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CASE REPORT

# Corpus Callosum Hematoma Revealing Imported Neuromalarial Disease: A Case Report

EL Khahouri I[1], Tougar S[1], Maghrabi O[1], Mashrouh W[1], Mabchour M[1], Charra B*[1]

[1]**Department of Intensive Care Medicine, Ibn Rochd University Hospital, Faculty of medicine and Pharmacy of Casablanca, Hassan 2 University, Casablanca, Morocco.**

***Corresponding Author: Email:** boubaker.ch68@gmail.com

ABSTRACT

Cerebral malaria, one of the most serious complications of Plasmodium falciparum infection, is characterized by the sequestration of parasitized red blood cells (HP) within deep cerebral microvessels. Herein, we report the case of a 30-year-old man with no particular medical history, with the notion of stay in an endemic area [Ivory Coast] for 3 months. , who was admitted to our medical intensive care unit for status epilepticus. Upon admission the patient was immediately intubated due to persistent seizures. He was febrile at 39°C. Lumbar puncture was performed and was sterile. A cerebral CT showed a slight cerebral edema with exaggerated hyper density of the cerebral tent. A cerebral MRI showed a hypersignal of selenium from the corpus callosum, corresponding to a hematoma of the corpus callosum which subsequently revealed an imported neuroma aria. A thick blood smear was then performed, showing the presence of plasmodium falciparum trophozoites, following which the patient was put on Artesunate. The evolution was favorable,and the patient was extubated without neurological complications .We also point out the challenges this diagnosis may pose, especially in a non-endemic country such as Morocco.

**Introduction**

Malaria is still one of the leading infectious causes of death in endemic countries, with approximately 429,000 deaths in 2015.[1] severe forms of malaria are usuallyrelated to Plasmodium falciparum. Neurological involvement or "neuramalaria" is very common and is a major determinant of the overall prognosis.

Severe forms of malaria, such as cerebral malaria, are characterized by sequestration of parasitized red blood cells (HP) within deep microvascular beds. However, the reason why only a small proportion of infected patients develop cerebral malaria remains unclear. Althoughmechanisms leading to development of the neurological syndrome remain poorly understood, parasite sequestration within the brain, metabolic disturbances, as well as the host's immune response have been clearly implicated in the pathogenesis of this disease.[2]

**Case presentation**

We report the case of a 30-year-old man, with no particular medical history, who was admitted to our medical intensive care unit for status epilepticus. Upon admission, the patient was agitated and had a Glasgow coma scale of 10\15, with equal and reactive pupils. Blood pressure was 120\70 mm Hg with a sinustachycardia of 120 beats per minute. He was febrile at 39°C with a generalized cutaneo-mucous jaundice. The patient was immediately intubated due to persistent seizures.

