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Case Report: Delayed or Recurrent *Plasmodium falciparum* Malaria in Migrants: A Report of Three Cases with a Literature Review

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Abstract. Emerging evidence indicates that migrants from *Plasmodium falciparum* endemic regions are at risk of delayed presentation of *P. falciparum* malaria. We report three cases of *P. falciparum* malaria occurring years after arrival in Europe. All patients were originally from Sub-Saharan Africa. Two subjects had controlled human immunodeficiency virus infection and one was a pregnant woman. We performed a literature review of all published cases of delayed presentation of *P. falciparum* in migrants and identified 32 additional cases. All cases but one originate from sub-Saharan Africa. There was a median time of 36 months between the last visit to a malaria-endemic country and clinical malaria (range: 3 months to 10 years). Pregnancy was the most frequently reported risk factor (11/35 or 31.4%). Parasitemia was ≤ 0.1% in 38% of cases (11/29 reported), and no death was reported. The underlying possible mechanisms for this delayed presentation in migrants from an endemic area probably include the persistence of submicroscopic parasitemia combined with decaying *P. falciparum*–specific immunity. Suspicion of *P. falciparum* delayed malaria should remain high in migrants, mainly from sub-Saharan Africa, even without a recent travel history, especially in those presenting risk factors for impaired parasite clearance or distinct immune responses such as pregnancy and HIV infection. In these patients, new prevention and screening strategies should be studied and blood safety policies adapted.

INTRODUCTION

Imported *Plasmodium falciparum* malaria is frequent: the World Health Organization estimates that more than 10,000 travelers suffer from malaria each year,[1] with immigrants "visiting friends and relatives" travelers being at higher risk.[2] In travelers, *P. falciparum* malaria mostly manifests within the 2 months of return from an endemic area.[3] Nevertheless, emerging evidence from both epidemiological studies and case reports indicates that migrants are particularly at risk to present clinical *P. falciparum* malaria later after return from an endemic area.[4–7] *Plasmodium falciparum* malaria has also been anecdotally reported in subjects, with no recent history of travel after blood transfusion or organ transplant[8] and in subjects living near airports probably after a bite of an imported mosquito (airport or odyssean malaria).[9,10]

We report three cases of *P. falciparum* malaria occurring years after arrival in Europe and review the literature for additional cases of delayed clinical *P. falciparum* infection in migrants from an endemic area.

CASE 1

In July 2016, a 27-year-old woman presented to her first prenatal consultation in the context of a second pregnancy after a miscarriage. Gestational time was 26 weeks. The patient was originally from the Republic of Guinea but had moved to Belgium 2 years earlier, without ever leaving Belgium since. She only mentions having received the visit of her parents 1 week before symptoms started. She complained of general fatigue, anorexia, shivering, diffuse arthralgia, diarrhea, and nausea since the past 3 weeks. Her physical examination was notable for tachycardia (110 beats/minute) and sub-pyrexia (37.9°C). The blood test revealed a normochromic normocytic anemia (Hb 9.2 g/dL) with a normal platelet count (164,000/μL). The alkaline phosphatases, gamma-glutamyl transpeptidase (gGT), lactate dehydrogenase (LDH), and C-reactive protein (CRP) were elevated (117 UI/L, 62 UI/L, 340 UI/L, and 14 mg/L, respectively). A blood smear revealed the presence of *P. falciparum* with a parasitemia level of 0.4%. A rapid antigen test (SD Bioline; Standard Diagnostics, Gyeonggi-do, South Korea) was positive for *Plasmodium* spp. and *P. falciparum*. *Plasmodium falciparum* serology was strongly positive by the indirect immunofluorescence assay (IFA) (> 1/640). The patient was hospitalized and treated with atovaquone/proguanil 1,000/400 mg once a day for 3 days after which the blood smear became negative, and the patient was discharged. The pregnancy outcome was favorable.

CASE 2

In December 2015, a 38-year-old man, originally from Guinea-Bissau, presented to the emergency department for 2-day long pyrexia with associated chills and nocturnal diaphoresis, generalized myalgia and asthenia, anorexia, weight loss with vomiting, and diarrhea. The patient had a history of HIV-1 infection treated by tenofovir/emtricitabine/efavirenz since 2013. Recent CD4 T-cell count and HIV viral load were 557/μL and < 20 copies/mL, respectively. The patient stated having recently traveled to Portugal where he shared an apartment with compatriots. He had returned to Brussels 2 weeks before symptoms appeared and had no history of travel outside Europe for the past 4 years. The blood test revealed thrombocytopenia (44,000/μL), hyperbilirubinemia (total/direct bilirubin: 2.1/1.2 mg/dL), and elevated alkaline phosphatases, gGT, LDH, and CRP (140 U/L, 142 U/L, 530 U/L, and 121 mg/L, respectively). A blood smear was performed and was positive for *P. falciparum* (3% parasitemia). *Plasmodium falciparum* serology was positive by IFA (> 1/160). The patient was hospitalized and received an oral treatment by

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dihydroartemisinin/piperaquine (960 mg/120 mg once a day for 3 days) with a favorable clinical and biological evolution, allowing discharge from the hospital 24 hours after his admission.

