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Editorial

Plasmodium knowlesi: The New Emerging Fifth Human Malaria Parasite

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In the early 1930's, *Plasmodium knowlesi* was first officially reported in India in a *Macaca fascicularis* specimen from Singapore. Naturally, this malarial parasite occurs in pig-tailed and long-tailed macaques that inhabit forested areas of Southeast Asia. *Plasmodium knowlesi* is morphologically similar to *Plasmodium malariae*, hence human infections could be diagnosed by the conventional methods. This malarial parasite causes mild and chronic infection in its natural hosts, such as *Macaca fascicularis* and *Macaca nemestrina*. In contrary, this parasite infections in rhesus macaques (*Macaca mulatta*) run a fulminant and lethal course if untreated. Humans are susceptible to *Plasmodium knowlesi* infection with severe clinical manifestations. The first confirmed human natural infection that could be transmitted from monkeys to humans was evidenced in 1967 by Chin., *et al.* using *Anopheles balabacencis* (part of the *Anopheles leucosphyrus* group) in their experiment. The vectors belong to the genus *Anopheles*, subgenus *Cellia*, series *Neomyzomyia* and group *Leucosphyrus.* The important vectors are *Anopheles cracens* and *Anopheles latens*. A case report of 38-year-old man came to the Netherlands in January 2009 to work as a rigger in the harbor of Rotterdam. He had lived in Kapit, Sarawak, in Borneo, Malaysia and hunted wild animals in the surrounding jungles. This man came to the hospital with a 5-day history of high fever, headache, low back pain, myalgia, and remarkable jaundice. A previous study demonstrated that commercially available rapid diagnostic antigen tests for human *Plasmodium* species can detect human *Plasmodium knowlesi* infections, although infections with a low parasitemia will not be detected. The definite diagnosis in this patients that confirmed by the polymerase chain reaction was *Plasmodium knowlesi* infection.

The spectrum of clinical manifestations includes high fever with chills and rigors, headache, malaise, myalgia, anorexia, abdominal pain, cough, tachypnea, tachycardia, nausea, vomiting, and diarrhea. Approximately 15 - 25 % of patients present with hepatosplenomegaly. In patients with severe illness, they may present with jaundice, respiratory distress, hypoglycemia, hypotension, thrombocytopenia, and renal failure. No presenting clinical manifestations can distinguish *Plasmodium knowlesi* malaria from *Plasmodium falciparum* or *Plasmodium vivax* malaria. Because of non-sequestering nature of *Plasmodium knowlesi*, neurological manifestations are not found in the course of the illness. Life-threatening or lethal complications may occur in a minority of cases although *Plasmodium knowlesi* has a benign clinical course. Treatment of uncomplicated *Plasmodium knowlesi* infection includes conventional antimalarial drugs, such as chloroquine, quinine, and mefloquine. Primaquine in a dose of 15 mg for 2 days is required for gametocyte clearance. Chloroquine with conventional doses of 10 mg base/kg, followed by 5 mg base/kg at 6, 24 and 48 hours-total dose 25 mg base/kg has a rapid parasite clearance. In severe and complicated *Plasmodium knowlesi* infection, treatment with intravenous quinine is required. Twenty mg/kg of quinine is loaded in 10% dextrose solution, followed by 10 mg/kg, dose 8 hourly for 7 days. Hypoglycemia and cardiac arrhythmia are the adverse side-effects of quinine. The use of intravenous artemisinin derivatives that are evidenced by clinical studies is limited. Nevertheless, there were favorable treatment outcomes in rhesus monkeys and excellent efficacy against human Plasmodium knowlesi strain in patients treated with artesunate.

In conclusion, *Plasmodium knowlesi*, although is benign clinical course and less prevalent, can develop severe illness. This parasite is still highly susceptible to artemisinin derivatives. Mefloquine is not useful in treatment of Plasmodium knowlesi malaria due to the potential resistance of the parasite.

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