

Post-Malaria Neurological Syndrome in a 10-Year-Old Girl: First Case Report from Oman with Review of the Literature

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Abstract

Introduction: Post-malaria neurological syndrome (PMNS) is a rare self-limiting neurological complication occurring after resolution from malarial infection; it is usually associated with, but not limited to, severe falciparum malaria. It is characterized by neuropsychiatric manifestations including mild neurological deficit to severe encephalopathy, with development of three main syndromes: delayed cerebellar syndrome, acute demyelinating polyneuropathy (GBS) and acute disseminated encephalopathy (ADEMs). We report a case of PMNS in a 10-year-old female child, and review the disease entity. **Method:** We searched PMNS-related journal articles and case reports in the English literature, using PubMed and Google search engines. A total of forty-nine cases meeting the diagnostic criteria of PMNS were selected in this review. **Conclusion:** PMNS is a rare complication of severe malaria that might be underreported, with variable presentation. Most cases are self-limited and would resolve within two months of clearance of parasitemia. There are few cases which develop severe manifestations and might benefit from steroid therapy.

Keywords

Malaria, Post-Malarial Neurological Syndrome

1. Case Report

A 10-year-old Sudanese girl, previously healthy, with normal developmental milestones and cognition, presents with acute onset neuropsychiatric manifestations for the past 3 days before her presentation. She developed mutism, generalized weakness in which she was unable to stand or walk, and drowsiness. She had no hallucinations, illusions, or delusions. No mood swings or sleep cycle disturb-

ances. She had no facial deviation, ocular abnormalities or vision changes, seizures, feeding or swallowing difficulties, or headache.

She returned from Sudan 2 weeks prior, and did not receive any malarial prophylaxis prior to travel. After 2 weeks, she developed a febrile illness of high-grade fever reaching up to 40, and was treated with antibiotics for presumed tonsillitis. However, her symptoms progressed to myalgia, nausea, vomiting, and continued to spike high grade fever. Clinical examination at that time revealed dehydration, no jaundice or pallor, and no hepatosplenomegaly. Neurological examination showed that she was drowsy and encephalopathic with Glasgow Coma Scale (GCS) (11/15), muscle tenderness, inability to stand, sit, walk, or talk with power of 4+, normal tone and reflexes, and no cerebellar signs which is going with severe malaria (as she was having prostration). Rest of examination was normal. She maintained normal blood sugar with no acidosis, and did not develop hypotension. Investigations during that febrile phase revealed anemia 7 g/dL, normal white blood cell counts of $5.5 \times 10^9/L$, thrombocytopenia $76 \times 10^9/L$, and high c-reactive protein (CRP) 72 mg/L. Peripheral smear showed plasmodium falciparum with parasitemia density of 4%. Liver function test showed transaminitis of alanine aminotransferase (ALT) at 72 IU/L, but normal renal function test. She was started initially on oral anti-malarial treatment with artemether and lumefantrine for 3 days and then shifted to intravenous artesunate when she developed neurological manifestations. After she finished her malaria treatment, she remained asymptomatic and was discharged home.

At her second presentation, she was drowsy with GCS 12/15, mute, but oriented and responded to questions with signs. Power 4+, and still unable to stand or sit; normal tone and reflexes. No signs of neurological deficit or meningeal irritation. Rest of her systemic examination was unremarkable apart from fever spikes every 4 hours initially.

Repeated test of peripheral smear showed Plasmodium falciparum gametocytes only with no ring stage (asexual parasites were not seen), hemoglobin 8.5 g/dL, normal WBC of $9.8 \times 10^9/L$, and normal platelets $440 \times 10^9/L$, normal renal and liver function test. Hemolytic markers were elevated including reticulocytes of 8.5%, and high lactate dehydrogenase (LDH) of 453 IU/L. Her CRP was elevated of 121 mg/L. Head CT scan was done to look for any intracranial collections which was reported as normal study. She was initially started on IV artesunate, ceftriaxone and acyclovir. Artesunate was stopped later with negative blood smear of active malaria.

Further investigations were done to look for other possible diagnoses like encephalitis/autoimmune disseminated encephalomyelitis (ADEM). Hence, cerebrospinal fluid (CSF) analysis was done which showed leukocytosis of 16 cells/uL, predominantly lymphocytes 90%, normal biochemistry (protein 0.17 g/L, glucose 3.5 mmol/L), negative gram stain for bacterial organisms, microbiological culture, bacterial polymerase chain reaction (PCR), and viral multiplex in the CSF. Magnetic resonance imaging was also done to look for other differentials

including vasculitis or ischemic changes which was normal. In view of those results, antibiotics and acyclovir were stopped. She showed gradual improvement on symptomatic treatment and physiotherapy (she was able to stand and take few steps alone, started talking in less than 2 weeks). She was discharged home with plan to continue her physiotherapy in the regional hospital. Subsequent outpatient follow-up showed further improvement and returned to her baseline activities.