CASE 3

A 30-year-old woman originally from the Republic of Guinea presented to the emergency department in January 2016, with complaints of intermittent and evening predominant pyrexia with associated chills, anorexia, generalized myalgia, and asthenia since 12 days. A diagnosis of noncomplicated cystitis was made at another hospital 3 days before, based on the presence of mild leukocyturia (33 leukocytes/μL; normal range: 0–10/μL). The patient was discharged with levofloxacin 500 mg once a day. She had a history of HIV-1 infection treated by emtricitabine/tenofovir, ritonavir, and atazanavir since 2013. The last CD4 T-cell count was 498/μL and HIV viral load was < 20 copies/mL. She had not traveled outside Belgium for the past 6 years but had recently been visited by a friend who had traveled from Rwanda. At admission, the patient was febrile (38°C) with tachycardia (134 beats/minute), confused, disoriented in time, and presented a tender right abdominal flank. The blood count revealed thrombocytopenia (36,000/μL) and leukopenia (2,830/μL) with lymphopenia (630/μL). The blood analysis was also notable for an elevated CRP level (91 mg/L), mild direct hyperbilirubinemia (total/direct bilirubin: 1.6/0.5 mg/dL), and elevated gGT and LDH levels (44 UI/L and 501 UI/L, respectively). The blood smear was positive for *P. falciparum* (0.3% parasitemia). *Plasmodium falciparum* serology was positive by IFA (> 1/640). The patient was treated with dihydroartemisinin/piperaquine (960 mg/120 mg once a day for 3 days). Three days later, there was a favorable clinical evolution with complete resolution of the neurological symptoms and gradual biological recuperation, and the patient was discharged.

In our three cases, there was no history of blood transfusion, tissue/organ transplantation, or intravenous drug use reported.

LITERATURE REVIEW

We reviewed the literature about the delayed occurrence of *P. falciparum* malaria in subjects from endemic areas. We selected the cases with sufficient information about time since last stay in an endemic area and outcome. MEDLINE searches were performed using the following MesH terms "Malaria, Falciparum" and "Recurrence" or "Emigrants and Immigrants" or "Travel". Articles published in English or French languages were identified. References and citing articles of identified articles were also searched. Thirty-five cases were identified, including the present cases, all of which are summarized in Table 1. All cases were originally from sub-Saharan Africa, except one case from Mexico reported in the United States. The median duration since last visit in an endemic country was 36 months. Pregnancy was the most frequently reported risk factor (11/35 or 31.4%), with first pregnancy in 4/11 cases. Besides the two cases presented in this study, HIV infection was reported in only one case. Parasitemia was ≤ 0.1% in 38% of cases (11/29 reported). All cases had a favorable outcome with no reported death. Only one pregnant patient had a terminated pregnancy secondary to miscarriage.

DISCUSSION

We reported three cases of *P. falciparum* malaria in migrants without a recent travel history in an endemic area and reviewed 32 additional cases published in the literature.

Plasmodium falciparum is able to persist in hosts for years as suggested by cases of transfusion-related transmission in a nonendemic area that occurred years after the blood donor had traveled to an endemic area.[11] Our analysis of the published cases indicates a median of 3 years between the last stay in an endemic area and the manifestations of clinical malaria. This finding adds to previous studies performed in migrants. A 10-year case–control study in two reference hospitals in Paris found that 2.3% of cases of *P. falciparum* malaria in migrants were diagnosed 59 or more days after arrival from the endemic area. The main risk factors associated with delayed presentation were pregnancy, HIV infection, and first migration.[4] Migrants from endemic areas with stable transmission have acquired *P. falciparum*–specific immunity and are probably able to control the parasite for years, resulting in asymptomatic low parasitemia. Accordingly, in a study performed in Spain on more than 200 asymptomatic migrants, 4.6% had a positive blood polymerase chain reaction (PCR) for *P. falciparum*. The median time since arrival was 4.5 months, and three subjects (1.4%) had arrived more than 1 year before.[7]

