CLINICAL VIGNETTE

Schistosoma Infection Presenting as Iron Deficiency Anemia

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Schistosomiasis is the second commonest parasitic disease in the world. It is most prevalent in sub-Saharan Africa. We describe a case of Schistosoma haematobium infection presenting as iron deficiency anemia and review its epidemiology, pathogenesis, clinical presentation, diagnosis, treatment and prevention.

Case

A 55-year-old female was admitted for evaluation of iron deficiency anemia. Patient is originally from Cameroon but had lived in the United States for over 25 years. During this time she had visited Cameroon several times, the last time a few months before admission. Two months before admission, she was diagnosed to have type 2 diabetes, essential hypertension, chronic kidney disease and microscopic hematuria. A renal ultrasound at that time had revealed mild bilateral hydronephrosis, felt most likely secondary to neurogenic bladder due to diabetes. Her hemoglobin was 10.9 g.

At the time of consultation during this hospital admission, her hemoglobin had dropped to 8.4 g. Labs were consistent with iron deficiency. Helicobacter pylori gastritis was found on endoscopy and two diminutive adenomatous polyps were removed during colonoscopy. Repeat urine analysis demonstrated large amount of microscopic blood. Repeat renal ultrasound revealed worsening bilateral hydronephrosis and over 500 cc of post void urine in the bladder. CT scan abdomen and pelvis confirmed bilateral hydronephrosis with severely dilated ureters. Right ureter was over 2.5 cm in diameter and left ureter over 2 cm in diameter. Urinary bladder was distended and bladder wall was thickened suggestive of either urinary bladder outlet obstruction or a neurogenic bladder. On cystoscopy, no bladder neck obstruction was noted. However, her ureteral orifices were small and very tight, intramural tunnels were scarred on both sides and there was marked dilation of proximal ureters. Each ureter was over 30 cm in length including tortuosity. The bladder wall demonstrated some erythema and bruising. Biopsies were taken of the abnormal appearing areas and bilateral ureteral stents were placed. Biopsies revealed eggs of Schistosoma haematobium (Figure 1). Her final diagnosis was Schistosoma haematobium urinary tract infection and iron deficiency anemia due to chronic urinary blood loss secondary to S. haematobium infection. She was treated with a single dose of 40 mg/kg praziquantel and microscopic hematuria resolved.

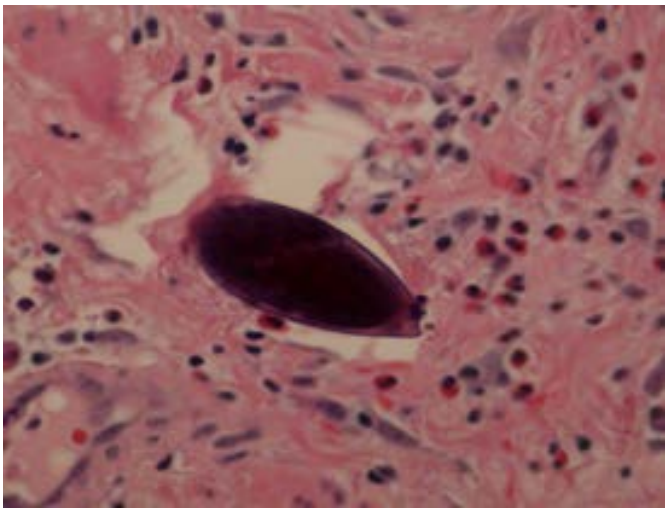

Figure 1.

Discussion

Schistosomiasis is also called Bilharzia after Theodor Bilharz who first recognized the parasite in 1852. It is caused by Schistosoma, a trematode (blood fluke). Over 200 million people are infected worldwide[1,2] with over 200,000 deaths annually.[3,4] According to Centers for Disease Control (CDC), schistosomiasis is one of the "neglected tropical diseases".[5] There are five different species of Schistosoma. The three commonest species are S. mansoni found throughout Africa, Nile River Valley in Sudan and Egypt, South America: including Brazil and Venezuela; S. haematobium found throughout Africa, Nile River Valley in Egypt and some areas of Middle East and S. japonicum found in Indonesia and parts of China and Southeast Asia. The other two species are S. mekongi in Cambodia and Laos and S. intercalatum in Central and West Africa.

