Splenic Infarct in Falci parum Malaria

Splenic infarction is common in myeloproliferat ive syndrome, sickle cell anemia and lymphomas. This condition has only occasional ly been reported in severe cases of malaria(l). We report a case of splenic infarct in falciparum malaria during an epidemic in Calcutta.

A previously healthy eleven years old boy was admitted with high fever for three days associated with chest pain and vomiting for two days. Prior to admiss ion, the patient was diagnosed outside as a case of falciparum malaria and was treated with oral chloroquine. On examination we found a feverish child with moderate pallor and hepatosplenomegaly. Both liver and spleen were five cm enlarged, firm and nontender. There was no purpuric spot, lymphaden opathy or bone pain. Rest of the examination was normal. Blood picture showed pancy topenia, elevated reticulocyte count and gametocytes of Plasmodhim falciparum. Blood sugar, widal test and urine examination were normal.

Apprehending chloroquine resistance, oral quinine was started on the fourth day. Two days later, the patient complained of severe pain and tenderness over left upper abdomen. Surgeons were consulted and an urgent ultrasonography (USG) was performed. USG screening showed a single splenic infarct area of 22 mm. Conservative management was started with analgesics, fluid and electrolyte therapy and proph y-

lactic antibiotics. The patient recovered gradually within a week and the USG was repeated after seven day. Repeat scan showed almost the same size of the infarct area. The patient recovered uneventfully and was discharged ten days after the episode.

The possible factors contributing to splenic infarction in falciparum malaria are tissue avascularity following rapid splenomeg aly and increased stickiness of the parasitized RBC's. USG, CT Scan and radioisotope study are the investigations of choice(2) and management is usually conservative. Prophylatic antibiotics and, rarely, splenectomy are required to treat splenic abscesses.

A.K. Sur, N.Khawash, P. Khawash Mitra, K. Ghosh, D. Sinharoy,

Department of Pediatrics, Ramakrishna Mission Seva Pratisthan and Vivekananda Institute of Medical Sciences, Calcutta 700 026.

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