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# Plasmodium vivax - Schistosoma mansoni coinfection in a Sudanese immigrant to Greece

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**Case Report**

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## Abstract

Systematic reviews of malaria–schistosomiasis coinfection focus exclusively on *Plasmodium falciparum*, leaving the coinfection of *P. vivax* and *Schistosoma*spp largely uncharacterized. We report the case of a 22-year-old Sudanese immigrant, residing in Greece, who was diagnosed with *P. vivax* malaria by microscopy, rapid antigen detection, and PCR. Serology for *Schistosoma mansoni* indicated concurrent chronic schistosomiasis. Treatment with Artemether–lumefantrine plus primaquine, and praziquantel was planned. However, he self-discharged before completing radical cure and was subsequently traced to the United Kingdom. This case illustrates the need of suspicion for polyparasitism in immigrant populations, and for cross-border mechanisms to ensure treatment.


## Full Text

*Plasmodium vivax*, one of five *Plasmodium* species causing human malaria, has the widest global distribution and is the dominant species in most countries outside sub-Saharan Africa [1]. *P. vivax* has long been considered largely absent from sub-Saharan Africa due to the high prevalence of the Duffy-negative phenotype that confers resistance to erythrocyte invasion. However, it remains endemic in the Horn of Africa and Madagascar and is increasingly reported across West, Central, and East Africa, including in Duffy-negative individuals [2]. Greece has been malaria-free since 1974, apart from almost yearly imported cases and several locally acquired *P. vivax* cases in 2009–2012. Of 44 imported cases in 2025, seven were *P. vivax*: five in immigrants, and two in travelers [3].

*Schistosoma* spp. are blood flukes that cause acute and chronic human schistosomiasis and are reported from 79 countries across tropical and subtropical regions globally [4]. *S. mansoni*, *S. japonicum*, *S. mekongi*, *S. guineensis* and *S. intercalatum* cause intestinal and hepatosplenic schistosomiasis, while *S. haematobium* causes urogenital schistosomiasis. In Greece, no autochthonous transmission of *Schistosoma spp.* has been documented, although sporadic imported cases among immigrants have been reported.

Malaria and schistosomiasis overlap extensively across the WHO African and Eastern Mediterranean regions, making co-infection frequent. In 2024, the WHO African Region accounted for 94% of the estimated 282 million malaria cases, and the Eastern Mediterranean Region (in which WHO places Sudan) for a further 11.1 million, of which 26.3% were due to *P. vivax*. Sudan alone contributed 44.6% of the regional total [2]. Sudan and South Sudan are recognized among the countries most affected by schistosomiasis, due to both *S. mansoni and S. haematobium, with 8,868,182 people requiring* preventive chemotherapy in 2024 *[*5]. Despite this overlap, evidence on the frequency, clinical presentation, sequelae, and determinants of malaria and schistosomiasis co-infection remains limited. Furthermore, it is confined almost entirely to *P. falciparum*, being essentially absent for the *P. vivax–S. mansoni* combination.

We present the case of a 22-year-old immigrant from Sudan, with fever of unknown origin, who was diagnosed with *P. vivax – S. mansoni* co-infection.

He presented with a six-day history of fever up to 39 °C, preceded by chills and accompanied by malaise, without other symptoms. At fever onset, he had consulted a private dentist for concurrent dental pain and was given nimesulide, amoxicillin, and metronidazole, with resolution of both fever and dental pain. After three symptom-free days the fever and chills recurred, although the dental pain had resolved.

The patient had arrived in Greece by sea, as an undocumented immigrant, seven months earlier and had since lived in a refugee camp in central Athens. His medical history included malaria in childhood, reportedly treated with intravenous and oral antimalarials. He reported that in Sudan he lived in a village beside a small river, in which he swam regularly.

The clinical examination, except that he was febrile, was unremarkable. The chest radiograph was normal. Abdominal ultrasound demonstrated splenomegaly (16 cm), and whole-body computed tomography showed homogeneous hepatosplenomegaly without other abnormalities.

Laboratory testing revealed leukopenia, microcytic anemia and thrombocytopenia: WBC 4.8 × $10^3$/μL (neutrophils 3.5, lymphocytes 0.6, monocytes 0.7, eosinophil 0.0) × $10^3$/μL, Hb 9.6 g/dL, Hct 31.2%, MCV 82.4 fL, MCH 25.3 pg, MCHC 30.8 g/dL, PLT 55 × $10^3$/μL. CRP was 8.53 mg/dL and procalcitonin 38.13 ng/mL (reference < 0.5). Renal and hepatic indices were normal (urea 16 mg/dL, creatinine 0.74 mg/dL, AST 15 IU/L, ALT 28 IU/L, total bilirubin 1.10 mg/dL, ALP 45 IU/L, γ-GT 36 IU/L, LDH 171 IU/L). Serology for HBV, HCV, and HIV was negative, and urinalysis revealed no pathological findings. Blood cultures were all negative.

