

Cerebral Malaria – Causes and Prevention

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Cerebral malaria is that the most severe pathology caused by the malaria parasite, *Plasmodium falciparum*. The pathogenic mechanisms resulting in cerebral malaria are still poorly defined as studies are hampered by limited accessibility to human tissues. Nevertheless, histopathology of post-mortem human tissues and mouse models of cerebral malaria have indicated involvement of the barrier in cerebral malaria. In contrast to viruses and bacteria, malaria parasites don't infiltrate and infect the brain parenchyma. Instead, rupture of the barrier occurs and should cause hemorrhages leading to neurological alterations.

Causes of neurological manifestations in malaria

- High-grade fever alone can produce impairment of consciousness, febrile convulsions (in children) and psychosis. These manifestations subside with the decrease within the blood heat. Such cases and patients with unimpaired consciousness after seizures tend to possess good prognosis.
- Antimalarial drugs like chloroquine, quinine, mefloquine and halofantrine can also cause altered behaviour, convulsions, hallucinations and even psychosis. Absence of high-grade fever and of falciparum parasitemia may suggest such an opportunity.
- Hypoglycemia, either thanks to severe malaria or thanks to drugs like quinine, can also present with similar manifestations. Hypoglycemia is more common in pregnancy. It's going to be worthwhile considering this possibility altogether cases and to administer 25% to 50% dextrose intravenously.
- Hyponatremia, most frequently within the elderly and caused by repeated vomiting, is another important cause for neurological manifestations.
- Severe anaemia and hypoxemia also can cause cerebral dysfunction, particularly in children.
- There might be other causes for neurological dysfunction in patients with malaria like vascular disease, other neurological

infections and diseases. Focal neurological deficits, neck rigidity, photophobia, papilloedema and neurological sequelae are very rare in falciparum malaria and such an image would therefore suggest these other possibilities.

- A strict definition of cerebral malaria has been recommended for sake of clarity and this needs the presence of unarousable coma, exclusion of other encephalopathies and confirmation of *P. falciparum* infection. This needs the presence of *P. falciparum* parasitemia and therefore the patient to be unrousable with a Glasgow Coma Scale score of 9 or less, and other causes (e.g. hypoglycemia, bacterial meningitis and viral encephalitis) ruled out. To differentiate cerebral malaria from transient postictal coma, unconsciousness should persist for a minimum of 30 min after a convulsion. The deeper the coma, the more severe is that the prognosis. If necessary, a spinal puncture should be performed to rule out bacterial meningitis. However, all patients with *P. falciparum* malaria with neurological manifestations of any degree should be treated as cases of cerebral malaria.

Prevention

The preventive measures like the control of the vector or the plan to break the life cycle of the parasite, globally utilized in the tropics are widely known. So far, the best failure within the fight against malaria is resistance and therefore the lack of an efficient vaccine. Resistances are thanks to genetic mutations that cause alterations of the targets where the drugs act. The present approach to the event of a vaccine is predicated on the utilization of recombinant proteins or the entire attenuated organism. The vaccines are designed for several stages of the life cycle of the parasite: the pre-erythrocyte for the prevention of infection, the blood phase for the prevention of clinical disease and therefore the vaccines within the sexual phase to dam transmission. The foremost advanced candidate vaccine for *P. falciparum* is in phase III clinical trial efficacy trials.

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