SERIES OF TWO CASE REPORTS ON COMPLICATED VIVAX MALARIA

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ABSTRACT

Plasmodium falciparum and P. vivax cause a significant majority of malaria infections in human beings. P. falciparum is responsible for the most severe form of malaria. Till now P. vivax was known to be associated with uncomplicated infections, however, over the last few years the trends have shown that P. vivax is no longer a benign disease (Dhanpat et al., 2009; Bruno et al., 2010).

Keywords: Plasmodium Vivax, Hepatitis, Diabetic Ketoacidosis

INTRODUCTION

Plasmodium falciparum is a known culprit of complicated malaria since long time. But now a days more and more cases of vivax malaria with abnormal features are increasing (Beg et al., 2002). Major severe P. vivax clinical syndromes documented include thrombocytopenia (Makkar et al., 2002; Rodriguez-Morales et al., 2005), cerebral malaria (Ozen et al., 2006), acute kidney injury (Das, 2008) hepatic and pulmonary dysfunctions (Tanios et al., 2001).

CASES

Case 2

A 45 year old male presented in the casualty of Santosh hospital with the history of fever from 5-6 days which was associated with chills and rigors, generalised bodyache, headache and yellowish discoloration of eyes but no rash.

He also had 2-3 episodes of vomiting. There was no significant past medical or surgical history and no history of any addiction as well.

The patient was conscious, oriented, icterus present. On examination the blood pressure was 100/70mm Hg, pulse 108 /min, respiratory rate 20/min and the patient had 103°F temperature. Clinical examination revealed no significant abnormality except for mild hepato-splenomegaly.

The investigations revealed:-Hb 12.5gm%, TLC 12800/mm³, DLC N84,L15,E1 platelet count was 20000/mm³,malarial parasite antigen positive for P. Vivax, dengue serology was negative, blood urea 36 mg%,S. creatinine 0.91mg%, blood glucose 86mg/dl, S.Bilirubin- total 4.17mg%, direct 2.30mg%, SGOT- 830U/L, SGPT- 1000U/L, alkaline phosphatase 230U/L, prothrombin time - 19sec with INR 1.7, S.Albumin 3.8gm%

Chest X ray and ECG were normal

USG whole abdomen revealed mild hepato-spleenomegaly

The patient was treated accordingly with i.v antimalarial (Artesunate) and supportive treatment with i.v fluids and antipyretics. The patient became asymptomatic in 3-5 days. The liver function test started reverting and improved within 10days and patient was discharged on day 10.

Case 2

A 55 years old female not a known diabetic or hypertensive presented in the casualty of Santosh Hospital with high grade fever since 7days which was associated with generalised weakness, bodyache, headache, pain in abdomen, loss of appetite, vomiting and loose motion. There was no significant medical or surgical history and no history of any addiction. The patient was conscious, oriented and had following vitals BP 110/70mmHg, pulse 110/min, Temperature 101°F. Respiratory rate 26/min. Systemic examination revealed no abnormality except for increased respiratory rate with kussmaul breathing and mild splenomegaly.
Case Report

Lab findings revealed: Hb - 8.0 gm/dl, TLC - 9600/mm³, DLC N05,L30,E03,M02, platelet count - 1.56 lac/mm³, RBS- 526mg/dl, FBS- 437mg/dl, HbA1c-15.2%, urine for ketones positive, malarial parasite antigen positive for P. Vivax, blood urea-80mg%, S.creatinine- 2.92mg%, S.bilirubin total 0.72mg%, direct 0.26mg%, S.Na+ 130 meq/l, S.K+ 6.29meq/l, SGOT- 44U/L, SGPT- 42U/L, Alkaline phosphatase-212U/L. Arterial blood gas analysis showed pH 6.9, PaO₂=90mmhg, HCO₃=18meq/l, PaCO₂=40mmhg, BE=-5
Chest X-ray and ECG was normal
USG whole abdomen revealed mild splenomegaly
The patient was treated accordingly with i.v antimalarial (Artesunate) and for diabetic ketoacidosis with regular insulin and i.v fluids. The patient gradually improved and became asymptomatic in 3-5 days with laboratory investigations reverting to normal in 10-15 days. Patient was discharged on day 15.

DISCUSSION

Malaria is still a leading cause of morbidity and mortality. According to World malaria report 2014 (WHO), 128 million malaria infections and 58400 deaths were reported in 2013. The majority of severe and fatal malaria is caused by P. falciparum. Severe complicated malaria is a well recognised feature of P. falciparum malaria which usually manifests as cerebral malaria, though occasionally with liver failure, thrombocytopenia and other complications. Hepatic involvement in malaria has largely shown severe infection with P. falciparum infection. P. vivax earlier termed as benign tertian malaria because of uncomplicated disease and was rarely fatal. P. vivax had been reported recently with symptoms of severe disease and even deaths.
P. vivax malaria presenting with hepatic failure is rare with one report in 2014 by Nigam. Our case presented with symptoms and signs of deranged liver function and coagulation profile along with high grade fever with chills and rigors, without evidence of pre-existing liver disease and more importantly patient improved dramatically with i.v. artesunate therapy.
Second patient presented with diabetic ketoacidosis and acute renal failure. Renal failure in malaria is mostly due to acute tubular necrosis. Ischaemia is heralded by parasitaemia blocking capillaries, pooling of blood in the extremities, fluid loss, leaking of plasma through capillaries, etc. The patient improved up to a satisfactory level with conservative management and no haemodialysis was required (Amitabh et al., 2010).

Conclusion

There are significant changes noted in the epidemiology of malaria and the clinical presentation of vivax malaria has changed over last decade. Deregulated inflammation is supposed to play an important role in causation of multi-organ dysfunction. Early recognition, early diagnosis, prompt antimalarial therapy and good supportive care can help in preventing mortality in complicated vivax malaria.

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REFERENCES

Case Report

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