A rapid immunochromatographic test (*P. falciparum*/pan, Abbott) was positive for non-falciparum malaria. Giemsa-stained thick and thin blood films showed trophozoites, morphologically identified as *Plasmodium vivax,* and multiplex PCR confirmed it. (**Figure 1**) Quantitative glucose-6-phosphate dehydrogenase activity was normal, permitting the use of primaquine. The patient was treated with artemisinin-based combination therapy (ACT), specifically artemether/lumefantrine (20/120 mg; four tablets at 0, 8, 24, 36, 48, and 60 h, 24 tablets in total), to be followed by primaquine phosphate 0.5 mg/kg/day (30 mg base) for 14 days.

An extensive serological screening was carried out, which was significant for a strongly positive *Schistosoma mansoni* IgG result (index value 51, positive >11) (*Schistosoma mansoni* IgG, Demeditec Diagnostics GmbH, Germany). Stool microscopy for ova in a single stool sample examined by the formalin-ethyl acetate sedimentation method was negative, as is common in light or chronic infection. Positive, low-titer serology for several viruses (Coxsackievirus, Echovirus, Epstein–Barr, HSV-1/2) and bacteria (*Chlamydophila pneumoniae*, *Mycoplasma pneumoniae*, *Rickettsia conorii*) was attributed to antibody cross-reactivity during acute malaria [6]. Chronic schistosomiasis was diagnosed, and praziquantel was indicated according to Centers for Disease Control and Prevention (CDC) [7] and World Health Organization (WHO) [4] guidance.

By the third hospital day, his fever and chills had declined, and by day 7, platelet count, CRP, and procalcitonin had normalized, although anemia persisted. Praziquantel was sourced through the

National Organization for Medicines and the National Public Health Organization. Before its arrival, however, the patient self-discharged, having received the complete ACT course but only five of 14 primaquine doses and no praziquantel. The National Public Health Organization traced him to Birmingham, United Kingdom, where he had arrived via Belgium and France. The receiving health authorities completed his primaquine regimen and administered praziquantel.

We describe a case of concurrent *P. vivax* malaria and serologically diagnosed schistosomiasis.

*P. vivax* has long been considered uncommon in sub-Saharan Africa because Duffy negativity limits erythrocyte invasion, but this generalization does not extend to the Horn of Africa. This case, illustrates that vivax malaria should not be dismissed as a possibility in febrile immigrants from this region.

The patient had not travelled since his arrival in Greece seven months prior; reinfection is therefore implausible. The most likely explanation for his infection is relapse due to the presence of hepatic hypnozoites, from an infection he had acquired probably in Sudan, or at some point during his travels whence he sailed to Greece. Given that most malaria in Sudan is due to *P. falciparum*, radical cure for *P. vivax* would not have been routinely indicated.

In the present case, diagnosis of hepatosplenic schistosomiasis was based on a high titer of anti-schistosome antibodies and a compatible epidemiological history of exposure to freshwater in a highly endemic area. Infection was attributed to *S. mansoni* based on the specificity of the ELISA kit used, the presence of homogeneous hepatosplenomegaly, on computed tomography, and the lack of symptoms suggestive of urogenital schistosomiasis in the patient. The fact that a single stool examination was negative does not exclude the diagnosis, as the ease of ova detection depends on the parasitic burden and the duration of the infection. Additional testing for urinary schistosomiasis was not undertaken, because imaging and urinalysis were completely normal. Schistosomiasis is frequently associated with peripheral eosinophilia, which was absent in our case. Eosinophilia may, however, have been masked by the concomitant malaria, an infection known to cause eosinopenia during the acute phase [8].

*Plasmodium–Schistosoma* co-infection is well described, but the evidence is either entirely derived from *P. falciparum*, or only rarely distinguishes between *P. falciparum* and *P. vivax* [9].

Praziquantel was unavailable locally and required sourcing from another part of the country. However, the patient self-discharged before completing primaquine and before praziquantel arrived. These developments highlight two areas of interest: the national availability of antiparasitic drugs, and the continuity of care for mobile populations across European borders.

ECDC guidance concludes that screening newly arrived migrants from endemic countries for schistosomiasis and strongyloidiasis is likely to be effective and cost-effective, “conditional on burden in migrants' country of origin” [10]. Global coverage for schistosomiasis was reportedly below 40% in 2024, so preventive chemotherapy cannot be reliably assumed for arrivals from endemic countries, including our patient [4]. Effecting such guidance requires surveillance and control measures for polyparasitism

among migrants arriving in Europe. Although the National Public Health Organization of Greece has established tools for the early diagnosis and appropriate treatment of malaria, considerable work remains regarding surveillance, reporting systems, and drug supply for other imported parasitic infections, such as schistosomiasis.

