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Research Paper

Sickle cell Anaemia with Plasmodium Vivax and Tuberculous Infection-A Case Report

Medical Science

KEYWORDS : Sickle cell anaemia, plasmodium vivax, acute illness

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ABSTRACT

Sickle cell anaemia (SCA) is prevalent in areas where there is endemicity for malaria. Heterozygous condition of sickle hemoglobin is protective against plasmodium falciparum infection. Except from South Africa reports stating about the association of malaria with sickle cell anaemia were rare. Very few cases were recorded about association of Plasmodium vivax infection in sickle cell anaemia. The consequences of malaria in SCA may be very severe during acute illness. We present a rare case of vivax malaria in SCA who had been suffering from tuberculosis.

INTRODUCTION

The global birth prevalence of sickle cell disease is 300 000 per year and up to 70% of these births occur in sub-Saharan Africa(1).Malaria is thought to be a major cause of severe morbidity and mortality in these regions though the heterozygous condition is protective against plasmodium falciparum(2).Reports of association of malaria (vivax or falciparum) with sickle cell anemia are available from South Africa but surprisingly rare from other parts of the world(3). The first detailed report on the importance ofmalaria as a cause of morbidity and mortality in patients living with SCA was published inJanuary 2010(2).

Sickle cell disease patients frequently experience vaso-occlusive crises which lead to infarction of spleen and autosplenectomy (4). Malaria acts as a precipitating factor for the vaso-occlusive crises experienced by these patients (5).Increased attacks of malaria were observed in SCA patients during hospitalization and the consequences of malaria in SCA appear to be severe during acute illness (6). Some studies state that severe complications and high parasitemia were found in patients of sickle cell anaemia than the normal counterparts which is contrary to the present belief that sickle gene is protective against heavy parasitemia. It is also observed that the parasitemia is severe in old age group with lower Hb concentration and increased aspartate transaminase levels and may even lead to the death of the patient(2).

CASE REPORT

Male aged 35 yrs attended medical OP department with complaints of fever with chills and rigors since 5 days and cough with expectoration and chest pain since 15 days. History revealed that the patient is a known case of SCA and has been suffering from tuberculosis of the lungs and kidneys since 4 month and taking antituberculous drugs. On examination the patient had a high grade fever, severe degree of anaemia and yellowish discoloration of skin. Spleen was not palpable. The patient was subjected to routine urine examination, hemogram with peripheral smear and x-ray chest.

Hemogram showed Hb 5gms/dl. TLC-14.600/cu.mm, DC-polymrphs-72%, lymphocytes-14%, metamyelocytes-5%, monocytes- 7%, eosinophils 2%. Platelets 1.8lakhs/cu.mm, PCV 17%, ESR 45mm/1hr

Peripheral smear showed microcytic hypochromic RBC with severe anisocytosis and poikilocytosis, polychromatophilic RBC, nucleated RBC, Sickled RBC and fragmented RBC (Fig-1).There are plenty of ring forms, trophozoites(Fig 2) and schizonts(Fig-3) of Plasmodium vivax with pigmentation in the RBC.

X-ray chest showed diffuse opacities in both the lungs.

The patient was diagnosed as SCA with Plasmodium vivax malaria and tuberculous infection. The patient was hospitalized and put on antimalarial and antituberculous drugs. He developed hemolytic crisis and painful crisis with acute chest syndrome. He was treated as an inpatient for 15 days and was discharged.

DISCUSSION

Malaria and sickle cell anaemia still continue to be major public healthproblems challenging the infectious disease medicine and hematology fraternities.(7)

Sickle cell anemia is prevalent in areas endemic for malaria as a mechanism of natural selection because of the protective effect offered by the heterozygous condition. Selective advantage of HbAS heterozygotes over HbS homozygotes in the clinical course of malaria can be explained by the so called "balanced polymorphism where the genetic mutation (HbAS) is preferred for the severe complications that occur with P.falciparum(7) .Various mechanisms like genetic , molecular and immunological factors have been proposed to explain the protective effect of HbS against malaria. According to the sickling phagocytosis model which was established 40yrs back there will be phagocytosis of the AS parasitized red cells(8). Even today this model holds good and there is no evidence contrary to this model. The other protective mechanism could be the decreased adherence of parasitized RBC to the endothelium thus decreasing the risk of cerebral malaria.

