

Original Research Paper

Plasmodium Vivax Cerebral Malaria Complicated With Venous Sinus Thrombosis In Gujarat

Dr. Kuntal H Jivani

Resident doctor, Department of Medicine, smt.nhl mmc, Ahmedabad

Malaria is one of the most prevalent parasitic infections in the world[1]. Four species of Plasmodium affect humans, but more recently Plasmodium knowlesi was described in Southeast Asia as a fifth species in primates that rarely affects men[2]. However, the most important species that contribute to increased morbidity and mortality in humans are Plasmodium falciparum (P. falciparum) and Plasmodium vivax (P. vivax)[1]. After P. falciparum, infection with P. vivax is the second most common cause of malaria in the world.It is difficult to treat P. vivax because of the hypnozoites and frequent relapses after initial management[1,3]. The previous old paradigm of P. vivax as "benign tertian malaria" has been challenged recently by reports of severe disease with complications such as acute respiratory distress syndrome, acute renal failure, severe thrombocytopenia, retinal hemorrhage, toxic shock, and cerebral malaria[4-10]. The purpose of this report is to describe a case of cerebral malaria as a complication of P. vivax infection

KEYWORDS

Cerebral malaria, vivax malaria, Plasmodium vivax, Venous sinus thrombosis

Case report

A 40 year old female from ambawadi,ahmedabad was admitted with one week complaints of fever,headache and nausea. On admission,she was drowsy but responding to deep painful stimulus. She was having neck rigidity and left sided focal seizures. With Glassgow coma scale of 9, she was transferred to intensive care unit.

Initial laboratories showed Hemoglobin 12.0 g/dl,total WBC count 9000/cumm with 78% polymorphs and 13% lymphocytes and platelet count of 3,19,000/cumm.Perpheral smear showed trophozoites and ring form of plasmodium vivax.Her urine routine examination showed dark coloured urine with 1-2 rbcs,2-4 pus cells and 8-10 epithelial cells.Urine protein and sugar was absent.Her serum creatinine was 0.79,urea 16,glucose 80,pottasium 3.5,sodium 134,ALT 33,Billirubin total 1.07 and direct 0.45.Patient tested negative for routine viral markers HIV,HbsAg and HCV RNA.Her prothrombine time was 15.7,INR 1.35 and aPTT 27.4 sec.

Her cerebrospinal fluid examination report was normal and brain computed tomography was normal but magnetic resonance imaging showed thrombosis in superior sagittal sinus. She was tested negative for antinuclear antibody by immunoflurescence and lupus anticoagulant.

Treatment was started with injectable artesunate and clindamycin under suspicion of mixed infection with p.falciperum. The patient showed recovery after treatment was started and peripheral smear was negative for parasites after treatment.



1-MRI Brain image of the patient showing superior sagittal sinus thrombosis

Discussion

Severe malaria is associated to P. falciparum and accounts for increased mortality worldwide^[1].P.vivax has been neglected in clinical studies of severe malaria and the Previous studies believed that P. vivax was unable to produce cytoadherence with microvascular sequestration, therefore, it was considered impossible to cause organ dysfunction like in P. falciparum malaria. Recently, sequestration of P. vivax in the pulmonary vasculature has been noted and it is believed that organic dysfunction can be generated by an inflammatory response amplified by the release of cytokines^[11,12].

However, there is little known about this presentation with P. vivax and its relation with individual immune status. Cerebral complications have been described in 45 patients, the majority in children from India and Pakistan. In 1921,Rossle described a lethal hemorrhage in the cerebellum of a 21 year old patient during a course of tertian malaria^[13]. In 1932, Bruetsch reported a 61 year old woman with fatal tertian malaria induced as treatment for syphilitic psychosis; at autopsy "numerous parasitized red blood cells and young plasmodia" were observed in brain tissue^[14]. Other complications mentioned are mainly seizures, altered consciousness reaching even coma, acute inflammatory demyelinating polyneuropathy and facial paralysis.

Cerebral malaria manifested by isolated bilateral sixth cranial nerve palsy is rare in adults but more commonly described in children^[15-20]. Krishnan et al. described three adults with P. falciparum malaria with fever, altered consciousness and focal neurological deficits (one of whom also had seizures); brain CT scan showed haemorrhagic infarction of the cerebral cortex and subcortical white matter with surrounding oedema suggestive of venous infarction in all of them; the diagnosis of cerebral venous thrombosis was missed in the first case and was detected only at autopsy; in the other two patients, superior sagittal sinus thrombosis was confirmed angiographically, one patient survived and the other died due to increased intracranial pressure, two of them had P. vivax co-infection^[21,22]. Another report by Sarkar and Bhattacharya shows three adult patients with P. vivax malaria complicated by seizures and symptoms of diffuse meningoencephalitis[10]. More recently, the same author presented the first case of P. vivax malaria with left thalamus haemorrhage which is an unusual site of bleeding even more in the absence of severe thrombocytopenia or disseminated intravascular coagulation^[23].

This clinical scenarios show how P. vivax infection could lead

to involvements other than the commonly described ones with different outcomes and should call our attention in order to rule out this co-infection when severe malaria appeared even if routine test shows P. falciparum; besides,we must watch closely patients with neurological findings because they could have sinus thrombosis.Although we do not clarify the cause of the superior sagittal sinus thrombosis in this patient and there were no clear consequences of this event, we believe that this information can be added to future studies to enable a better understanding of the disease.

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