Acute renal failure in favism revealing familial glucose-6-phosphate dehydrogenase deficiency

Sir,

A previously healthy 28-year-old man of North Africa origin presented to the emergency department with a 10-day history of jaundice, vomiting, and abdominal pain, which came with chills, without fever. Two days before his admission to hospital, the patient had noticed dark colored urine and developed oliguria after ingestion of fava beans. The patient was not on medication. There was no history of urinary infection or hematuria, but he noticed a history of intermittent jaundice (six episodes) coinciding with the ingestion of fava beans or after inhalation of pollens with passing of dark colored urine, once associated with reduced urine volume. Physical examination revealed a fully conscious patient but ill-appearing jaundice without any other clinical abnormality. Investigations revealed normocytic normochromic regenerative anemia with initial hemoglobin 4 g/dl, reticulocyte count was 161000/ mm^3, total leucocytes count 10000/mm^3, polymorphs 76%, lymphocytes 13%, and C-reactive protein (CRP) was 36 mg/L. Urine was dark brown, albumin +++ and Blood ++; no bile salts and pigments or red blood cells were seen, but hemoglobinuria was confirmed. Blood smear and hemoglobin electrophoresis gave normal results, red cell fragility to normal saline was impaired. Blood smear study for malaria parasites, flow cytometry, Coombs' test, search for irregular agglutinins were negative. Anti-neutrophil cytoplasmic antibody (ANCA), anti-nuclear antibodies (ANA), and anti-DNA tests showed negative results. Results for chest X-ray and the renal ultrasound were normal.

The importance of G6PD deficiency in favism is due to the risk of acute intra-vascular hemolysis, which usually begin 24 hours after ingestion of the beans, and hemoglobinuria may continue for several days.[1,2] Urine output was 150 ml/day 24 hr after admission; blood urea at this stage was 372 mg/dL, and serum creatinine 28 mg/dl. Hemodialysis was started immediately and was continued for 14 days. He was given three units of blood during the first session of hemodialysis. Jaundice and hemoglobinuria also subsided within one week. From day 10, he entered into the classical diuretic phase, with urine volume gradually rising to 2900 ml/day. Serum creatinine came down to 1 mg/dl on the 25th day, with no abnormalities in the urinary sediment. The hemoglobin improved to 9 g/dl with blood transfusion.

The development of severe hemolytic anemia shortly after ingestion of fava beans raised strong suspicion on the presence of a G6PD deficiency. Laboratory testing by enzymological method revealed erythrocyte G6PD activity of 34 Mu/xEryth (reference value, 118-144 Mu/xEryth). Subsequently, the patient's general and renal condition recovered completely.

Four members of the patient's family – his mother, one brother, and two uncles – reported a history of episodes of jaundice after ingestion of fava beans. Only his mother and brother were examined, and the brother was found to have G6PD deficiency. The erythrocyte G6PD activity in the patient was 20 Mu/xEryth, but he experienced no alteration in the renal or hematologic function.

Although the fact that acute renal failure is an uncommon complication in G6PD deficiency, its consequences remain serious, involving the vital and renal prognosis. Renal lesions occur mainly due to acute tubular necrosis and tubulointerstitial nephritis. We believe that early diagnosis of G6PD deficiency and essentially preventive treatment by expulsion of oxidizing products, such as drugs and fava beans, can help avoid dramatic complications, particularly those of acute kidney failure.

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