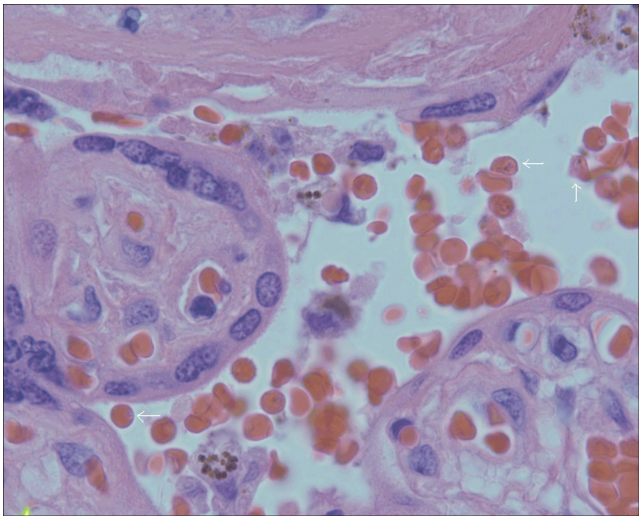

Figure 2: Placenta section, H&E stain, 1000X. Arrows depicting parasitized RBCs in maternal circulation.

This patient immigrated to the United States from Cameroon one week prior to her illness, reporting multiple mosquito bites prior to immigration, as well as a history of malaria treated in her previous pregnancy. We are uncertain as to the details of the prenatal care that our patient received prior to her arrival in the United States. Oral quinine and intravenous clindamycin were initiated 24 hours before emergent cesarean section. Zero percent parasitemia was demonstrated on her peripheral smear after 48 hours of therapy. Although the cord blood had very low parasitemia, the infant's peripheral smear demonstrated no (0%) parasitemia; he remained well and did not receive therapy.

Malaria is rarely diagnosed in West Virginia, however it must be considered as part in the differential of any febrile patient immigrating or returning from an endemic region. Malaria is a protozoan infection caused by the parasite *Plasmodium*. Four species of *Plasmodium*

(*falciparum, vivax, ovale,* and *malariae*) cause the majority of infections in humans, with recent recognition of a new potential human species, the simian pathogen, *P knowles*. Malaria is most commonly transmitted by the bite of the female Anopheles mosquito, but can be transmitted via blood transfusions and acquired congenitally via transplacental or peripartum infection. Once acquired, *Plasmodium* undergoes a complex life cycle, typically resulting in an acute febrile illness 1-2 weeks after transmission, associated with rigors, chills and temperatures up to 40°C. Ruptured schizonts in the erythrocytic cycle of the human blood stage result in fever, which characteristically occurs every 48 hours (*P. falciparum*, *P vivax*, *P ovale*) and every 72 hours (*P malariae*).

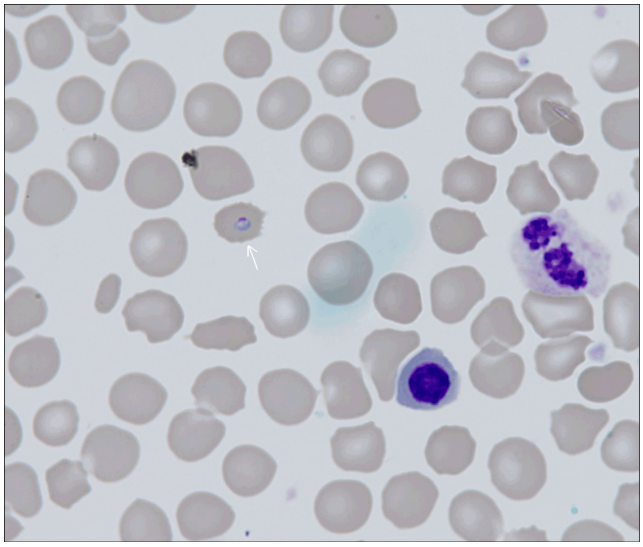

Figure 3: Peripheral smear of the cord, arrows depicting single ring trophozoite.

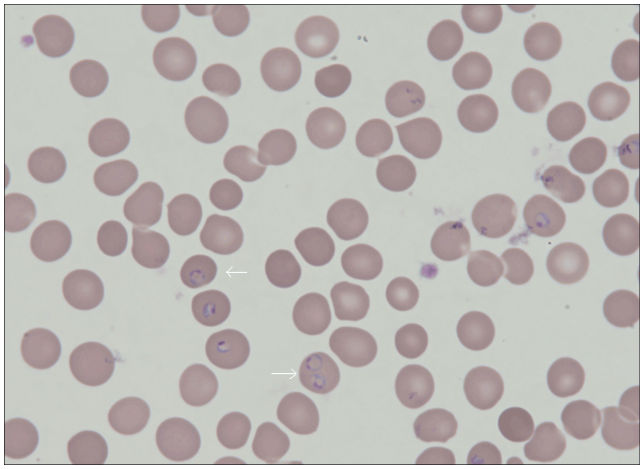

Figure 4: Peripheral smear of the mother's blood, Wright stain, arrows depicting multiple ring trophozoites, 5.1% parasitemia.

The susceptibility of a person to *P. falciparum* malaria is partly determined by the level of immunity; pregnant women have been shown to have a risk of severe malaria that is three times as high as that among non-pregnant women [1]. Elucidation of the immunologic alterations and adaptations that occur during pregnancy suggests that older concepts of pregnancy as a state of systemic immunosuppression are oversimplified; instead pregnancy may be viewed as a modulated immunologic condition, not a state of immunosuppression. Decreases in adaptive immunity seen in later stages of pregnancy with diminished numbers and function of CD4+, CD8+, and natural killer cells could affect antiviral, antifungal, or antiparasitic responses and delay clearance of the offending microorganism. Pregnancy thus may lead to an increased risk of mortality in malaria from higher degrees of parasitemia, in part due to this altered immune response to hormonal, immunological and hematological changes. Furthermore, the placenta is an active immunologic site, capable of interacting with and demonstrating a tropism of specific pathogens (e.g., listeria or *P. falciparum*); *P. falciparum* parasites selectively accumulate in the placenta. Syncytiotrophoblastic chondroitin sulfate A (CSA) and hyaluronic acid have been identified as adhesion molecules for parasite attachment to placental cells. While CD36 cells are the main endothelial receptor in the vasculature system, *P. falciparum* membrane protein-1 (PfEMP1) variants bind to CSA, but not CD36 in the placenta [1,2]. Lack of immunity to PfEMP-1 variants during pregnancy, may leave a woman highly susceptible to new infection from malaria, which may be less severe if there has been previous exposure to CSA binding of parasites. Placental infection from malaria elicits the production of inflammatory cytokines which activates the maternal immune system and may lead to placental damage and miscarriage or preterm labor, intrauterine growth retardation, anemia, or stillbirth [3].

Preventative strategies with perinatal care and education along with mosquito nets are a very important part of maternal care in endemic areas [4,5]. Intermittent preventive treatment with suphadoxin-pyrimethamine in certain parts of the world has also shown to help prevent malaria in pregnancy [4]. Treatment of malaria in pregnancy includes chloroquine to treat uncomplicated malaria caused by all species, unless there is concern about *P. falciparum* chloroquine-resistant species. When *P. falciparum* chloroquine-resistance is suspected, mefloquine or a combination of oral quinine and intravenous clindamycin is recommended [6]. Prompt recognition, diagnosis, and treatment of malaria in a pregnant woman is vital for the successful outcome for mother and child.

References

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