Case Report

Reversible myelopathy in *Plasmodium vivax* malaria: Report of a case and review of literature

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Malaria is a major health problem, accounting for up to 500 million febrile illnesses and several million deaths annually¹–². Malaria is endemic in India and continues to account for almost a 1000 deaths per year since 2002. Almost all of the mortality is accountable to *Plasmodium falciparum* infection³. *Vivax* malaria is generally thought as the “benign tertian malaria” and is unaccompanied by any adverse complications. Recently few complicated cases are being reported and the designation of benign malaria is being challenged⁴. Two recent retrospective analyses from India highlight this picture⁴–⁵. There are few reports of neurological complications in mono-infected *vivax* malaria patients⁶. We report a case of *P. vivax* infection in a patient who presented with reversible anterior spinal cord syndrome with MRI evidence of myelitis.

Case report

A 35 yr-old male patient was admitted to the SSKM Hospital, Kolkata, India on 12 August 2011, with high grade intermittent fever for two days and weakness in both the lower limbs for one day. He developed urinary incontinence and loss of pain sensation below mid abdomen. He did not report any weakness of upper limbs, res-

![Fig. 1: T2 weighted MRI of dorsal spine showing hyperintensity at the level of T6–T7 spine.](image-url)
piratory embarrassment or any positive sensory symp-
toms. There was no history of recent immunization or any history suggestive of recent upper respiratory tract infection or skin infection. The patient was haemody-
namically stable and was oriented to time, place and per-
son. There was no evidence of meningeal involvement.
Neurological examination revealed: increased muscle tone with difficulties in passive movement and 2/5 grade muscle power in both lower limbs; loss of pain, tempera-
ture and pressure (mild degree) sensations below the umbilicus suggestive of lateral and ventral spinothalamic tract involvement. Fine touch, vibration and position
senses were normal in all the dermatomes. Lower abdomi-
nal and cremasteric reflexes were lost and plantar reflexes
were bilaterally extensor. Deep tendon reflexes were brisk
in the knees and the ankles. Examination of cranial nerve
did not reveal any abnormality. Complete blood counts,
serum electrolytes, liver and renal function tests were nor-
mal. Cerebrospinal fluid (CSF) had 8 cells/ml (all lymphocytes), protein 64 mg/dl, and glucose 68 mg/dl. CSF was negative for oligoclonal bands and his IgG index was
0.01. Peripheral blood smear examination showed tro-
phozoites of *P. vivax*. Peripheral blood thick and thin smear examination and *Plasmodium* LDH test (done twice) did not show any parasitological evidence for *P. falci-
parum* infection.

His spinal MRI showed non-enhancing, long TR
hyperintensity at the level of T6 – T7 spine suggestive of
myelitis (Fig. 1). MRI of brain was normal. Serology for
HIV-I and II, Herpes simplex virus and enteroviruses were
negative. The patient was started a course of oral chloro-
quine followed by primaquine (30 mg once daily for 14
days). From the Day 4 of initiation of chloroquine therapy
he gradually started to regain his muscle power. By the
Day 6, his bladder complaint was diminished. After one
month follow-up he has residual lower limb hypertonia
and brisk jerks without any motor or sensory deficit.

**DISCUSSION**

Our patient presented with acute onset of fever with
involvement of bilateral pyramidal, and anterolateral
spinothalamic tracts without any evidence of posterior
column involvement. Demonstration of vivax parasit-
aemia and antigenemia clinched the diagnosis with fur-
ther aid from spinal MRI. Normal MRI of brain helped to
exclude multiple sclerosis. The patient suffered from vivax malaria, complicated with reversible acute myelitis showing prompt response to antimalarial therapy.

*Plasmodium vivax* malaria usually shows a benign
clinical course unlike *P. falciparum*, which often presents
features of organ dysfunction. In falciparum malaria, the *P. falciparum* erythrocyte membrane protein-1 (PfEMP-1) is important in pathogenesis. This adhesion
molecule is anchored at RBC membrane and interacts with
a variety of host adhesion molecules causing sequestra-
tion of parasites in cerebral microcirculation with result-
ant hypoperfusion. Cytokines and chemokines like TNF,
interleukins also play major role in endothelial dysfunc-
tion. All these factors trigger widespread activation of
inflammatory process and endothelial damage. *Plas-
modium vivax* is devoid of PfEMP-1 and the parasite
burden is usually less than *P. falciparum*. This could
possibly explain lower rate of complication in *P. vivax*
malaria.

This scenario is changing rapidly with increasing
number of complications being reported in *P. vivax*
malaria. In 2009, Sharma and Khanduri from Delhi, India
demonstrated that thrombocytopenia, hepatic dysfunction,
renal damage, positive direct Coomb’s test and acute res-
piratory distress syndrome (ARDS) were associated with
vivax malaria, but none of the 221 vivax malaria patients
developed any neurological complications. In another
study in India among 50 vivax malaria patients, thromb-
ocytopenia was the commonest complication followed by
liver dysfunction (62%), respiratory involvement (28%),
renal impairment (22%), shock (16%), severe anaemia
(6%), and cerebral malaria in only two cases.

Neurological complications of *P. vivax* malaria are
extremely rare, though these are increasingly being re-
ported. The most common complication is cerebral ma-
laria with the hallmark of encephalopathy. Some of the
reports highlight the presence of meningeal signs in ceb-
ral malaria complicating *P. vivax* infection in the ab-
sence of bacterial or other pathogens. Other neurologi-
cal complications reported in vivax malaria are, periodic
paralysis, facial diplegia, aphasia with hemiparesis and
Guillain-Barre syndrome.

There are two reports of acute disseminated enceph-
alomylitis with vivax malaria. The first one reported in a
24 yr-old Japanese male person with neurological distur-
bances two weeks after complete recovery from *P. vivax*
infection. MRI of the brain showed multiple high-intensity
spotty lesions in the left cerebral cortex and subcortex. In
2011, another group from Kolkata, India reported a case of
diabetic woman with mixed *P. falciparum* and *P. vivax*
infections. While recovering from the episode she developed
acute onset bladder retention followed by paraparesis. MRI
of spine showed T2 hyperintensities suggestive of resolv-
ing myelitis. MRI of the brain showed multifocal and
confluent areas of demyelination mostly involving the
corpus callosum and periventricular region.
Our case emphasizes the growing spectrum of neurological manifestations of vivax malaria. To the best of our knowledge, this is probably the first case report which shows reversible isolated spinal myelopathy with a clinical picture of anterior cord syndrome occurring as a complication of vivax malaria.

REFERENCES


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