Imported Malaria: Problems With Diagnosis and Prevention

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Despite efforts at large-scale control, malaria remains a widespread source of morbidity and mortality. An estimated 300 million cases occur annually worldwide, approximately one million of which are fatal in tropical Africa alone.[1] Infrequently encountered in the United States, malaria may go unrecognized even among immigrants from endemic areas. Inadequate prophylaxis of travelers to endemic areas contributes to the rising incidents of malaria in US civilians.[2] These considerations prompt a review of recent experience with malaria in Nebraska.

METHODS

Information regarding the incidence and species of malaria diagnosed by blood smear in Nebraska in the past ten years was obtained from the Nebraska State Department of Health. Records of nine patients with a confirmed diagnosis of malaria in the Omaha metropolitan area in the past five years were reviewed.

RESULTS

Thirty patients have been diagnosed with malaria in Nebraska over the past ten years. Plasmodium vivax was the most common pathogen. Although P falciparum was reported only twice, it was fatal in one patient.

The presenting problems of nine Omaha area patients were reviewed. Only five gave a history of recurrent fever usually considered typical for malaria. Five patients presented with abdominal pain. Diarrhea and anemia were each found in three patients. Four patients required two or more visits to a physician before the diagnosis was considered and made. In five cases the diagnosis apparently was initially suggested by findings on routine complete blood count differential smears. Subsequent thick and thin blood smears confirmed the diagnosis.

Travel history was available for eight patients. Five were recent immigrants from endemic areas. One was a Vietnam war veteran with a history of malaria in the military service. Two patients traveled from Nebraska to endemic areas; only one of these two travelers received antimalarial prophylaxis, and she discontinued her prophylaxis prematurely after returning home.

DISCUSSION

During 1982, 930 cases of malaria diagnosed in the United States were reported to the Centers for Disease Control. Only 17 of the 930 persons acquired their infection in the United States. Transmission was congenital in seven cases and through blood transfusion in nine cases. One patient was accidentally infected by a laboratory anopheline mosquito. While malaria in foreign civilians was most common (61.7 percent of all reported cases), the number of malaria cases in US civilians was larger than in any year since 1966.[2]

The principal source of imported malaria has been Southeast Asia, first from US military personnel who served in Vietnam, and more recently among refugees. Other endemic areas commonly responsible for imported malaria in the United States include Mexico, Central America, Haiti, India, and Africa.[2,3]

Malaria can be a difficult diagnosis for physicians in nonendemic areas. In a review of civilian patients hospitalized for malaria in New York City, Kean and Reilly[4] reported that local physicians made the correct diagnosis in only 13 percent of confirmed cases. Nearly all patients noted fever, chills, and headache. Other common symptoms included myalgia, nausea, vomiting, abdominal pain, weight loss, fatigue, sore throat, lethargy, and confusion. Many patients were considered to have viral syndromes, especially patients with abdominal complaints,[4] a frequent symptom in this series as well.

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Since the vast majority of malaria cases represent imported infections, a thorough travel history may suggest the diagnosis. Definitive diagnosis is based on finding and identifying the erythrocytic stages of malarial parasites in stained blood films. Thick smears may be needed when parasitemia is low. Two or three thick smears should be taken at intervals of six to 18 hours for three successive days, if necessary.[5] Therapy should be appropriate for the malarial species identified on blood smear. Accurate travel history may help suggest the species, since the species have varied patterns of geographic distribution. In addition, P falciparum is commonly resistant to chloroquine in certain areas of South America, Panama, India, Southeast Asia, Indonesia (including the Philippines and New Guinea), and East Africa.[6]

The treatment of choice for uncomplicated malaria due to all species (except chloroquine-resistant P falciparum) is oral chloroquine phosphate. Susceptibility testing for Plasmodium species is unavailable. All cases of malaria that are due to P falciparum acquired in areas with known chloroquine resistance should be considered resistant to chloroquine, in which case quinine sulfate plus pyrimethamine-sulfadoxine (Fansidar) is the treatment of choice.[7]

Primaquine phosphate should follow chloroquine therapy of malaria that is due to P vivax or P ovale. Chloroquine is ineffective against parasites in the extraerythrocytic stages that occur with these species. Primaquine kills malarial parasites in organs such as the liver, thus decreasing the chance of relapse. Patients should be screened before treatment, since primaquine can cause hemolytic anemia in patients with glucose-6-phosphate dehydrogenase deficiency. Primaquine should not be used during pregnancy.[7]

Malaria can be prevented with prophylactic medication. Unfortunately most cases among US civilians are a result of inadequate prophylaxis. Kean and Reilly[4] found only three of 24 civilian travelers took the recommended course of antimalarial prophylaxis. In this series only one of two travelers took any prophylaxis, and that course was inadequate.

Prophylaxis should generally begin two weeks before entering and continue through six weeks after leaving an endemic area. In areas without chloroquine-resistant P falciparum, chloroquine phosphate is the prophylactic drug of choice. Pyrimethamine-sulfadoxine is indicated for single-dose therapy or prophylaxis where chloroquine-resistant P falciparum is found. Severe or fatal cutaneous reactions (erythema multiforme, Stevens-Johnson syndrome, toxic epidermal necrolysis) have been reported with pyrimethamine-sulfadoxine. Patients allergic to sulfonamides should not receive this medication; it should be reserved for high-risk patients. If used, pyrimethamine-sulfadoxine should be discontinued immediately in the event of any ill effect, especially a mucocutaneous reaction.[8] Since P vivax is also found in most of these areas, chloroquine should be taken as well. P falciparum resistant to pyrimethamine-sulfadoxine has been reported in areas of Southeast Asia (Thailand, Kampuchea), in Indonesia, Papua New Guinea, Brazil, Columbia, and East Africa. In these areas, tetracycline[6] or quinine[7] may be useful prophylaxis for selected patients.

Controversy surrounds the addition of primaquine phosphate to the final two weeks of chloroquine prophylaxis to prevent an attack after departing from areas where P vivax and P ovale are endemic. It is generally not recommended for the average traveler. It might be indicated for such individuals as missionaries or Peace Corps workers returning home after extended, heavy exposure in areas where P vivax and P ovale are prevalent.

The worldwide problems of increased transmission and microbial resistance of malarial parasites extend to nonendemic areas. Work on a malaria vaccine continues, but such a vaccine will not be available soon.[9] Travelers will continue to be at risk of contracting malaria. Physicians should remain alert and remember that although the diagnosis can be difficult, prevention remains relatively simple.

References

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