**a- Imaging:**

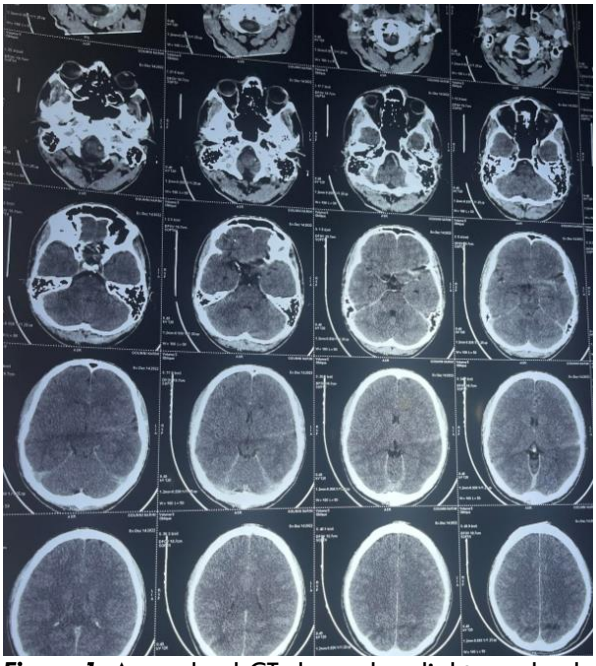

***Figure 1:*** A cerebral CT showed a slight cerebral edema with exaggerated hyper density of the cerebral tent.

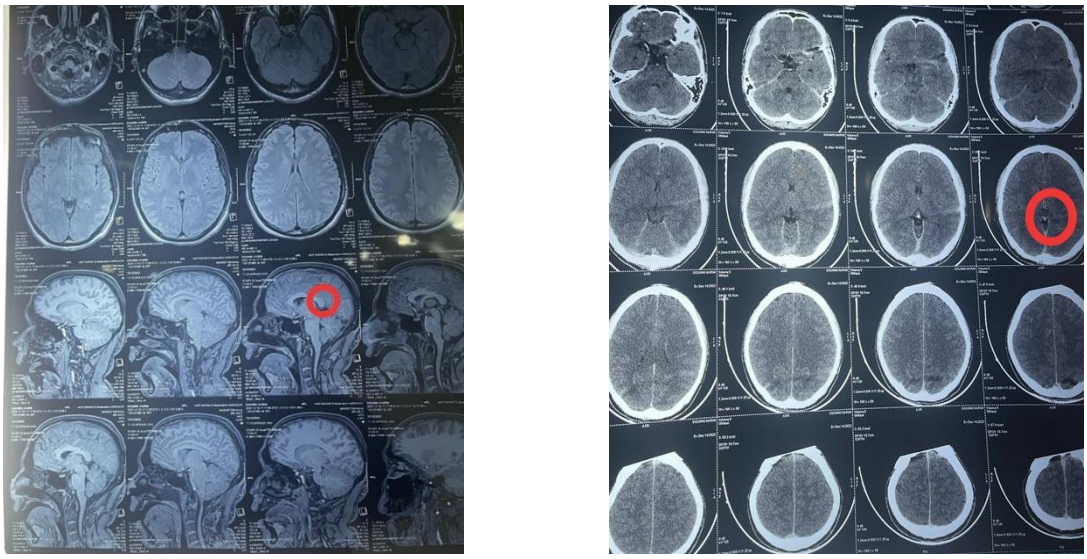

***Figure 2:*** A cerebral MRI showed a hypersignal of selenium from the corpus callosum, corresponding to a hematoma of the corpus callosum.

**b- <u>Blood results:</u>**

The blood count revealed anemia at 7.7 g\dl, thrombocytopenia at 57000/mm$^3$, leulocytosis 13790\mm3 with neutrophilia at 10910 \mm3and a normal lymphocyte countat 1970\mm3.The C-reactive protein (CRP) was positive at 232 mg\l. Renal function was preserved with urea at 0.79 g\l andcreatinine at 64.8 mg\l.

There was aliver cytolysis with slightly elevated aminotransferases: 64\96 IU\l;hyperbilirubinemia at 64.8 mg/lwith conjugated bilirubin at 44mg/l and unconjugated bilirubin at 35.6 mg/l;gamma glutamyl transferaseat 88 IU/l; and alkaline phosphatase at 90 IU/l.

Lumbar puncture was performed and was sterile along with other bacteriological samples (protected distal bronchial sampling, cytobacteriological examination of urine, and blood cultures).

Interrogation with the patient's family found out the notion of stay in an endemic area [Ivory Coast] for 3 months. A thick blood smear was then performed, showing the presence of plasmodium falciparum trophozoites, following which the patient was put on Artesunate.

The evolution was favorable,and the patient was extubated without neurological complications. However, three days later, the patient presented psychiatric disorders mainly auditory and visual hallucinations with persecutory delusions. The patient was transferred to the infectious diseases department two weeks later

**Discussion**

Malaria is still one of the leading infectious causes of death in endemic countries; with approximately 429,000 deaths in 2015.[1]Its prevalence has increased markedly in recent years, especially in poor countries. Neuromalaria, caused by the sequestration of parasitized red blood cells within deep cerebral microvessels, is accompanied by magnetic resonance imaging (MRI) abnormalities in 80% of cases.

In severe cerebral malaria, pathological studies have revealed the existence of vascular and perivascular lesions.[4,5,8]These lesions sit electively in the cerebral white matter (corpus callosum, internal capsule, and semioval center) and more incidentally within cerebral cortex.Involvement of the cerebellum is usual (medium cerebellar peduncles, central white matter, lamellae, cortex).

