Malaria in the Traveler

How to Manage Before Departure and Evaluate Upon Return

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KEYWORDS

• Malaria • Prevention • Chemoprophylaxis • *Plasmodium* • Fever in returning traveler

KEY POINTS

- Medications for malaria chemoprophylaxis work well, and choice of agent is largely dependent on cost and side effects.
- There can be a long lag between acquisition of the malaria parasite and development of disease (months).
- Cyclic fevers are not characteristic of malaria, and many patients present with gastrointestinal disease.
- Rapid diagnostic tests are as sensitive as classic blood smears but do not depend on experienced microscopists.
- In patients from nonendemic areas who return with malaria, it should be treated as a sepsis syndrome.

OVERVIEW

This article familiarizes health care providers with the prevention, diagnosis, and treatment of malaria, with specific focus on patients from nonendemic areas who plan to travel for a limited time to an area with risk of malaria transmission.

Providers must understand malaria, because it is a disease estimated to kill approximately 600,000 people per year worldwide. Despite public health interventions that have reduced the global burden of morbidity and mortality, it remains highly prevalent throughout much of the world. For the general practitioner, it is important to recognize the clinical manifestations of malaria because it is the most common cause of fever in returning travelers[1] and one of the few conditions that can kill patients rapidly if mismanaged. In 2012, approximately 2000 cases were seen in the United States,

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including 6 preventable deaths.[2] After reading this article, the authors hope that readers will be able to prevent such needless loss of life.

MALARIA

Malaria is an infection caused by any of 5 species of the *Plasmodium* parasite: *P falciparum*, *P vivax*, *P ovale*, *P malariae*, and *P knowlesi* (Fig. 1). These single-celled eukaryotic organisms are transmitted to humans by the bite of an *Anopheles* mosquito. Within minutes of the bite, parasites enter the liver, where they develop and multiply asymptomatically. After 10 to 14 days, parasites leave the liver and enter

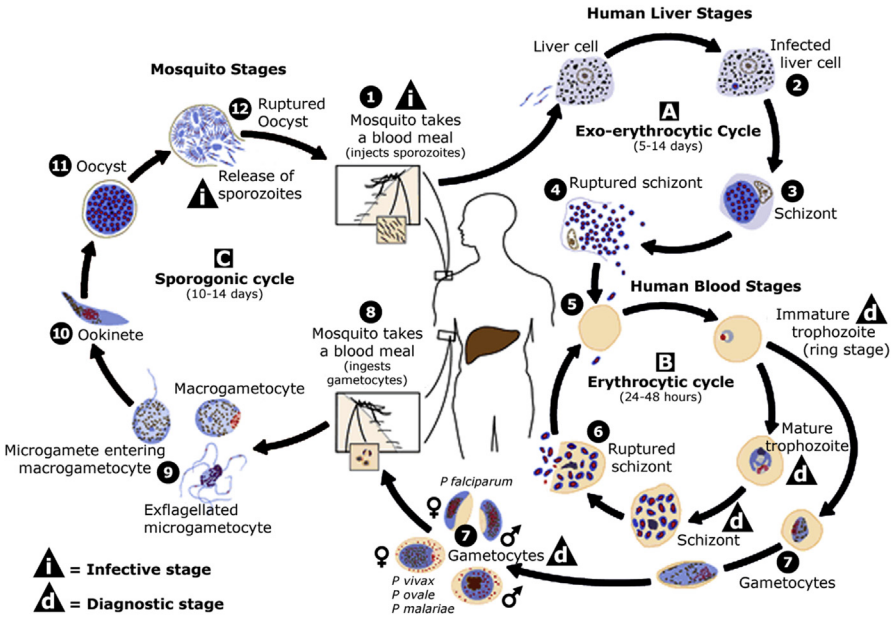

Fig. 1. Malaria life cycle. The malaria parasite life cycle involves 2 hosts. During a blood meal, a malaria-infected female *Anopheles* mosquito inoculates sporozoites into the human host (1). Sporozoites infect liver cells (2) and mature into schizonts (3), which rupture and release merozoites (4). (In *P vivax* and *P ovale*, a dormant stage [hypnozoites] can persist in the liver and cause relapses by invading the bloodstream weeks, or even years, later.) After this initial replication in the liver (exoerythrocytic schizogony [A]), the parasites undergo asexual multiplication in the erythrocytes (erythrocytic schizogony [B]). Merozoites infect red blood cells (5). The ring stage trophozoites mature into schizonts, which rupture, releasing merozoites (6). Some parasites differentiate into sexual erythrocytic stages (gametocytes) (7). Blood-stage parasites are responsible for the clinical manifestations of the disease. The gametocytes, male (microgametocytes) and female (macrogametocytes), are ingested by an *Anopheles* mosquito during a blood meal (8). The parasites' multiplication in the mosquito is known as the sporogonic cycle (C). While in the mosquito's stomach, the microgametes penetrate the macrogametes generating zygotes (9). The zygotes in turn become motile and elongated (ookinetes) (10), which invade the midgut wall of the mosquito where they develop into oocysts (11). The oocysts grow, rupture, and release sporozoites (12), which make their way to the mosquito's salivary glands. Inoculation of the sporozoites (1) into a new human host perpetuates the malaria life cycle. (*From* Centers for Disease Control and Prevention. About malaria. Available at: http://www.cdc.gov/malaria/about/biology/. Accessed August 26, 2015.)