2. Discussion

PMNS is generally self-limited condition which follows recovery from severe *Plasmodium falciparum*, with a latency period reaching up to 100 days [1] [2]. However, there are rare cases which develop post plasmodium vivax, in which two cases are reported in literature from India and adult case [3] [4]. The incidence of development of PMNS is 0.7 and 1.8 per 1000 cases, which is more common in patients with severe infections when compared with uncomplicated falciparum infection with a relative risk of 299 (40 to 2223) as per Mai *et al.* [5]. Another reported risk factor is the use of mefloquine as described in the later study, which was a randomized control trial in which 4.4% of the patients receiving mefloquine developed PMNS compared to 0.5% in patients who received quinine which was a significant value $p = 0.012$). In order to make the diagnosis of PMNS the following criteria must be fulfilled: recent malarial infection with aparasitemia on blood film, and full recovery from neuro psychiatric symptoms within two months of acute illness [5]. Our case fulfilled this criteria for diagnosis as the patient developed acute malarial infection evident on the blood smear, and then developed neurological symptoms within two months from the illness (after it has been cleared from blood smear; no ring stage was seen). There are several neurological syndromes that can occur following complete recovery including: delayed cerebellar ataxia (DCA), acute inflammatory demyelinating polyneuropathy (AIDP) and acute disseminated encephalomyelitis (ADEM) [6]. Other neurological manifestations include seizures, myelopathies, peripheral neuropathies, myopathies extrapyramidal syndromes, intracranial hemorrhage, and less commonly reversible cerebral vasoconstriction syndrome (RCVS) and posterior reversible encephalopathy syndrome (PRES) [7]. As per literature review the most common presentation were confusion (52%), seizure (33%), speech abnormalities (19%), and tremor (17%) [7].

The postulated pathogenesis is not understood but there are few hypotheses described in literature which are: molecular mimicry, structural brain changes after the infection, cerebral micro-vasculature obstruction from parasitized red blood cells causing hypoxemia, from coinfection with viral illness resulting in encephalitis, adverse drug reaction resulting in neurotoxicity, blood brain barrier disruption [8]-[10]. In one study conducted by Schnorf *et al.*, they found that 1 patient's CSF resulted for positive IgG antibodies against varicella zoster virus, which could explain the pathophysiology for the disease developed [8]. However,

those serological results could be falsely positive, since *P.falciparum* can release different antibodies due to polyclonal B-cell activation in a mechanism which is not understood yet [11] [12].

Around 60% to 77% of the cases with PMNS had CSF analysis showed increased opening pressure, lymphocytic pleocytosis, and elevated protein; in which our case showed presence of lymphocytes predominantly [13]. On the other hand, some cases will have a completely normal CSF analysis. In addition, some reported cases will have high CSF inflammatory markers such as TNF, IL-2, and IL-6 which supports that the pathogenesis is inflammatory, especially with trending down of the levels post steroid therapy [10] [14]. In our case neuroimaging was normal, which is the most common finding, and only less than 50% of the patients will have abnormal findings and this is usually seen in patients with ADEM like presentation which they will have bilateral cerebral white matter lesions [15] [16]. Those changes seen in images may persist for a duration which is unknown, and location of lesions does not necessarily correlate with the clinical picture (for instance having ataxia does not necessitate presence of cerebellar lesions) [17].

There is no specific standardized treatment for PMNS, however there are couple of studies suggesting the use of steroids and its benefits. In studies reported by Hsieh *et al.* and Schnorf *et al.*, patients were having worsening of neurological symptoms until steroids were initiated which resulted in a dramatic response and recovery within few days [10] [18]. In both the cases reported, the patients had severe form of PMNS resembling as encephalopathy, and the other with myoclonus and postural tremor, which started to improve after 24 hours of initiating steroids. Another reported case by Panda *et al.* of a 12-year-old girl manifesting as neuropsychiatric symptoms with normal MRI findings, received IV methylprednisolone for 5 days with great improvement in symptoms, and returned to her normal functional status after one month with no resolution of symptoms [4]. There are 55 cases reported that received corticosteroids as part of treatment, in which all of them showed clinical improvement (2 of them had gradual recovery while the rest improved within few days). 27 cases (49%) of those received oral prednisolone and the rest received IV methylprednisolone for 3 - 5 days [13] [19].

3. Conclusion

In conclusion, PMNS is rare complication that develops after documented malaria, with no clear pathogenesis explaining its development. Usually, it resolves completely with no neurological deficits and no specific treatment. However, severe cases, especially those with encephalopathy, will require a trial of corticosteroids, with dramatic recovery afterwards.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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