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Cerebral Venous Sinus Thrombosis as a Rare Complication of Plasmodium Vivax Malaria

KEYWORDS: Cerebral venous sinus thrombosis, plasmodium vivax ,malaria.

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ABSTRACT *Cerebral venous sinus thrombosis(CVT) has been reported to be associated with many systemic illness and infection. Inspite of malaria being endemic in India cases of CVT in malaria is very rare. Common causes of altered sensorium/coma in malaria are hypoglycemia, cerebral malaria and electrolyte imbalance. Here we report a 28 year old male presenting with fever, seizures and focal neurological deficits. He was diagnosed as Plasmodium vivax malaria. On imaging of brain revealed evidence of raised intra cranial pressure and CVT. A hypercoagulable state resulting from severe malaria may be responsible for this potentially fatal complication. He was treated with antimalarials and anti-coagulants. He was responded well and was discharged in a stable condition.*

INTRODUCTION: malaria can cause various neurological symptoms like seizures, focal deficits, altered sensorium. It can also present with rare manifestations such as polyneuritis, psychiatric symptoms, Gullian Barre Syndrome and CVT [1].CVT is potentially fatal condition which if diagnosed early and treated favours good outcome. We report a case of CVT in a patient with Plasmodium vivax positive malaria.

CASE REPORT: A 28 year old previously healthy male presented to us with 3 days history of high grade, intermittent fever with chills and rigors. He was treated with antipyretics in a local hospital. On day 4 of his illness he developed 10-15 episodes of generalize tonic clonic seizures with each episode lasting for a minute or two. No history of trauma or significant drug intake was present .

On examination he was drowsy and arousable with a GC score of 8. No pallor or evidence of dehydration was present. His pupils were 2.5mm in size and sluggishly reacting to light. Cranial nerve examination was normal and there is no sensory deficit. Motor power was 0/5 on left side with deep tendon reflexes 2+, Babinski positive. There was no neck stiffness and cerebellar examination was normal. Other system examination was unremarkable.

On further investigating the patient rapid check for malaria antigen was positive for plasmodium vivax and blood smear examination revealed vivax malaria (figure 1). Laboratory studies showed hemoglobin 14gm%, total count 9700/cu.mm , platelets 120000/cu.mm. serum biluribin was 1.6mg/dl. Renal function tests and electrolytes were normal. Chest X-ray and urinalysis was normal. Screening for HIV, HBsAg, HCV were non reactive. Leptospiral antibody tests and Immuno flourescence for Typhus were negative. Coagulation profile revealed no abnormality. On Computed Tomography of Brain revealed hypo dense filling defect in superior saggital sinus and right transverse sinus suggestive of CVT (figure 2). With all the above workup he was diagnosed as a case of CVT in plasmodium vivax malaria.

He was treated with antimalarials, anticoagulants and antiepileptics for which he responded well. Supportive treatment and physiotherapy was advised and patient was discharged in a stable condition. He was under follow up and his weakness improved to 4/5. Tapering of antiepileptics was planned and stopped.

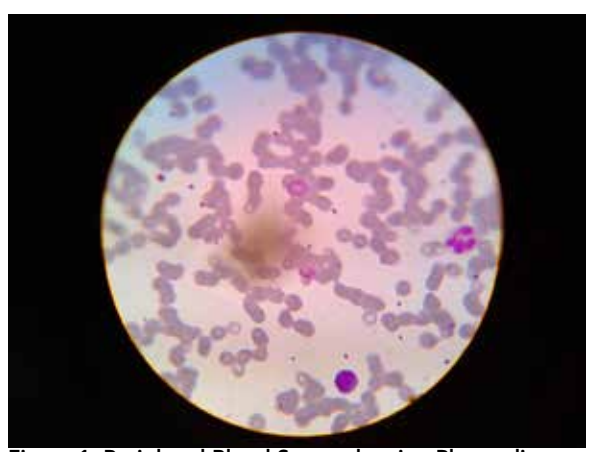
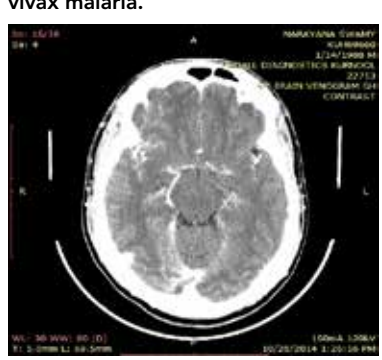


Figure 1: Peripheral Blood Smear showing Plasmodium vivax malaria.



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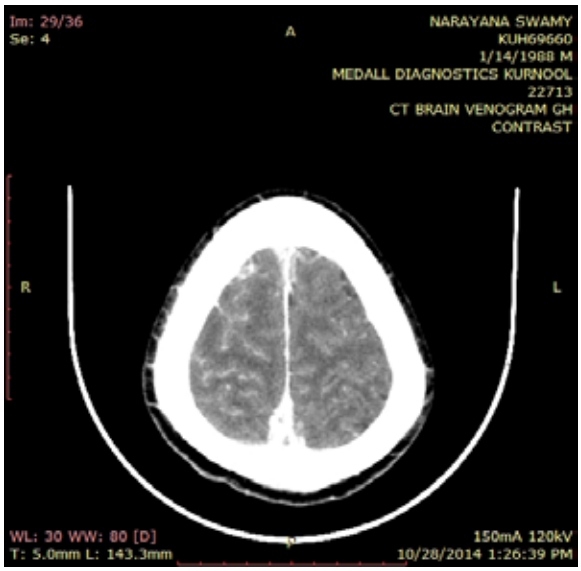

Figure 2: CTscan of Brain showing hypodense filling defect in the right transverse sinus and superior saggital sinus.

DISCUSSION:

Severe complications of malaria has been traditionally attributed to falciparum malaria or mixed infections. In recent times many cases of unusual complications has been associated with isolated infections of plasmodium vivax malaria.

Plasmodium vivax malaria can cause both sequestration and non sequestration related complications [2]. Clinicolaboratory profile of 6 patients of vivax malaria cerebral malaria was studied by Sachdev and Mohan[3], which varied in presentation from encephalopathy, convulsions, coma to focal deficits. The mechanism of cerebral venous thrombosis in a case of cerebral malaria remains unclear and is probably multi factorial, hypercoagulable states induced by severe malaria may be responsible for this condition as proposed by Krishnan et.al [4]. Altered phospolipid in malaria infected red blood cells causes increased Von Willibrand factor and other coagulable factors [5]. Tissue factor expression by the endothelium and amplification of coagulation cascade by infected red blood cells may be associated with procoagulant states in severe malaria according to work done by IVO MB Francishetti and others [6,7,8].

Thrombolytic and anti coagulant therapy in CVT remain important and should be judged in individual case. Randomised control trials demonstrated the benefit of anticoagulants and are generally recommended for the patients who has no other contraindications [9].

Complete recovery is expected in many patients with anti coagulant therapy. Our case responded well to anticoagulants.

All the potential risk factors for CVT like hypercoagulable states, dehydration, trauma, drug induced thrombophilias, systemic or local infections were ruled out in our case and so the cause of CVT was attributed to plasmodium vivax malaria.

CONCLUSION: Plasmodium vivax malaria is showing increasing tendency to cause complications which is not known to be associated with it, as reported from various malaria endemic areas. Further studies are required to know the exact mechanism causing such phenomenon. Clinicians must be aware of such unusual presentations which will help to reduce the morbidity and mortality associated with the disease.

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