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Opinion

Plasmodium Vivax: Outbreaks and Overview

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Plasmodium vivax, a human malarial parasite occurs in both tropical and temperate zones, between 45o N and 40o S. In 2014, Egypt (Aswan Governorate) had an outbreak of *Plasmodium vivax* after several years of maintaining zero indigenous cases. Twenty-one confirmed cases during May-June 2014 were detected. In 2011, as of 27 December, 20 *Plasmodium vivax* malaria cases were identified from Lakonia, Evrotas, and other geographical areas in Greece. During the mid-20th century, malaria transmission in the United States was largely eliminated. Between 1951 and 1990, in the United States, 21 introduced malaria outbreaks that all were caused by *Plasmodium vivax*. Nevertheless, 14 of these outbreaks occurred in California (seven outbreaks during 1986, 1988, and 1989 (2 outbreaks in 1989 in San Diego County)). During 1997 to 2002, four separate probable mosquito-transmitted malaria outbreaks had been reported to the United States Centers for Disease Control and Prevention (US CDC), including one outbreak from Virginia. This report described the investigation of two patients with *Plasmodium vivax* malaria that occurred in northern Virginia in August 2002. In 2002, in Kyrgyzstan, 2,744 malaria cases were registered mainly in the Fergana valley with 98 % of *Plasmodium vivax* and 2 % of *Plasmodium falciparum*. The investigation revealed that malaria outbreak in Kyrgyzstan was imported from Uzbekistan, Azerbaijan, Tajikistan, and Afghanistan. Because of only intraerythrocytic schizogony occurring, neither transfusion nor congenital malaria is expected to relapse. Spreading of *Plasmodium vivax* infection is exclusively by female anopheline mosquitoes. Individuals with *Plasmodium vivax* (or *Plasmodium ovale*) infection may have relapses after several weeks, months or years. *Plasmodium vivax* and *Plasmodium ovale* primarily infect young erythrocytes, whereas *Plasmodium malariae* infects older erythrocytes. *Plasmodium falciparum* infects erythrocytes of all ages. The diagnosis of *Plasmodium vivax* malaria is made by history of traveling, physical examination, amoeboid shape of growing trophozoites, appearance and size of erythrocyte, Maurer 's dots, irregular parasite cytoplasm, appearance of parasite pigment, number of merozoites (12-24, average is 16), parasite' s stages identified in circulating blood. Mixed infections is found approximately 5 %, mostly are *Plasmodium vivax* and *Plasmodium falciparum*. Chloroquine remains the treatment of choice for *Plasmodium vivax* malaria, except in the geographically contiguous Papua Guinea and Indonesia' s Irian Jaya (Western New Guinea) region. Korea and India are areas that developed chloroquine resistance. Artesunate is the drug of choice for *Plasmodium vivax* malaria when chloroquine resistance and chloroquine contraindication developed. Detecting gametocytes of *Plasmodium falciparum* in an individual obviously infected with *Plasmodium vivax* is diagnostic. Individuals who lack certain Duffy blood group determinants are protected against *Plasmodium vivax* infection. Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency has been associated with protection from malaria, but scientific evidences are less striking than with these other genetic abnormalities. In conclusions, mostly, *Plasmodium vivax* outbreaks are due to local transmissions. To prevent transmission and control the disease on long term, the development of an integrated preparedness and response plan for malaria that covers all aspects from surveillance, clinical management, laboratory diagnosis, entomological surveillance, vector control, and communication is necessary.

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