

A Case of Recurrent Plasmodium Falciparum

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INTRODUCTION

CASE & RESULTS

DISCUSSION

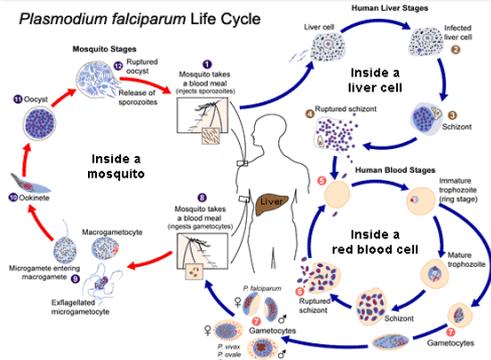


Figure 1: Life Cycle of *Plasmodium* spp

Although malaria was widely eliminated in the US in 1950s, it is still a common global health issue. In 2015 estimated 214 million malaria cases and 438,000 people died worldwide. Given increasing ease of travel, malaria is becoming a more important diagnosis for US patients, especially in more immigration laden regions.

Malaria is caused by parasites of the *Plasmodium* species, transmitted by the *Anopheles* mosquito in the life cycle illustrated (Figure 1).

Plasmodium falciparum species produces the most severe form of malaria. It may cause an uncomplicated malaria similar to other *Plasmodium* species. This is characterized by constitutional symptoms of fever, malaise, fatigue, sweats, nausea, and vomiting. Symptoms usually occur in a cyclic pattern. However, *P. falciparum* also has the potential to cause severe disease with normocytic normochromic anemia, renal failure, acute respiratory failure, hypotension, and hypoglycemia. It is also capable of causing cerebral malaria. *P. falciparum*-infected erythrocytes attach to the blood vessel walls and do not circulate freely in bloodstream. Complications include neurologic defect may persist, including ataxia, palsies, speech difficulties, deafness, and blindness.

A 38-year old African man presented with sudden onset fever and chills for three days. He also had nausea and vomiting 5-6 times per day. Ibuprofen alleviated his symptoms, but they relapsed on the third day. He had recently visited his home country Senegal for 2 weeks and just returned 6 days ago. He did not take malaria prophylaxis. He denied contact with ticks and rodents, but did report mosquito exposure. The patient is an immigrant from Africa and had a history of malarial disease 5 years ago, which had self-resolved.

Physical examination was grossly normal with the exception of mild right upper quadrant tenderness. Blood parasitology was positive for *Plasmodium falciparum*. Blood smear shown below (Figure 2). Interestingly, patient had a positive PPD and was tested for Mycobacteria species. His blood work was positive for co-infection with *Mycobacterium avium* complex, which he was asymptomatic for. HIV test was negative. Patient was started on quinine sulfate 648 mg PO TID and doxycycline 100mg IV Q12H. Blood glucose dropped below 60mg/dl after treatment initiation. Patient was on POCT glucose monitoring as both malaria and quinine may induce hypoglycemia. After treatment for 5 days, patient had three consecutive negative blood parasites smear. Patient was discharged with an oral 7-day course of quinine and doxycycline.

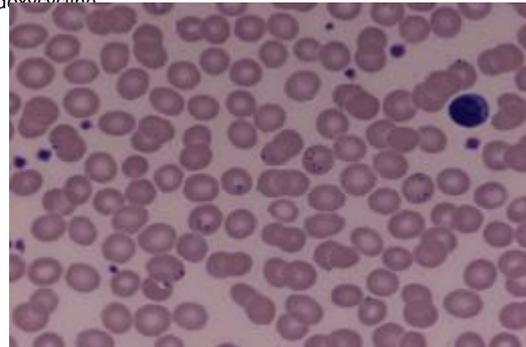


Figure 2: Peripheral smear of patient's blood demonstrating intracellular *Plasmodium*

This case demonstrates the importance recognizing malaria as a potential diagnosis in the US. Although our hospital demographics include a large immigrant population, increasing travel accessibility may increase malaria cases in the US.

The patient developed hypoglycemia after initiating quinine therapy. It is unclear whether this complication is due to malarial disease or quinine side effect. *P. falciparum* is known to interfere with metabolism and induce hypoglycemia. Quinine has been reported to induce hyperinsulinemia, thus causing hypoglycemia. Additionally, renal insult may decrease quinine excretion and augment its effects. Since *P. falciparum* may cause renal impairment, blood glucose must be monitored carefully when starting treatment. In this case, D5NS infusion was used to successfully treat hypoglycemia.

Blood smear with *P. falciparum* <5% is considered hyperparasitemia. Given patient's prior history of malarial infection, it is thought he may have developed a partial immunity protecting him from a more severe course of disease. Given the patient's ethnicity, a sickle cell prep can be considered. In this case, the patient had a positive PPD with negative HIV testing. Further testing demonstrated co-infection with asymptomatic *Mycobacterium avium* complex. Although experimental models have suggested that Mycobacteria may have a protective effect against malarial parasites, these studies were conducted with *M. tuberculosis* and *P. yoelii*. The protective effect has not yet been demonstrated with MAC and *P. falciparum* although there may be a similar mechanism.

REFERENCES

1. Mali S, et al. Malaria surveillance – United States, 2008. MMWR Surveill Summ. 2010;59(SS07):1-15.
2. Newbold C, et al. Cytoadherence, pathogenesis and the infected red cell surface in *Plasmodium falciparum*. Int J Parasitol. 1999;29(6):927-37.
3. Centers for Disease Control and Prevention. Malaria: Disease. <http://www.cdc.gov/malaria/about/disease.html>
4. Roberts DJ, et al. The clinical and pathophysiological features of malarial anaemia. Curr Top Microbiol Immunol. 2005;295:137.
5. White NJ, Breman JG. Harrison's Principles of Internal Medicine, 19th ed, Kasper D, Fauci A, Hauser S, et al (Eds), McGraw Hill, New York 2015.
6. Page KR, Jedlicka AE, Fakheri B, Noland GS, Kesavan AK, Scott AL, Kumar N, Manabe YC. 2005. Mycobacterium-induced potentiation of type 1 immune responses and protection against malaria are host specific. Infect Immun 73:8369-8380