CASE REPORT

Vivax Malaria with Spleenic Abscess and Symmetrical Peripheral Gangrene

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Abstract

Changes in spleen structure, frequently encountered during malaria, may result either in a simple asymptomatic enlargement or in serious complications such as hematoma, rupture, or infarction. Hematoma or infarction of the spleen might be followed by the development of a spleenic abscess, a clinical condition that has been reported rarely. The occurrence of gangrene following infectious diseases is not uncommon; but the occurrence of symmetrical gangrene as a complication in vivax malaria is comparatively rare and is barely mentioned in any of the textbooks. The occurrence of symmetrical gangrene in malaria is mentioned by only a few authors; of all the complications and sequel of malaria, gangrene is also probably the rare. And occurrence of both of these together in a patient of P. Vivax malaria is an interesting case to be reported.

Keywords: Vivax Malaria, Spleen, Gangrene,

CASE REPORT

A 22 year old patient was admitted to PBM Hospital Bikaner with chief complains of intermittent fever with chills and rigors for 4 days, joint (ankle) pains and blackening of foot from 2 days. There was no history of DM, HTN and TB no past history of any bleeding daisthesis. CVS and peripheral arterial disease abnormality neither patient was a reported to be a smoker but history of occasional alcohol consumption was given. On examination all peripheral pulses were palpable and there was symmetrical blackening of both feet extending up to ankle and there was no line of demarcation. Patient was conscious and oriented to time place and person with normal reflexes and plantar were flexor. Blood pressure was 140/80mm of Hg and pulse was 98 mins there was no murmur heard on examination and S1 S2 appeared normal, abdomen on examination was soft and tenderness was present in left hypochonrial region, spleen was palpable 3cm below the left costal margin. Respiratory system was normal on examination without any added sounds and equal air entry.

Laboratory Findings

TLC was 12000 haemoglobin was 8.1 gm /dl and platelet being 70000 MCV-MCHC on higher side 97 and 31 respectively.LFT and RFT were in normal range. Urine routine showed normal finding. Bleeding time 3.5mins and clotting time 8.5mins and D-dimer was more than 10000. Card test for vivax malaria was positive on two different occasions though peripheral blood smear examination failed to show parasite as patient had consumed anti- malarial in periphery, chest X-ray was reported normal, USG abdomen showed moderate spleenomegaly with heterogenous hypoechoic collection of about 18 cm along the lateral border of spleen s/o spleenic abscess. Echocardiography showed no vegetation and no clot. ANA was negative and so was anti phospholipids antibodies, color Doppler was normal up to dorsalis pedis artery.
Discussion

Although the association of Symmetrical peripheral gangrene and spleenic abscess with Plasmodium falciparum malaria was documented separately in literature, but their occurrence together and that to vivax malaria appears to be a rare phenomenon. Symmetrical peripheral gangrene is an uncommon complication of falciparum malaria reported mainly in Asian countries. The exact pathogenesis for bilateral gangrene remains uncertain and may be multifactorial; most of the cases reported in the literature were associated with DIC. In 2004 S.Kakati et al reported case of symmetrical peripheral gangrene and falciparum malaria. Peripheral gangrene was reported in a case series of 3 cases by Vipa Thanchartwet et al in 2006. In 2010 ratan kr et al reported symmetrical peripheral gangrene and neurological manifestation in a case of plasmodium falciparum malaria.

Symmetrical peripheral gangrene was considered as cutaneous marker of DIC (Molos MA, Hall JC in1985). Other causes of symmetrical peripheral gangrene include septicaemia which is the most common cause in clinical setting as conclude by T P stossel et al in 1970, rest being asplenia, immunosuppression, diabetes mellitus, renal failure, cold injury to the extremities, myoglobinureaemia, increased sympathetic tone and the use of vasopressor drugs. We reported a case of a 22 year-old man admitted in our hospital for Plasmodium vivax malaria with disseminated intravascular coagulation (DIC) and peripheral gangrene of his toes. To our knowledge this was the first case reported to be associated with vivax malaria.

Early in 1985 Mohanthy D et al reported about the hypercogulable state in P. Falciparum malaria. The mechanisms involved in the activation of the coagulation cascade in severe falciparum malaria were studied in 22 adult patients by R Clemens et al who concluded that there was a reduction in plasma antithrombin III (AT III) concentrations and elevation in thrombin-AT III complexes were associated with significant reductions in factor XII and prekallikrein activities, and an increase in the C1 inhibitor antigen/activity ratio. These results suggest that the intrinsic pathway of the clotting cascade is activated in severe malaria. This may cause activation of the complement system and release of bradykinin and PMN-elastase and could contribute to the pathogenesis of severe malaria.

Infected red cells may play an important role in initiating photoagulate activity or the indirect effects of clotting.
cascade activation may lead to the blockage of small capillaries and DIC. Cytoadherence and rosetting lead to micro-circulatory obstruction in malaria. Several vascular receptors for the adhesive surface protein of infected erythrocytes have been identified - which causes adhesion of the infected red cells to the vascular endothelium. The molecules responsible are CD-36, intra-cellular adhesion molecule-1 (ICAM-1), thrombospondin (TSP), vascular cell adhesion molecule-1 (VCAM-1), endothelial leukocyte adhesion molecule-1 (ELAM-1) and histidine, rish protein as mentioned in Manson's Tropical Infectious Diseases 23rd Edition.

Splenic enlargement is common in malaria, it depends on the immune status of the individual. If the disease remains untreated it can progress and result in splenic rupture. P.vivax malaria is the most closely associated with splenic rupture. Spontaneous rupture of the spleen occurs almost exclusively during a primary attack (Zingman and Viner, 1993). The incidence of splenic hematoma without rupture is unknown (Mokashi et al, 1992). Splenic infarction is rarer than rupture. P. vivax was the common species present. Each patient presented with left hypochondriac pain which usually develops at the end of the first week of fever. Clinicians must be aware that left hypochondrial pain occurring during treatment for acute malaria may be due to splenic infarction. The clinical outcome is favourable management. The spleen plays an integral role in the host defence against plasmodium and other intravascular parasites (Hamelet al,2002). An attempt at splenic salvage should be made in order to avoid future fatal malaria infections and possibility of remission (WHO, 2006). A conservative approach, including complete bed rest, close observation of vitals and hematocrit levels, a correct dose of anti malarial medication, fluid resuscitation, blood transfusion as needed and avoiding coughing and vomiting to prevent an increase in intra-abdominal pressure are all important unnoticed (Bonnard et al, 2005).

References