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CASE REPORT

PERNICIOUS SYNDROME OF FALCIPARUM MALARIA AND LEUKAEMOID REACTION

Abdul Salam Khan, Abdur Rahim Khan and Saifur Rehman

A female child aged 11 years, was referred to our Clinical Laboratory by private local medical specialist. The patient had 10 days history of fever of sudden onset. She had been given injection Amoxycillin and Injection Chloroquine by a general practioner.

On examination the child was very sick and pale. The pulse was 140 beats/min, regular, temperature 100°F in axilla, systolic murmur was audible in a wide area over the praecordium. The liver palbable 3 fingers below the right subcostal margin. X-ray chest showed mitralization and shifting of right border of the heart (fluid overload). Lung fields were congested.

Hematological findings were Hb 7.0 gm/dl ESR, 95mm in 1st hour, TLC, 55,500/m3 and DLC, Neutrophils 83%, Metamyelocytes 4%, Myelocytes 02%, Band forms 04% and Lymphocytes 2%. The Neutrophils showed toxic granulations. The reticulocyte, count was not done as the patient expired. Normoblasts were 5%. Both basophilic and polychromatic forms were seen. The blood film was positive for malaria. The slide was teaming with gametocytes of plasmodium falciparum. In some RBC's ring forms of the parasites were seen. There were about 10-12 gametocytes/HPF. The RBC's were enlarged in size. There was no poikilvaytosis. Some target cells were also present.

Urine analysis showed a dark coloured urine. Test showed Bilirubinuria and urobilinogen was markedly increased. Microscopically there were 20-25 RBC/HPF and 6-7 pus cells/HPF.
The widal test was negative. Blood chemistry results were, serum Bilirubin, 4.2 mg/dl (mainly direct conjugated BILIRUBIN), ALT 70U/1, ALP 31.5 KA Units and Urea 150 mg/dl. The patient could not be screened for G-6PD as she expired on the second day.

Discussion

The patient came from WANA, South Waziristan near the Afghan border.
In response to persisting infection by virulent organism's resistant to the

From Gumal University, D.I. Khan
ABDUL SALAM KHAN, Dept. of Basic Medical Sciences, Faculty of Pharmacy
ABDUR RAHMAN KHAN, Dept. of Chemistry (Biochemistry Section)
SAIFUR RAHMAN, Physician, D.I. Khan Cantt.

bactericidal effect of neutrophils, the granulocytes increase in number from 50,000 to 100,000 cells/ul. Counts in excess of 100,000 cells/ul have been observed in patients with osteomylitis, empyema, septicaemia and tuberculosis. By convention, leukocytosis exceeding 50,000 cells/ul is called as leukaemoid reaction.[1] In this case there was marked leukocytosis with a shift to the left. The patient was highly toxic due to marked degree of parasitae- mia (about 100,000 organisms/ul).

Anaemia is usual in malaria; it is often only mild or moderate but is sometimes severe particularly in falciparum infections.[2] The extremely high level of parasitaemia is responsible for severe anaemia in this case. It has been suggested that various pernicious syndromes that occur in falciparum malaria are caused by combination of anaemic hypovolemia and capillary obstruction. The injected red cells adhere to vascular endothelium of the target tissues, producing different pernicious syndromes.[3] Such local circulatory changes occurring in the brain liver, kidneys and bone marrow may result in irreversible damage to these and other organs.[4]

The presence of marked anaemia and obstruction of renal, hepatic and pulmonary microvasculature due to high degree of parasitaemia is responsible for the clinical and biochemical manifestations in this case. The authors have already reported a case of leukaemoid reaction associated with vivax malaria.[5] The patient belonged to the same area. The severity of malaria in the same locality may be due to the role of genetic and environmental factors.

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