Case Report
Report of a Case of Congenital Malaria *Plasmodium malariae* in France

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**Summary**

Congenital malaria (CM) has been considered to be rare, even in malaria-endemic areas but the disease can result in significant neonatal morbidity. Because of its rarity, the disease may go undiagnosed for a prolonged period in a seriously ill infant. We report the first case of *Plasmodium malariae* CM from a HIV mother. HIV could have facilitated the transfer of erythrocytic persistent *P. malariae* through the placenta to the fetus.

**Introduction**

An estimated 1 million deaths occur annually among African children as a consequence of malaria. The reported low incidence of congenital malaria (CM) suggests that the placenta acts as a major barrier to the parasite. The incidence of placental malaria is ~30% in endemic countries whereas the incidence of CM is estimated to be 0.3% [1]. The disease is diagnosed when parasites are found in a newborn within 7 days of birth or later if there is no possibility of post partum infection. We report here the first case of CM *Plasmodium malariae* in France.

**Case Report**

A 30-year-old HIV+ woman, delivers by planned caesarean section an apparently normal female baby, weighing 3.2 kg (7 lbs). At delivery azidothymidine is administered i.v. for 7 h. The mother is a native of the Democratic Republic of Congo (DRC), who came to France 2 years ago and has not travelled outside France since then. The only abnormality found in the baby at delivery is an anaemia (12.3 g/dl haemoglobin) attributed to anti-retroviral drugs toxicity.

At 6 weeks post-delivery, the infant is brought in for a fever of 1-day duration. She is found to have a temperature of 38.5°C and both hepatomegaly (3 cm) and splenomegaly (3 cm). Serological tests for Hepatitis B and Hepatitis C virus are negative, and polymerase chain reaction (PCR), DNA and RNA for HIV are negative. More routine laboratory exams show: haemoglobin 6.4 g/dl, platelets 122 000/µl, LDH 1080 IU/ml and blood smears showing 1.8% of RBCs parasitized with *P. malariae* (Fig. 1; confirmed by PCR). A transfusion of 125 ml of blood (serologically negative for CMV) is given to the child. Following treatment with the orally administered anti-malarial drug, parasitaemia drops to 1% by day 3. At day 7, some parasite remnants are found and the child is afebrile. Drug testing *in vitro* of the parasite shows it to be sensitive to chloroquine. A thick blood smear of the mother, taken 5 days after that of the infant, is indeed positive, while on the thin smear no parasites can be detected.

**Discussion**

CM has been considered to be rare, even in malaria endemic areas but this disease can result in significant neonatal morbidity. In studies carried out in Africa, *P. falciparum* congenital infection rate is just around 7%, while in the placental surface *P. malariae* is the predominant species [1]. *Plasmodium vivax* is the more frequent species of CM in Asia and America [2]. The more frequent species in Europe is *P. falciparum*. Only 6 cases of *P. malariae* CM have been reported worldwide since 1950 [3, 4]. Two cases were secondary to blood transfusion [5]. Particularly long intervals between

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infection and maternal transmission are observed (2 years in our case, 22 years in one case of the literature). The mean age of onset of symptoms was 3.5 weeks, longer than that in endemic countries where most of the cases are symptomatic in the first week of life [6]. There was a relationship between the age of symptoms and the Plasmodium species. Clinical features are not specific. Reduced birth weight was observed in 30% in endemic countries. The clinical picture we observed of fever, hepatomegaly, splenomegaly, irritability is consistent with the described clinical presentation of CM but can mimic other neonatal infectious diseases. Laboratory data revealed haemolytic anaemia, thrombocytopenia and hyperbilirubinaemia. Diagnosis of malaria is often established by the haematologist technologist who noted malaria parasites on routine blood smears. PCR could help in the correct determination of this disease [7]. The incidence of CM is higher in infants of non-immune mothers. Passively transferred immune IgG has been postulated to be protective in utero and in the first few months of life [8]. It is unlikely that the infection of the infant occurs as a result of transplacental transmission of the Plasmodium, since the most likely mechanism of infection is breakdown of the placental barrier that result in subsequent transmission of maternal red blood cells to the infant during labour or parturition. The onset of symptoms, typically at 4–6 weeks of age, is the estimated half-life of maternal IgG in the infant. Fetal haemoglobin, by limiting the growth of parasites may also protect the infant initially.

Materno-fetal transmission of P. malariae in our case could have been facilitated by HIV mother co-infection. The interactions between HIV and malaria during pregnancy are complex. Studies in Kenya and Malawi have shown that the prevalence and density of malaria parasites are higher in pregnant women who are also HIV+ [9]. HIV might influence the transfer of parasites through the placenta to the fetus [10]. The risk of the infant dying during the post-neonatal period has also been shown to be 3.4-fold higher in children born to HIV+ mothers with placental malaria than in those born to HIV+ mothers without placental malaria [11]. It has been demonstrated in some studies that HIV alters the patterns of malaria during pregnancy so that women of all gravidities are at the same level of risk, with the implication that prevention strategies need to be targeted to all pregnant women [12]. Impaired cell-mediated immunity caused by HIV could lead to increased frequency and severity of malaria parasitaemia [13]. There is also an association between increasing immunosuppression (as measured by the nearest CD4-cell count) and increasing parasite density. This studies concern interactions between HIV and P. falciparum in immune populations in areas of malaria endemicity. In our case, the mother lived in a non-endemic area since 2 years. The Plasmodium species isolated was not P. falciparum but P. malariae, known to persist as an asymptomatic erythrocytic disease for many years following an untreated primary infection. HIV infection could have facilitated the transfer of erythrocytic persistant P. malariae through the placenta to the fetus in non-immune women.

Plasmodium malariae CM disease may go undiagnosed for a prolonged period in a seriously ill infant specially in non-endemic area. This infection must be considered in mothers who have travelled or immigrated from endemic country.

References

