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## **Neurological involvement associated with *Plasmodium vivax* malaria from Pakistan**

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### **Background:**

*Plasmodium vivax* is the most common species causing malaria outside Africa with approximately 13.8 million reported cases worldwide. More than 80% of *P. vivax* malaria cases are estimated to occur in Pakistan, India and Indonesia (WHO 2015). *P. vivax* is associated with increased morbidity but reduced mortality when compared to *Plasmodium falciparum*. Recently, studies from Indonesia, Papua New Guinea, Brazil, India, and Pakistan have shown that *P. vivax*, can independently prove fatal or result in severe morbidity. The most common complications of *vivax* malaria reported globally are anemia and non-cardiogenic pulmonary oedema/ acute respiratory distress syndrome. Other complications are severe thrombocytopenia,

pancytopenia, jaundice, splenic rupture, acute renal failure and rarely cerebral involvement.

Cerebral malaria is a known complication of *P. falciparum* with over 575,000 cases leading to neurological, behavioural and cognitive defects reported annually.<sup>(1)</sup> However, cerebral malaria due to *P. vivax* mono infection is rarely reported. Malaria remains an endemic disease in Pakistan with an estimated health care burden of 1.6 million cases annually with *P. vivax* accounting for 67% of reported cases. We report a severe *P. vivax* infection with cerebral involvement in a child.

### **Case Report**

A nine-year old boy presented to the emergency department of The Aga Khan University Hospital (AKUH) with a 3-day history of high grade fever accompanied by projectile vomiting and abnormal behaviour. A day prior to his admission he developed generalized tonic-clonic seizures associated with dizziness and unconsciousness. He had a coma score of 5/15 on presentation with a tachycardia (167/min), tachypnoea (70 breaths/min), oxygen saturation of 92%, blood pressure of 133/87 mm of Hg and a temperature of 38°C. There was hypertonia in all limbs. The rest of the physical examination was normal. The full blood count showed anaemia (Hb 94g/L), a leucocytosis ( $13.1 \times 10^6/\text{mL}$ , neutrophils 75% and monocytes 8%) and normal platelet count ( $304 \times 10^6/\text{mL}$ ). A cerebral CT scan showed oedema with increased intracranial pressure. Thick and thin peripheral smear with Giemsa stain was suggestive of *P. vivax* with 1-10 per 100 high power fields. Stained blood films were also independently reviewed by an external expert parasitologist who confirmed the original diagnosis. Polymerase chain reaction (PCR) and an immunochromatography test (ICT) definitely excluded *P. falciparum* infection. Prothrombin time and activated partial thrombin time were in the normal range, and the direct antiglobulin test was negative. Although no lumbar puncture was performed empirical therapy for bacterial and viral meningitis was initiated. Initially Vancomycin, Meropenem, Acyclovir, steroids and anticonvulsants were started in emergency. The patient was mechanically ventilated. He required vasopressor support with norepinephrine. Blood cultures and urine cultures were all negative, Antibiotics were

deescalated and stopped as blood film confirmed malarial parasite *P.vivax* and started with artemether lumefantrine. On the third day, he developed high urine output and hyponatremia secondary which was managed with fludrocortisone. On presentation his sodium levels were 145mmol/L (normal ranges 135-145 mmol/L) and dropped to 125mmol/L on third day. His sodium level was corrected to 135 in 72 hours and child gained consciousness. The GCS score improved to 15/15 and he was extubated on the sixth day of admission and discharged on the ninth day. Post-treatment microscopy revealed blood films negative for *P.vivax*.

## **Discussion**

*P. vivax* malaria has historically been termed, 'Benign Tertian Malaria' but it is increasingly felt that this is an erroneous label. Rossle in 1921 described a lethal cerebellar haemorrhage in a 21-year old soldier suffering a vivax infection (3). Patients presenting with cerebral symptoms such as acute febrile encephalopathy, raised intracranial pressure, intracerebral haemorrhage, hemiparesis, aphasia and seizures following vivax malaria have been reported previously (4, 5).

Several cases of cerebral malaria attributed to *P.vivax* infection have been reported in recent years, especially in Asia and South America. This may be due to an overall increase in *P. vivax* malarial infections. Most of these cases have been reported from India where *P. vivax* is the predominant species (6). Although the mechanism by which *P. vivax* causes cerebral malaria is not fully understood, a suggested pathogenesis is the sequestration of infected erythrocytes in cerebral microvascular beds(7).

In our case, the peripheral blood smears with Giemsa stain were repeatedly performed and parasitaemia remained low. Immuno-chromatography testing showed no trace of *P. falciparum*, and PCR helped confirm mono-infection by *P. vivax*. Advances in molecular and serodiagnosis of malaria parasites have increased the sensitivity and specificity, and so the substantial accuracy, of diagnosis of *P. vivax*. (8, 9)

Physicians must be aware of the aggressive pathology of severe *P. vivax* infections and institute prompt and accurate diagnosis in treatment. Furthermore, an increasing

incidence of severe malaria cases due to *P. vivax* are already placing a huge burden on an already strained healthcare system.

The study was approved by the AKUH ethical review committee (reference no. 3541-Path-ERC-15). Informed consent was obtained from the patient for the publication of this report.

The authors declare that they have no competing interests, and received no funding.

**Author contributions:** MAB, SAZ, YAK, MUH were responsible for clinical work, designed and planned the study. NKG performed Microscopy, PCR genotyping and interpretation. AAW and YAK, MUH, NKG prepared the initial manuscript. MAB and SAZ reviewed the final draft. All authors read and approved the final manuscript.

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