More rarely, cases of cerebellar syndromes occurring well after a simple healed malaria attack have been described.[6, 7]Cerebral edema was found in around 63% of cases.[3]Ischemic, hemorrhagic, and thalamic lesions, as well as lesions of the white matter, and non-specific lesions were reported as well. In 2014, RJ Maude et al.[4] in a prospective study including 43 adults, 72% of whom were in a coma, showed abnormalities on MRI in 79% of cases. The abnormalities were in 51% of cases a moderate or diffuse cerebral edema, most often non-vasogenic, and without increased intracranial pressure. FLAIR hypersignals were also found in 26% of cases.[4]

In imported neuromalaria, cerebral edema can be so severe thatcan rapidly lead to death.CT scan or MRI often show a moderate increase in brain

volume which would be related to a rise in cerebral blood volume related to parasitic sequestration within cerebral microvessels 9.

In our case, the patient had presented diffuse cerebral edema in the initial cerebral CT scan while cerebral MRI showed a hypersignal ofthe corpus callosum which agrees with data from the published literature.

The prognosis of neuromalariahinges on neurological sequelae with about 11 to 25% of sequelae (spasticity, ataxia, hemiplegia, language disorders, blindness, cognitive disorders, epilepsy). In the SEAQUAMAT study, which included 1,461 patients including 202 children, only 0.67% of patients were discharged from hospital with neurological sequelae (psychosis, memory impairment, cerebellar ataxia, hemiplegia, extrapyramidal rigidity).[8]

Classified among the sequelae, the post-malaria neurological syndrome (PMNS) ismore commonly described in adults but with still uncertain pathophysiology. PMNS is found at around 1.2/1000.[10]It corresponds to the appearance of neurological or psychiatric symptoms occurring within two months of treated P. falciparum infection. PMNS is different from an authentic neurological sequela, which may alsocomplicate the evolution of severe neuromalaria but without a free interval. The evolution of our patient was favorable but with the appearance of psychiatric disorders mainly hallucinations and persecutory delusions.

**Conclusion**

In non-endemic countries such as Morocco, mortality from severe imported malaria remains as high as 10%. To reduce mortality, it would be of paramount to improve prophylactic measuresalong with optimization of management strategies. Finally, it would also be critical to stress the importance of chemoprophylaxis in case of a stay in an endemic country.

**References**

1. *World Health Organization. World malaria report 2016. Geneva: http:\www.who.int\malaria\publication\worldmalariareport2016\report\en*
2. Warrell, D.A. (1987) "Pathophysiology of severe falciparum malaria in man," *Parasitology*, 94(S1). Available at: https://doi.org/10.1017/s0031182000085826.
3. Mohanty, S. *et al.* (2011) *Brain swelling and mannitol therapy in adult cerebral malaria: A randomized trial*, *OUP Academic*. Oxford University Press. Available at: https://academic.oup.com/cid/article/53/4/349/445837 (Accessed: April 27, 2023).
4. Maude, R.J. *et al.* (2014) "Magnetic resonance imaging of the brain in adults with severe falciparum malaria," *Malaria Journal*, 13(1). Available at: https://doi.org/10.1186/1475-2875-13-177.
5. Seydel, K.B. *et al.* (2015) "Brain swelling and death in children with cerebral malaria," *New England Journal of Medicine*, 372(12), pp. 1126–1137. Available at: https://doi.org/10.1056/nejmoa1400116.
6. *Laurent V et al. Cerebral malaria during severe imported malaria in adults: clinical features, prognostic indices and brain imaging. Abstr. P 568, ICAAC 2012, San Francisco*
7. Greenwood, B., Marsh, K. and Snow, R. (1991) "Why do some African children develop severe malaria?," *Parasitology Today*, 7(10), pp. 277–281. Available at: https://doi.org/10.1016/0169-4758(91)90096-7.
8. Grau, G.E. and Lou, J. (1993) "TNF in vascular pathology: The importance of platelet-endothelium interactions," *Research in Immunology*, 144(5), pp. 355–363. Available at: https://doi.org/10.1016/s0923-2494(93)80080-i.
9. Looareesuwan, S. *et al.* (1995) "Magnetic resonance imaging of the brain in patients with cerebral malaria," *Clinical Infectious Diseases*, 21(2), pp. 300–309. Available at: https://doi.org/10.1093/clinids/21.2.300.
10. Piguet, P.F. *et al.* (2000) "Delayed mortality and attenuated thrombocytopenia associated with severe malaria in urokinase- and urokinase receptor-deficient mice," *Infection and Immunity*, 68(7), pp. 3822–3829. Available at: https://doi.org/10.1128/iai.68.7.3822-3829.2000.