In conclusion, this is a report of concurrent *P. vivax* malaria and chronic schistosomiasis in a migrant from Sudan. Both infections are common in his country of origin, and their coexistence is more likely an underrecognized occurrence. Clinicians in non-endemic settings should, therefore, consider polyparasitism in febrile migrants, rather than stopping after obtaining a first positive result. The management of this case also emphasizes that firstly, praziquantel was not stocked locally and had to be located through national channels over several days. It has become apparent that antiparasitic drugs used infrequently in Europe require defined national reserves and rapid access pathways. Second, regimens that depend on prolonged adherence are poorly suited to highly mobile populations. Therefore, establishing cross-border arrangements to ensure continuity of antiparasitic treatment would reduce the risk of malaria relapse and of long-term sequelae, such as those of chronic schistosomiasis, in patients who are lost to follow-up.

## Declarations

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All authors read and approved the final manuscript.

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# Figures

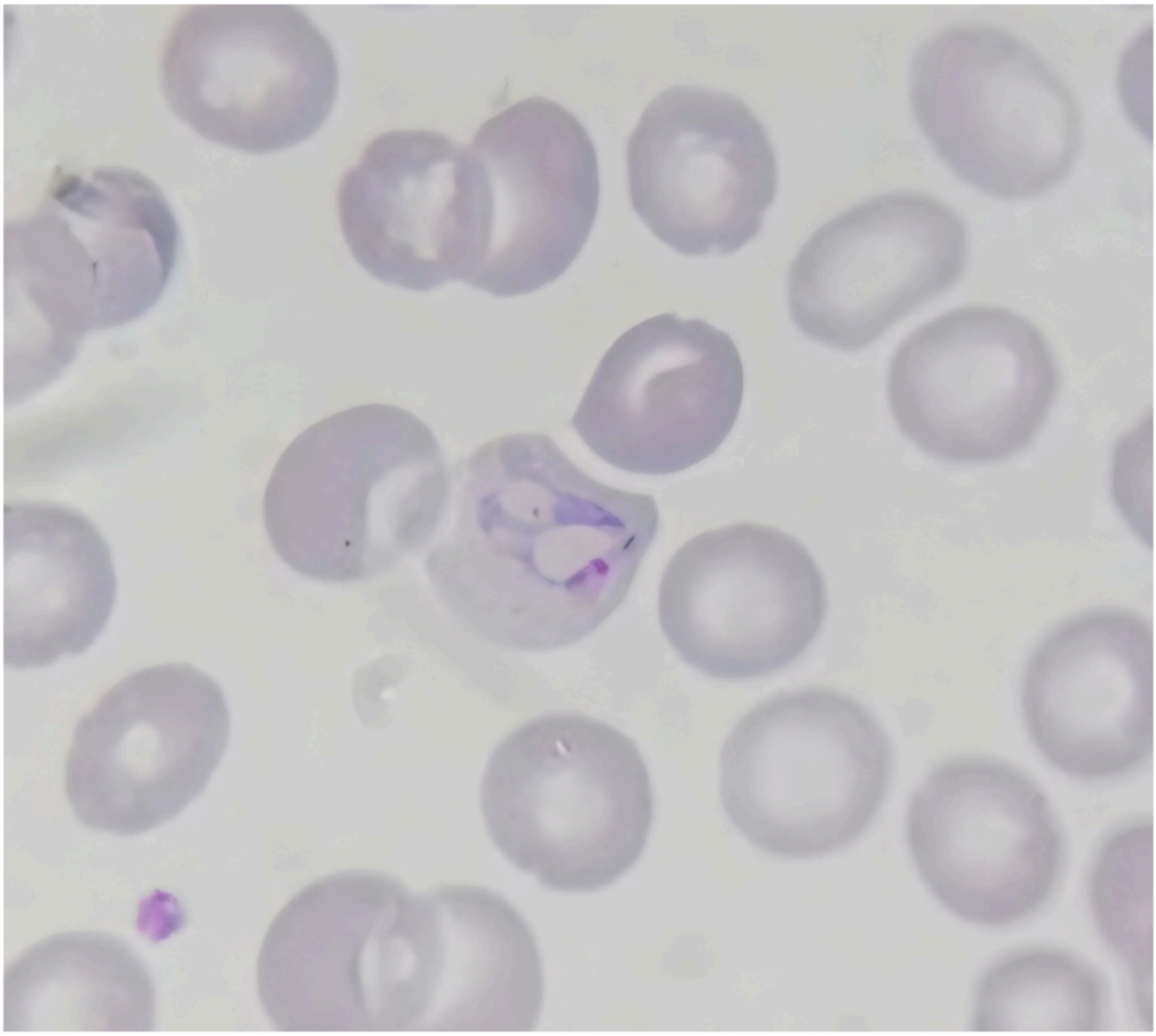


**Figure 1**

*Plasmodium vivax* trophozoite within an enlarged erythrocyte (Giemsa stain, 100x).