

Unilateral Facial Nerve Palsy LMN Type: An Unusual Complication of *P. vivax* Infection

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Abstract

A case is reported of *P. vivax* infection with unilateral Facial nerve palsy of Lower Motor Neuron (LMN) type, an unusual complication *P. vivax*. Temporary demyelination due to ischemia and immunological injury is the possible explanation.

Keywords: Facial nerve palsy, *P. vivax*

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Introduction

Malaria is most important tropical parasitic disease, affecting about 247 million people each year among the 3.3 billion people at risk, resulting in nearly a million deaths mostly children under age five years.¹ Typically, uncomplicated malaria present as an undifferentiated febrile illness. However, besides cerebral malaria neurological complications are known to occur after recovery from malaria, with no parasitaemia, usually following *Plasmodium falciparum* malaria and occurring after an interval of 2 days to 2 months. The syndromes are an acute disseminated encephalopathy, known as post-malaria neurological syndrome (PMNS); a delayed cerebellar syndrome; and an acute idiopathic demyelinating polyneuropathy (AIDP). In 1994, Nguyen described a post-malaria neurological syndrome (PMNS) in 1.8% of the patients after severe falciparum infection.² Most cases described in the literature occurred in adults living in endemic area for Malaria, mainly in Asia & Africa, but additional cases were described in non-immune hosts (93%) who travelled to endemic countries without taking prophylaxis.³ The

literature on *P. falciparum* as a cause of seventh cranial N, Palsy are scanty. The most frequent cause of Facial Nerve Palsy is Bell's palsy, in this condition no specific cause can be ascertained. The diagnosis is therefore made having excluded reasonable possible causes.^{4,5} Sim et al., reported facial diplegia in 20-year-old boy infected with *P. vivax* in Korea, while Dugue et al., published bilateral 6th cranial nerve Palsy caused by *P. falciparum* in a 14-year-old caucasian living in the Democratic Republic of Congo.^{6,7}

Cranial Nerve Palsy resulting from *P. vivax* infection has been scanty documented. This report, therefore, aims at attracting clinicians especially those working in malaria-endemic areas on this rare and unusual complication of malaria.

Case Report

A 63 years old male presented with the history of fever for last five days. Two days after the onset of fever patient noticed deviation of angle of mouth towards the left side. There was no antecedent head injury, convulsion, altered sensorium and any feature of respiration tract infection. The patient was not a known case of diabetes, hypertension and also there was no history of ear discharge, rashes or associated hearing and speech impairment. Clinical examination revealed fully conscious patient with normal vital signs except axillary temp of 101.0F.

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On neurological examination right sided Facial Nerve Palsy of LMN type was noted with no other neurological deficit. Systemic examination was normal. His blood smear and MP Antigen test was positive for *P. vivax*. MRI brain revealed no abnormality. CSF analysis showed normal pressure with normal cytology and biochemistry. CSF was negative for HSV serology.

Laboratory tests showed deranged LFTs - SGOT-286.4 IU/L, SGPT-228 IU/L, with normal bilirubin & albumin level. His renal function tests were normal with normal electrolyte level. Routine urine and blood culture yielded no growth. The retroviral screening was negative. He was admitted and treated with the Artemisinin combination therapy (ACT) (IV Artesunate and oral Doxycycline) and other supportive treatment. Fever subsided within 48 hours, and neurological recovery of facial nerve started after five days of the ACT. The patient was not given steroids during his stay in the hospital. Patient discharged after seven days of admission in an afebrile state with complete recovery of facial weakness.

Discussion

The mechanism underlying facial nerve palsy in Malaria caused by *P. vivax* is unclear. It has been postulated that malaria parasite may damage the peripheral nerves by the vascular occlusion, thus causing anoxic stagnation in the vasa nervosa, which may lead to temporary demyelination and recovery after the disappearance of the parasitemia and establishment of normal blood flow in vasa nervosa.⁶ In malaria, asexual stage infections are accompanied by the release of cytokines and other immunological mediators that may mimic Guillain-Barre-syndrome.^{7,8} It is likely that these immunological mediators may have caused demyelination in the index patient giving rise to Guillain-Barre like syndrome affecting only the facial nerve. However, no serological test was done for the purpose of isolating antibodies against neuronal tissues. It may also be due to the local effect of the parasites on the facial nerve since the paralysis is the lower motor neuron type. This assertion is subject to future studies to determine vulnerable areas of the facial nerve that can be affected by *P. vivax*. In the index patient recovery of facial paralysis was complete on discharge after seven days of admission, which is a sharp contrast to Bell's palsy where recovery usually starts in

three weeks of onset of illness with complete recovery occurring by three to six months.⁵ Meningitis, diabetes mellitus and intracranial pathology were excluded since CSF examination, CECT Brain, MRI Brain showed no abnormal features. Routine Biochemistry was normal except for deranged LFTs, which recovered with recovery from illness. The only evidence of the cause of fever and facial nerve paralysis in this patient is *P. vivax* infection.

Conclusion

Clinician practicing in malaria endemic area should be aware of facial nerve palsy LMN type as a rare and unusual neurological manifestation of malaria not only with *P. falciparum* but also with *P. vivax* infection.

Competing Interests

The authors declare that they have no competing interests.

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