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Case Report: Plasmodium Vivax Malaria With Ocular Involvement

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Introduction

Malaria remains one of the most important parasitic infections worldwide. Although Plasmodium falciparum is the species most often associated with severe outcomes, there is increasing recognition that Plasmodium vivax can also cause life-threatening complications. Traditionally considered a benign form of malaria, P. vivax has been linked to multi-organ dysfunction, severe anemia, and, more rarely, ocular involvement.

Ocular manifestations of malaria have been best studied in falciparum infection, where malarial retinopathy with retinal whitening, vessel discoloration, hemorrhages, and papilledema correlates strongly with cerebral malaria and patient outcome [3,4,6,9]. Histopathology and angiography demonstrate microvascular obstruction and blood–retinal barrier disruption [7]. With newer technologies such as optical coherence tomography (OCT) and fluorescein angiography (FA), more detailed insights into microvascular and structural damage are now possible [1,2].
Ocular involvement in P. vivax malaria is less commonly documented but is increasingly recognized. Case reports and small series describe retinal hemorrhages, neurosensory detachments, internal limiting membrane detachment, and optic neuritis [8,10,15]. In some observational studies, ocular findings correlate with systemic disease severity [15]. These observations challenge the long-held perception that P. vivax is uniformly benign. We report here a case of P. vivax malaria complicated by ocular involvement, contributing further evidence to this spectrum.

Case Presentation

A 35-year-old Ethiopian male with no known comorbidities had recently arrived by sea. During his journey, he developed fever, chills, generalized weakness and headache. He also developed abdominal pain. On arrival to our hospital, he have severe symptoms included altered consciousness, hypotension, jaundice, and profound weakness.

There was no history of seizures, blackwater urine, or respiratory distress.
He reported frequent evening mosquito exposure during the rainy season. He did not use insecticide-treated bed nets, repellents, or chemoprophylaxis. He denied prior episodes of malaria and had not received malaria vaccination.
Past Medical History: No known chronic illnesses (HIV, renal, or hepatic disease). No drug allergies. No history of G6PD deficiency. No regular medications.
Social/Occupational History: He was a migrant worker traveling by sea under poor and crowded living conditions, without preventive health measures. No sick contacts were reported.

Clinical Examination: Vital signs: BP 82/44 mmHg, HR 125/min, RR 28/min, Temp 36.5°C. He was icteric with diffuse abdominal tenderness. Neurological assessment confirmed impaired but arousable consciousness.
Laboratory Findings: Hemoglobin 7.76 g/dL, platelets 101 ×10⁹/L, WBC 6.98 ×10⁹/L, INR 1.13, AST 55 U/L, ALT 24 U/L, creatinine 58.7 μmol/L, BUN 5.8 mmol/L, total bilirubin 50.8 μmol/L (direct 27.3 μmol/L). Both RDT and peripheral smear were positive for P. vivax, parasitemia index 1.7%.
Ophthalmologic Findings: The patient developed progressive bilateral blurring of vision during admission. Seen by ophthalmology and fundus examination revealed multiple retinal hemorrhages, whitish lesions around the optic disc, and optic disc swelling. Differential diagnoses included malaria-related retinopathy, CMV retinitis, herpetic retinitis, and syphilitic retinitis. Advanced retinal imaging was not available.
Management: He was treated with intravenous artesunate for 5 days, then oral pyrimethamine–sulfadiazine for 3 days, followed by primaquine for 14 days. Supportive care included hydration with normal saline and analgesia. Ophthalmology recommended follow-up.

Discussion

This case underscores the potential severity of Plasmodium vivax malaria, which is often misperceived as a benign condition. Ocular involvement in malaria has been most extensively studied in P. falciparum, where malarial retinopathy is a well-established prognostic marker in cerebral malaria. The spectrum includes retinal whitening, hemorrhages, vessel discoloration, and papilledema, all of which correlate with microvascular sequestration and impaired perfusion [3,4,6]. Histological studies confirm parasitized RBC sequestration, rosetting, and disruption of the blood–retinal barrier [7,29].

In P. vivax, although retinal changes are less common, recent literature demonstrates that this parasite can cause significant ocular morbidity. Case reports have documented hemorrhages, neurosensory detachment, and optic neuritis [8,10,15]. OCT and FA studies provide mechanistic insights, showing paracentral acute middle maculopathy and acute macular neuroretinopathy linked to ischemia of the deep retinal layers [1,2]. These findings parallel systemic pathology, as P. vivax is now recognized to cause severe anemia, hepatic dysfunction, and neurological complications.
Our patient's presentation with altered consciousness, jaundice, severe anemia, and ocular involvement highlights this broader pathogenic potential. Although CMV retinitis and herpetic retinitis were considered in the differential, the temporal relationship with acute malaria, positive blood smear for P. vivax, and improvement after anti-malarial therapy strongly support malaria-related retinopathy as the cause. Importantly, ocular symptoms improved in parallel with systemic recovery, consistent with previous reports.
Clinicians should recognize that P. vivax may cause vision-threatening complications even in the absence of cerebral malaria. Routine ophthalmologic evaluation should be considered in malaria patients presenting with visual symptoms, regardless of the Plasmodium species. Further studies are needed to define the prevalence, mechanisms, and long-term outcomes of ocular vivax malaria, particularly with the use of modern imaging.

Outcome

The patient demonstrated gradual systemic improvement with anti-malarial therapy. He became hemodynamically stable, his jaundice resolved, and his level of consciousness normalized. At follow-up after discharge, he reported significant improvement in systemic symptoms and near-complete resolution of visual complaints, with no new ocular findings documented.

Conclusion

Ocular involvement in Plasmodium vivax malaria, although rare, is an important clinical entity. This case adds to the growing body of evidence that vivax malaria can cause severe systemic disease and vision-threatening complications. Awareness of such presentations can facilitate early recognition and management, improving both systemic and ocular outcomes.

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