Congenital Malaria: Diagnosis and Therapy

To the editor:

Malaria is the leading cause of parasitic death in the world, causing 120 million cases of infection annually.[1] In contrast, congenital malaria remains relatively rare with only about 300 cases reported in the literature in total; however, congenital malaria can result in significant morbidity and mortality. Many infants with congenital malaria present during the second month of life with nonspecific signs and symptoms.[2] We describe a 5-week-old infant with congenital malaria whose presenting symptoms were jaundice and hepatosplenomegaly whose mother had emigrated to the United States from an endemic region for malaria.

Our patient was a full-term black male infant who presented with jaundice at 5 weeks of age to his primary care provider. He was initially evaluated at a community hospital at 6 weeks of age where a sonogram revealed hepatosplenomegaly. He was referred to Children's National Medical Center (CNMC) when his peripheral smear showed ring forms suggestive of malaria. The infant's mother was from Liberia and was diagnosed to have had multiple episodes (>15) of malaria. She had malaria during her pregnancy twice, once at 3 months and again at 5 months' gestation when she presented with fever. The mother was administered chloroquine for 10 days on both occasions as treatment. She emigrated to the United States at 6 months' gestation. She informed neither her obstetrician nor pediatrician of her recent malaria episodes. She did not receive any prophylactic antimalarial medications during her pregnancy. The child was born full term via normal vaginal delivery. Review of systems revealed a "slight fever" at home in the child.

On examination, the infant was alert and in no apparent distress. Vital signs revealed a temperature of 38.2°C taken rectally, pulse of 168 beats/minute, respirations of 36 breaths/minute, and blood pressure of 99/49 mmHg. The physical examination was remarkable for mild jaundice, scleral icterus, hepatosplenomegaly, and mild jaundice. Initial leukocyte count was 8,200/mm^3 with 15% segmented neutrophils, 1% band forms, 28% monocytes, and 51% lymphocytes. Hemoglobin was 6.0 g/dL. The thin malaria preparation revealed *P. falciparum* ring forms and gametocytes and percent parasitemia was determined to be 4.4%.

The patient was admitted to CNMC. Initial blood and urine fluid cultures were negative. The patient was treated with quinine 25 mg/kg/day divided every 8 hours. Throughout the hospital course the infant remained afebrile. The patient was discharged after 96 hours of hospitalization with 0% parasitemia and reticulocytes of 13%. He completed a total of 5 days of oral quinine and received one fourth tablet of Fansidar® at the end of his quinine therapy. In clinic follow-up the infant was without fever or jaundice but had persistent hepatosplenomegaly. The percent parasitemia was 0% and reticulocytes were 2.2%.

There have been 49 cases of congenital malaria reported in the United States since 1950.[2] For women having an overt attack of malaria during pregnancy, the rate of congenital malaria is estimated to be 1% to 4%.[3] Congenital malaria is more common in infants of women who have immigrated from areas in which malaria is endemic than in women who have been raised to maturity in such areas.[4]

A classic presentation of congenital malaria includes fever, anorexia, lethargy, anemia, hyperbilirubinemia, thrombocytopenia, and splenomegaly. The mean presentation of congenital malaria is at 5.5 weeks with a range of 0 to 60 weeks. In a review of 49 infants with congenital malaria, fever was noted in 100% of cases and hepatosplenomegaly in 84% of cases. Bilirubin and hepatic enzyme values are elevated secondary to intravascular hemolysis and hepatic congestion in approximately one third of the cases. Most of these abnormalities revert to normal several days after successful therapy. Nonspecific findings include failure to thrive, poor feeding, regurgitation, and loose stools. Congenital malaria can also be complicated by other illness such as pneumonia and septicemia.[2]

To make the diagnosis, a single peripheral blood smear may not be enough.[2] The cord blood and placenta should be smeared for the malaria parasite. If the placenta is heavily infected with trophozoites, this should raise one's suspicion for congenital infection. Although there currently are no clinical trials to support this management, some authors feel that if the mother has fever and parasitemia at delivery, then the patient should be treated empirically.[5] The therapy of congenital malaria is outlined in Table 1. If the percent parasitemia in the infant is less than 5%, one can treat with oral medications. At 5% parasitemia or greater, the patient will need intravenous therapy with quinidine gluconate; with 10% or higher parasitemia, exchange transfusion must be considered. The infant and mother

Table 1

THERAPY OF CONGENITAL MALARIA

- For chloroquine-susceptible strains:
  oral chloroquine phosphate (10 mg of base/kg followed by 5 mg of base/kg at 6, 24, and 48 hours)
- For chloroquine-resistant *P. falciparum*:
  oral quinine sulfate (25 mg/kg/day in three doses for 5 days) and clindamycin (20–40 mg/kg/day in three doses for 5 days) or quinine and Fansidar (Fansidar dose for infants <1 yr is a single dose of one fourth tablet given on the last day of quinine therapy)

should be followed up as outpatients to ensure eradication of the malaria parasite. Examinations should assess for fever, splenomegaly, and jaundice. Complete blood counts and percent parasitemia should be obtained at 1 and 4 weeks posttreatment. For additional expertise in management, the CDC (Division of Parasitic Disease) should be contacted.

Roopa Viraraghavan, MD, MPH[1]
Barbara Jantausch, MD[2]
[1]*Division of Anti-Infective Drug Products, Food and Drug Administration, 9201 Corporate Blvd., Room S316, HFD-520, Rockville, MD 20857, and*
[2]*Department of Infectious Diseases, Children's National Medical Center, George Washington University Medical Center, Washington, DC*

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Should Pediatric Hospitalists Replace the Staff/Ward Attending?

To the editor:

I often wonder if pediatric residents feel the way I did 17 years ago. Each monthly rotation seemed to end just as I was getting comfortable. Who would be my next senior resident(s)? Who would be the staff/ward attending? Would she be a good clinician and teacher or would she rather be in her research lab? Do residents today think the same way? Health care today is so different. The growth of managed care is forcing pediatric providers away from the hospital and academic center and more toward the community.[1] Many argue that radical changes in pediatric training programs will be necessary and the hospital inpatient service will become an anachronistic site for learning clinical medicine.[2,3] Because hospital length of stay is shortening, most of the important diagnostic problems will be solved outside the hospital.

Some managed care organizations have embraced the hospitalist model for delivery of inpatient care. Hospitalists are inpatient "specialists" who manage the care of patients during their entire hospital stay and temporarily take the place of the primary care practitioner.[4] This model is not new. Hospitalists have been common in Canada and Great Britain for many years. Proponents in this country believe hospitalists will utilize resources more judiciously and provide a higher level of care for inpatients.[4] These physicians tend to be more comfortable with sicker patients who might require the input of multiple specialists. Hospitalists are also in a position to pioneer new approaches to cost-effective quality care.[4]

Where does this leave pediatric resident education? As Wachler and Goldman have pointed out, the hospitalist model can create a core group of faculty members whose inpatient work is more than a required extra responsibility.[5] This model would increase the likelihood for house staff to be taught and supervised by attending physicians highly skilled in providing inpatient care and for house staff to witness quality improvement through positive interactions with specialty consultants, outpatient physicians, discharge planners, and managed care medical directors.[5] Learning sound clinical skills would be a part of this process.[6]

It might be time to replace the staff attending with the pediatric hospitalist for the teaching of residents and the delivery of care to underserved or complicated patients who enter academic medical centers without a private at-