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An Uncommon Cause of Portal Hypertension: Schistosomiasis

To the Editor:

A 25-year-old Sudanese man presented with recurrent right upper quadrant abdominal pain. The patient reported episodic abdominal pain, which began upon arrival to the US 2 years earlier. The patient lived in Sudan until age 21 years and described freshwater exposure until age 15. The patient denied history of fever, vomiting, diarrhea, or weight loss. He denied tobacco or alcohol use. Physical examination revealed palpable hepatomegaly 8 cm below the costal margin and splenomegaly extending 5 cm below the costal margin. Stigmata of chronic liver disease were absent.

Laboratory values were remarkable for leukopenia at 2450 per mm^3, a hematocrit of 48.4%, and thrombocytopenia at 41,000 per mm^3. A liver panel revealed an elevated total bilirubin of 1.2 mg/dL, alanine aminotransferase of 82 units/L, and aspartate aminotransferase of 114 units/L, while the international normalized ratio and albumin were normal. A peripheral blood smear was negative for schistocytes and 3 stool specimens were negative for the presence of ova and parasites. A urinalysis was negative for blood. Hepatitis A, hepatitis B, hepatitis C, cytomegalovirus, Epstein-Barr virus, and human immunodeficiency virus serology tests were all either negative or nonreactive. Transferrin saturation was not elevated and serum ceruloplasmin was normal. A bone marrow biopsy of the iliac crest was normal.

Computed tomography with contrast of the abdomen revealed splenomegaly measuring 16 cm with prominent splenic and portal veins, suggestive of portal hypertension (Figure, Part A). An abdominal magnetic resonance imaging revealed periportal edema (Figure, Part B). A subsequent abdominal ultrasound with Doppler revealed antegrade flow of the portal vein and patent hepatic veins with increased periportal fibrosis and edema characteristic of parasitic infection.

Of the various *Schistosoma* species found throughout the world, endemic to Sudan are *S. mansoni* and *S. haematobium*.[1] While *S. mansoni* tends to reside in the liver and portal venous plexus of its host, *S. haematobium* is a more frequent resident of the bladder, with hematuria being a prominent clinical manifestation.[2] *S. mansoni* infect freshwater snails, which release motile cercariae that penetrate the skin of a definitive host. The cercariae migrate through tissues and the vascular system, maturing to become egg-laying adult worms.[2] Eggs obstruct the portal vein, causing granuloma formation and hepatic fibrosis. The sequelae of

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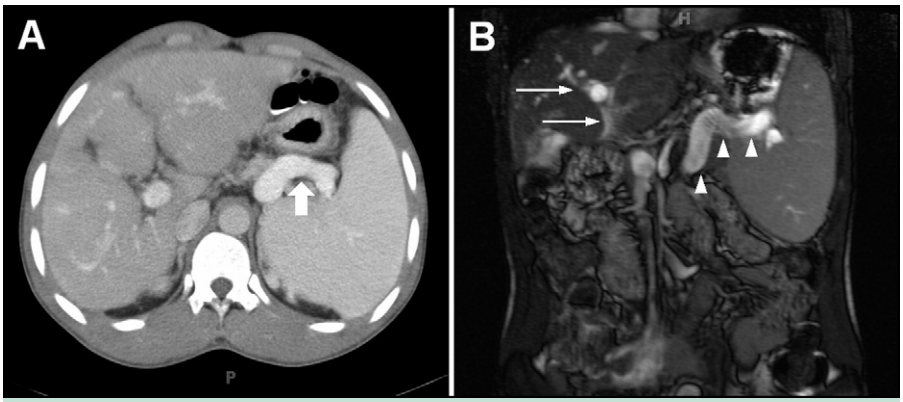

Figure Computed tomography with contrast of the abdomen (**Panel A,** transverse image) revealed splenomegaly, and splenic vein enlargement as evidence of portal hypertension. Magnetic resonance imaging with gadolinium (**Panel B,** coronal image) revealed splenomegaly, enlarged splenic and superior mesenteric veins, and periportal edema.

fibrosis include portal hypertension, splenomegaly, and gastrointestinal varices.[1]

Over the past 10 years, more than 21,000 Sudanese refugees immigrated to the US.[3] In a recent study of Sudanese refugees, approximately 44% were seropositive for *Schistosoma*, primarily *S. mansoni*.[4] At present, refugees are not routinely screened, and infected individuals are often missed by national control programs.[4] As fibrotic liver damage associated with *S. mansoni* infections occurs over the course of 5-15 years, by the time clinical symptoms are observed, the infection may no longer be present or detectable.[1]

Serology results obtained after discharge revealed an elevated immunoglobulin G antibody toward *schistosoma*. Given the marked liver involvement and absence of hematuria, *S. mansoni* is the most likely cause of our patient's portal hypertension. Before discharge, the patient was treated with 2 doses of praziquantel for putative infection and propranolol for management of portal hypertension. Although praziquantel should stop ongoing damage from a residual infection, the existing damage is not expected to improve despite antiprotozoal intervention.[1]

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