Late-onset clinical malaria in migrants is most probably secondary to decaying *P. falciparum*–specific immunity. A study conducted in Spain has shown that a significant proportion of immigrants who had left an endemic area for years still had a detectable amount of immunoglobulins G specific for the erythrocytic antigens of *P. falciparum*.[12] This persistence of long-lived humoral responses likely contributes to the control of *P. falciparum* blood stage parasites below the level of microscopic detection and is associated with a low frequency of biological or clinical signs or symptoms. Splenomegaly has been reported in three cases of delayed malaria.[13,14] Interestingly, in two of these cases, clinical malaria occurred after splenectomy,[13] whereas parasitemia was undetectable before surgery. The spleen being critical to allow for effective clearance of *Plasmodium*-infected red blood cells,[15] these cases illustrate the persistence of chronic submicroscopic *P. falciparum* infection.

Recrudescence of *P. falciparum* infection is likely favored by risk factors in addition to decaying immunity. In pregnant women, this phenomenon likely reflects the unique physiopathology of placental malaria, when the parasite is sequestered in the intervillous space. Specific IgG of multigravidae women inhibit binding of infected red blood cells to the placental ligand, chondroitin sulfate A, whereas those from primigravidae women and men do not,[16] making the primigravid women particularly vulnerable toward placental malaria and unfavorable outcome. Pregnancy was present in more than a third of the reported cases we identified in our literature review.

HIV infection is associated with more severe manifestations of *P. falciparum* infection, higher mortality, and prolonged parasitemia.[17] Parasite-specific memory B cells develop after repeated exposure in an endemic area and are critical for protection against the blood stage of *P. falciparum*.[18] Chronic HIV infection is associated with B-cell dysfunction that may persist even after antiretroviral treatment.[19] Accordingly, a recent study conducted in Rwanda using a protein microarray found that HIV-infected subjects had narrower IgG responses

TABLE 1

Review of published cases of delayed presentation of *Plasmodium falciparum* malaria in migrants

Reference, country, year	Country of origin	Gender, age	Latency	Risk factor	Parasitemia	Outcome	Potential cofounding factor
Carme, France, 1978[29]	Angola	M, 34	4 months ½	Not reported	Not reported	Favorable	Not reported
Revel, France, 1988[30]	Comoro Islands	M, 7	3 years	Not reported	Not reported	Favorable	Visit from family member
Krajden, Canada, 1991[31]	Ghana	M, 30	2 years and 8 months	Diabetes	1%	Favorable	Not reported
Georges, France, 1992[32]	Gabon	F, 13	9 months	Corticosteroids treatment	500/mm^3	Favorable	Not reported
Eloy, France, 1998[33]	Cameroon	F, 37	4 years	Not reported	2%	Favorable	Luggage from Cameroon
Omonuwa, USA, 2002[34]	Mexico	M, 29	1 year	Not reported	NA	Favorable	Not reported
Bidegain, France, 2005[13]	Central African Republic	M, 19	2 years	Splenectomy	0.1%	Favorable	Not reported
	Cameroon	M, 28	18 months	Splenectomy	2%	Favorable	Not reported
Giobbia, Italia, 2005[35]	Ghana	F, 29	4 years	Pregnancy	5350/mm^3	Favorable	Not reported
Howden, Australia, 2005[14]	Eritrea/Sudan	F, 28	9 years	Not reported	0.5%	Favorable	Not reported
Rapp, France, 2006[36]	Ivory Coast	F, 23	19 months	Pregnancy	0.01%	Favorable	Not reported
Greenwood, Sweden, 2008	Togo	M, 18	4 years	Sickle-cell disease	< 0.01%	Favorable	Not reported
Szmitko, Canada, 2008[37]	Angola	M, 29	8 years	Not reported	5.4%	Favorable	Not reported
Theunissen, Belgium, 2008[23]	Republic of Guinea	M, 30	9 years	Not reported	0.1%	Favorable	Contact with recently arrived migrants
Poilane, France, 2009[38]	Sub-Saharan Africa, not specified	F, 21	2 years ½	First pregnancy	0.3%	Favorable	Not reported
	Cameroon	F, 25	2 years ½	First pregnancy and untreated HIV infection	0.001%	Fetal death	Not reported
Cullen, USA, 2011[39]	Nigeria	F, 12	3 years	Not reported	NA	Favorable	Not reported
Kantele, Finland, 2012[5]	Cameroon	F, 32	13 months	Pregnancy	< 0.1%	Favorable	Not reported
	Democratic republic of Congo	F, 23	6 months	Pregnancy	< 0.1%	Favorable	Not reported
	Nigeria	F, 25	6 months	Pregnancy	< 0.2%	Favorable	Not reported
	Kenya	F, 26	3 months	Pregnancy	1.6%	Favorable	Not reported
Arends, The Netherlands, 2013[24]	Liberia	M, 23	5 years	Not reported	2.3%	Favorable	Contact with recently arrived migrants
	Sierra Leone	F, 34	10 years	Not reported	0.18%	Favorable	Contact with recently arrived migrants and airport visit
Gallien, France, 2013[25]	Sub-Saharan Africa, not specified	M, 40s	6 years	Not reported	< 0.01%	Favorable	Contact with recently arrived migrants
Berrevoets, the Netherlands, 2013[40]	Burkina Faso	M, 48	2 years ½	Not reported	3.2%	Favorable	Not reported
Odolini, Italy, 2014[41]	Burkina Faso	F, 17	4 months	First pregnancy	0.05%	Favorable	Not reported
Vantomme, Belgium, 2015[22]	Ghana	F, 52	4 years	Not reported	2.1%	Favorable	Contact with recently arrived migrants
Martelli, Italy, 2015[42]	Mali	M, 19	3 years	Not reported	0.002%	Favorable	Stay in Libya with other migrants
	Mali	M, 24	5 years	Not reported	2%	Favorable	Stay in Libya with other migrants
Dierksen, USA, 2016[43]	Liberia	F, 56	5 years	Not reported	< 2%	Favorable	Not reported
Salas-Coronas, Spain, 2017[44]	Mali	M, 38	10 years	Not reported	< 0.01%	Favorable	Not reported
Al Hammadi, USA, 2017[6]	Ghana	F, 29	2 years ¾	First pregnancy	3%	Favorable	Not reported
Dauby, Belgium, 2017	Republic of Guinea	F, 27	2 years	Pregnancy	0.4%	Favorable	Visit from family member
	Republic of Guinea-Bissau	M, 38	4 years	HIV infection	3%	Favorable	Contact with recently arrived migrants and airport visit
	Republic of Guinea	F, 30	6 years	HIV infection	0.3%	Favorable	Contact with recently arrived migrant