Though the heterozygous condition is protective still malaria is considered to be the most important cause of morbidity and mortality in sickle cell anaemia. Luzzato opined that HbAS patients do get the infection but they tend to have a less parasitemia and they usually do not get the severe form of the disease and they don't get cerebral malaria. The reason for this may not be the failure of the organisms to invade RBC but the consequences that take place there after. Beet has suggested that the rate of sickling in AS parasitized cells is more when compared to those of non-parasitized red cells within the very same blood sample(9). So it is reasonable to consider that the parasite triggers sickling and the sickled RBC will subsequently be removed by macrophages.

The possibility for the lack of protection against malaria in the homozygous patients could be attributed to the loss of splenic function which resulted from the splenic infarction and fibrosis. Clinical experiences have shown that the combination of sickle cell anaemia and malaria will cause worsening of anaemia (10) and severe consequences that aggravate vasoocclusive crisis and may kill the patient. Because of the loss of protective effect of

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the spleen these patients are prone to be infected by Pneumococci, H. influenza, Salmonella, Staphylococci, E.coli etc. Some of the studies showed occurrence of tuberculosis of the spine(11) and kidney also. According to some studies the complications of malaria in SCA is more in acute illness and during hospitalization. Arti Prasad et al felt that severe complications and heavy parasitemia were found in patients of sickle cell anaemia contrary to the current thought that sickle gene protects against heavy parasitemia and all patients including the one with vivax had complications and took longer than usual time to recover.

The protective effect offered by HbAS against falciparum malaria is not established in other types of malaria which are less intense in severity . Duffy antigen helps in the internalization of P. vivax in to the RBC. Those people who have Duffy null phenotype antigen are protected from P. vivax malaria. This null phenotype is common in people whose ancestors belonged to the African countries where P. vivax is endemic.

In the present case the patient had been suffering from the complications of sickle cell anaemia, he was transfused many times before and was on treatment for pulmonary and renal tuberculosis. After diagnosing malaria the patient was admitted, antimalarial drugs were given and his general condition was taken care of . His hepatic and renal functions were assessed while the patient is on treatment. It took about 15 days for the patient to recover from the symptoms. He was discharged and lost for fallow up.

CONCLUSION

Very few studies were recorded in the literature stating the incidence of malaria in sickle cell anaemia especially P.vivx malaria. Concomitant occurrence of P.vivax and tuberculosis in SCA is even more uncommon. The disease severity of malaria in SCA is more because of high parasitemia and severe anaemia. Occurrence of malaria in the background of acute illness worsens the clinical picture. Plasmodium vivax infection which runs a benign course normally may be associated with a severe disease process if it occurs in SCA.

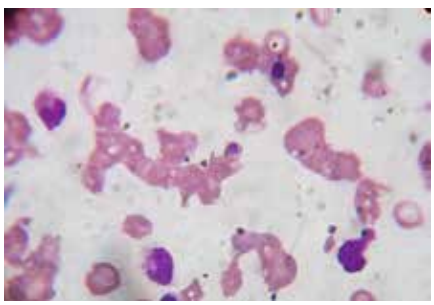

Fig-1 showing hemolytic picture with nucleated RBC(long arrow) and polychromatophilicRBC(short arrow) and ring forms of P.vivax (arrow head) 1000x

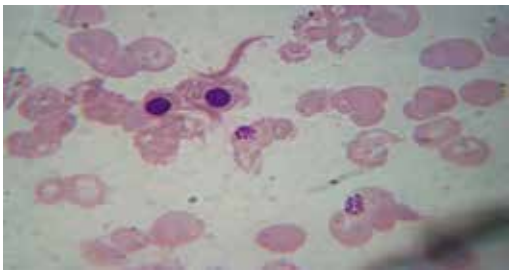

Fig-2 Showing irreversibly sickled RBC and Schizonts of P.vivax- 1000x

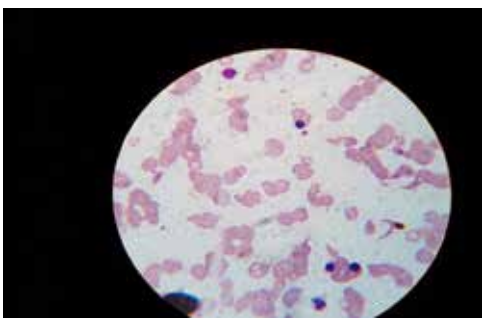

Fig-3 Showing Trophozoit in a sickled RBC mimicking P. falciparum gametocyte(long arrow)1000x

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