Case Reports

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Post-malaria neurological syndrome – a case of bilateral facial palsy after *Plasmodium vivax* malaria

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Background: Post-malaria neurological syndrome (PMNS) is defined as the acute onset of neurological or neuropsychiatric syndrome in a patient who had recently recovered from malaria and have negative blood film at the time of onset of neurological symptoms. This, therefore, distinguishes it from cerebral malaria, which occurs during the period of parasitaemia. The time from eradication of systemic parasitaemia to the development of this syndrome can be up to nine weeks[1]. The prevalence of PMNS in patients with malaria is 0.12% and is 300 times more common in patients with severe malaria in comparison to uncomplicated malaria[1]. The reported clinical features include generalised convulsions, acute confusional state, psychosis, tremors, cerebellar ataxia, motor aphasia, and generalised myoclonus[2,3]. Most of these patients made complete recovery without specific treatment. Almost all reported PMNS cases are associated with episode of *P. falciparum* malaria. We report a case of bilateral facial palsy developing on 14th day of afebrile period after successful treatment of vivax malaria and had complete recovery in next four weeks.

Case report: A 55 year-old male (RG) was admitted in the hospital with complaints of fever, nausea and vomiting for last four days. Fever was associated with chills and rigors, and occurring on alternate days. Patient was conscious, oriented and there was no icterus, pallor, lymphadenopathy or cynosis. Skin was of normal texture and other systems including cardiovascular, respiratory, nervous system were normal. Peripheral blood film (PBF) for malarial parasite was positive for *P. vivax* (Fig. 1), which was

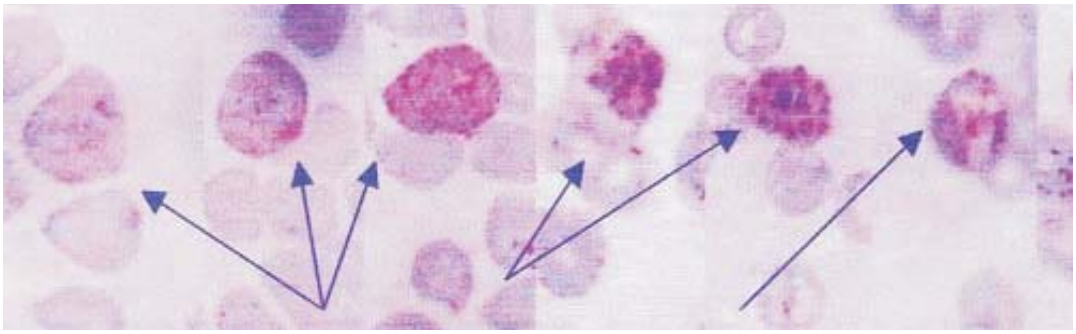

Fig. 1: Peripheral blood examination showing evidence of *P. vivax* infection

also confirmed by rapid diagnostic test (pLDH antigen for vivax was positive and pLDH as well as HRP2 for falciparum was negative). Parasite density was 15000/mm^3. Other relevant investigations were within normal limits. Patient was treated with oral chloroquine in the dose of 600 mg followed by 300 mg after six hours and then 12 hourly so as to make a total of 1500 mg. The patient became afebrile after two days and PBF done at the time of discharge was showing no evidence of parasites. On Day 14 of afebrile period, he was admitted again in the hospital with complaints of drooling of saliva from angle of mouth, alteration in speech and taste and inability to close his eyes during sleep. He was fully conscious and well-oriented. The detailed neurological examination revealed bilateral infra-nuclear facial palsy, absence of wrinkles of forehead, loss of taste sensation on anterior 2/3 of tongue and absent corneal reflex. All relevant investigations were done to rule out other locally prevalent infectious diseases as well as metabolic and neurological disorders. No specific treatment was given and the patient made complete recovery within four weeks.

PMNS occurs up to nine weeks after complete recovery from *P. falciparum* malaria[1]. Only two case reports are available in literature about PMNS after vivax malaria in which one case was of acute inflammatory demyelinating polyneuropathy[4] and another case was of acute disseminated encephalomyelitis like picture[5]. This case can also be presumed to be a definite case of vivax malaria as diagnosed by PBF examination which showed only vivax infection and rapid diagnostic test showing positive evidence of pLDH antigen of *P. vivax* and was negative for pLDH as well as HRP2 antigen of *P. falciparum*. Even if we presume that *P. falciparum* may not be seen in PBF in a patient of mixed infection, it could have been picked up by the presence of HRP-2 antigen in rapid diagnostic test (RDT). The presentation with neurological deficit after two weeks of complete recovery from confirmed attack of malaria is a strong evidence of the diagnosis of PMNS. However, we had tried to rule out other possibilities by appropriate tests.

The etiology of PMNS remains unclear and a wide range of neurological manifestations have been reported. In cerebral malaria, sequestration of parasitised red blood cell within cerebral vessels can result in local ischemic damage[6] but this mechanism cannot be implicated in PMNS, where by definition no parasitised red blood cells are present at the time of neurological involvement. There are no reports in literature about bilateral facial palsy in association with the use of chloroquine. Incidentally, *P. vivax* infection in this region had also been associated with severe manifestations[7]. The report of PMNS from the same region may be of great significance in the term of changing behaviour of organism in this part of world. These two observations (severe vivax malaria and PMNS after vivax infection) from the same geographical region is a matter of great concern.

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