F = female; M = male.

toward *P. falciparum*.[20] HIV-infected migrants might be at an increased risk of impaired parasite clearance and would tend to have persistent subclinical parasitemia leading eventually to *P. falciparum* recrudescence.

Despite waning of specific immunity toward *P. falciparum*, all subjects had high titers of anti–*P. falciparum* antibodies detected by IFA reflecting anamnestic responses.[18] A recent study identified the presence of anti–*P. falciparum* antibodies to be associated with uncomplicated imported malaria[21] like in our first two cases.

Odyssean malaria occurs after the transport of infected mosquitoes from endemic to nonendemic areas through human activities.[10] It is referred to "luggage malaria" where it is hypothesized that a *P. falciparum*–infected mosquito transported through luggage is at the origin of a clinical malaria in an area without vectorial transmission. In some cases reviewed in Table 1, the visit of a family member from an endemic country or being in contact with a recently arrived migrant is considered as a risk factor for "luggage malaria."[22–25] However, it is striking that those cases of delayed malaria referred to as "luggage malaria" are always reported in migrants from malaria-endemic countries.

In our three described cases in this study, luggage malaria may thus be hypothesized, given that all three patients had been recently in close contact with people returning from malaria-endemic countries. However, we believe that most of the so-called "luggage malaria cases" reported in the literature, at least those in migrants from endemic countries, are the result of the evolution of subclinical chronic parasitemia to clinical overt malaria because of decaying immunity over time in addition to other occasional factors associated with impaired parasite clearance.

This subclinical chronic parasitemia in migrants from endemic countries poses a risk of safety of blood products. *Plasmodium falciparum* malaria after blood transfusion originating from migrants who have left their countries for years[11,26] has been described. At worst, it could also be the source of malaria spread in a malaria-free country where the vector is reimplanting. Indeed, global climate warming provokes resurgence of disappeared diseases in Europe: malaria autochthonous cases have recently been described in Italy and Greece, although these countries have been declared malaria free for several decades.[27]

CONCLUSIONS

Suspicion of delayed malaria should be high in migrants even without a recent travel history, especially in those presenting certain risk factors such as pregnancy and HIV infection. In these patients, new prevention and screening strategies should be studied based on techniques able to detect low-density parasitemia such as PCR[7] or loop-mediated isothermal amplification.[28]

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