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TEACHING FILES (GRAND ROUNDS)

FULMINANT HEPATIC FAILURE IN A CHILD WITH FEVER AND JAUNDICE

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Clinical Problem:
A 7-year-old girl presented with fever for 6 days, jaundice for 5 days, vomiting and loose stools for 2 days and altered sensorium and decreased urine output for 1 day. On admission, her blood pressure (BP) was 82/52 mmHg, pulse rate was 138/min, respiratory rate was 42/min and capillary refill time was 2 seconds. Peripheral pulses were well felt. She had jaundice and no pallor. She had firm hepatomegaly and altered sensorium with Glasgow coma scale of 11/15 (E3M5V3). There were no focal neurological deficits or meningeal signs. Other systems were normal. Grandmother was simultaneously admitted with falciparum malaria in the same hospital. Investigations showed hemoglobin (Hb) of 12.1 gm%, white blood cells (WBC) of 18,900/cumm, platelets of 8,000/cumm. Peripheral smear showed *Plasmodium falciparum* with parasitic index of 15%. Total serum bilirubin was 15.4 mg/dl, direct bilirubin 11.5 mg/dl, serum glutamic oxaloacetic transaminase (SGOT) 121 U/l, serum glutamic pyruvate transaminase (SGPT) 63 U/l, international normalized ratio (INR)- 1.13, blood urea nitrogen 100 mg/dl, serum creatinine 1.83 mg/dl. She was given intravenous (IV) fluid bolus, platelet transfusion, started on anti-malarial therapy (artesunate and quinine) and IV antibiotics (piperacillin-tazobactam) along with dopamine drip up to 15 mcg/kg/min (required for 7 days) and noradrenaline drip of up to 0.3 mcg/kg/min (given for 48 hours) to maintain hemodynamic stability. Artesunate was given in a dose of 2.4 mg/kg IV, repeated after 12 and 24 hours and then every 24 hours for total of 7 days. On 7th day, oral Mefloquine 25 mg/kg as a single dose was given. Quinine dihydrochloride was given as an IV infusion, in a dose of 20 mg/kg first dose and then after 8 hours 10 mg/kg followed by 10 mg/kg every 8 hourly for total 7 days. Sensorium improved within 24 hours of starting anti-malarial therapy. On day 2, patient developed signs of capillary leak in the form of hypotension, poor perfusion and respiratory distress. Complete blood count on day 2 showed Hb 9.4 gm%, WBC 27,200/cumm, platelets 19,000/cumm. Parasitic index dropped to 5% after 24 hours of starting anti-malarial therapy and no malarial parasites were visible in the peripheral smear after 48 hours. Mechanical ventilation was required on 4th day in view of increasing respiratory distress and impending respiratory failure. She showed gradual improvement and was weaned and extubated on 7th day of hospitalization, following which she made a rapid recovery. During recovery, on 12th day of hospitalization, she developed cold antibody induced hemolytic anemia (Hb- 6.1 gm/dl, cold antibodies- positive), requiring blood transfusion. She was discharged after 15 days of hospital stay. On follow up, she was well, and her liver function tests (LFT), and renal function tests (RFT) were normal.

How to differentiate malarial hepatitis from viral hepatitis?

Discussion:
Malaria is a life threatening illness caused by Plasmodium parasites, transmitted through a bite of infected female anopheles mosquito. The cause of hepatopathy in falciparum malaria is erythrocyte membrane alteration caused in parasitized RBCs causing sequestration of RBCs in hepatic microcirculation (sinusoids), leading to clogging of microcirculation and resulting in ischemia and secondly, the effect of cytokines on the liver.[1,2] Srivastava et al described seven patients with severe falciparum malaria presenting like fulminant liver failure.[3] All of them presented with fever, jaundice, altered sensorium, oliguria, moderate to severe anemia, elevated liver enzymes (2-4 times the normal) and azotemia (serum creatinine 1.6-7 mg/dl). Only one patient had splenomegaly. Our case also had similar symptoms and laboratory values except for anemia. Our patient did not have pallor, her hemoglobin on admission was 12 gm/dl and she didn't have splenomegaly. Normal hemoglobin in our case could be due to hemoconcentration as the child was dehydrated on admission and on second day her hemoglobin dropped to 9 gm/dl. In a study by Devarbhavi et al, 25 patients with malarial hepatopathy simulating fulminant liver failure were compared with 25 patients with viral hepatitis induced fulminant hepatic failure .[4] The study showed that presence of hepatomegaly and normal prothrombin time favors malarial hepatopathy, as was seen in our case. If treated appropriately, malarial hepatopathy has better prognosis than viral induced fulminant hepatic failure. The study also showed that there were no statistically significant difference between

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duration of jaundice, duration of altered sensorium and time interval between onset of jaundice and onset of altered sensorium between the two groups. The study concluded that severe malaria should be considered in the differential diagnosis, when there is hepatomegaly and normal prothrombin time in the setting of fulminant hepatic failure, as was seen in our case.

Compliance with ethical standards

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