The life cycle of schistosomiasis is complex (Figure 2). Snail is the intermediate host and humans are definitive host. An infected person contaminates fresh water with eggs through feces or urine. The eggs hatch and release miracidia, which penetrate snails. Cercariae from the snail are released into the water after four to six weeks. Cercariae penetrate human skin, become schistosomulae, which migrate through the circulation to reach the liver where they mature into adults. Adult S. japonicum, S. mekongi, S. mansoni and S. intercalatum then migrate to the small and large intestine and the mesenteric venules of colon. S. haematobium migrates to the vesical venous plexus. The

female worms then begin to lay eggs in the venules of the mesenteric or perivesical systems. These eggs are subsequently excreted in stool or urine.[5,6]

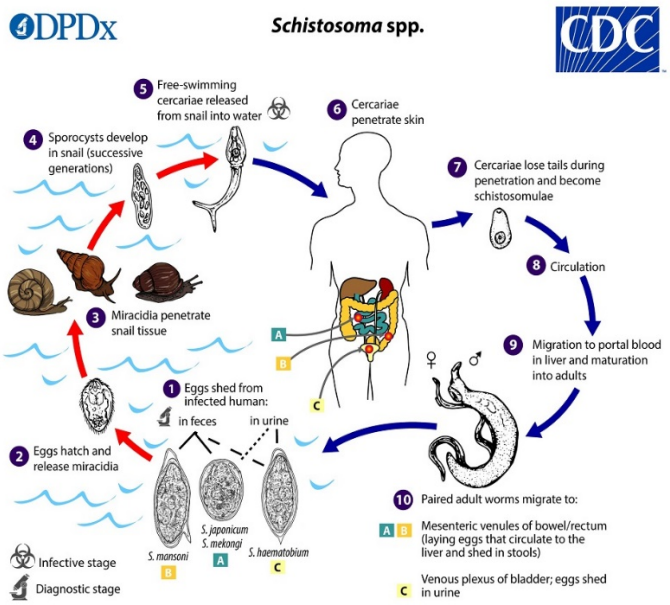

Figure 2.

Most infected individuals remain asymptomatic. Symptoms are caused by host immune response to migrating eggs. Some people, after swimming in fresh water develop swimmer's itch due to cercariae penetrating the skin. Acute schistosomiasis syndrome, also called Katayama fever, is typically seen 3 to 8 weeks after infection mostly in travelers who have no immunity.[7] Patients may develop fever, urticaria, angioedema, myalgia, arthralgia, diarrhea and dry cough. Symptoms are usually mild and resolve spontaneously.

Symptoms of chronic infection depend upon the organ involved. S. mansoni and S. japonicum which infect the intestinal tract can cause chronic, intermittent abdominal pain, diarrhea, colon ulcerations, bleeding and iron deficiency anemia. Hepatosplenic schistosomiasis can lead to periportal fibrosis, portal hypertension, splenomegaly and varices. Patients can develop pulmonary hypertension and cor pulmonale. Genitourinary schistosomiasis (S. haematobium) can cause dysuria, urine retention, hematuria, bladder ulcerations, hydronephrosis and stricture formation. There is increased risk of bladder cancer, immune complex glomerulopathy and nephrotic syndrome. Female genitalia lesions and ulcerations are also reported. Schistosomiasis can also lead to transverse myelitis, seizures and motor or sensory symptoms.

Patients with schistosomiasis may develop anemia, thrombocytopenia and about 50% of patients will have eosinophilia.[8] Eosinophils can also be found in CSF in patient with neuroschistosomiasis. Serology for antibody testing is the best test for screening in returning travelers, while in the residents of endemic areas, presence of eggs in the stool or urine, or presence of schistosome antigen or DNA in the blood, urine, and/or stool; and positive blood antibody tests are preferred.[9]

Treatment of acute schistosomiasis syndrome initially is prednisone 20 to 40 mg daily for 5 days.[10] About 4 to 6 weeks later praziquantel should be given. In chronic S. mansoni and S. haematobium infection, the drug of choice is praziquantel 40 mg/kg in one or two divided doses; and for S. japonicum praziquantel 60mg/kg in 2 divided doses. One time dose is usually sufficient but the dose may have to be repeated after two weeks. In praziquantel resistance or refractory cases, oxamniquine and mefloquine can be used.

No vaccine is available against schistosomiasis. To prevent schistosomiasis infection, swimming or wading in freshwater in countries where schistosomiasis occurs should be avoided. Swimming in ocean and chlorinated swimming pools is safe. Bathing water should be boiled for 1 min. Improved sanitation, mass drug treatment of entire communities, targeted treatment of school age children and development of a vaccine is required for the control and eradication of schistosomiasis.

Conclusion

Schistosomiasis is the second commonest parasitic infection worldwide but it is rarely seen in the United States. However, schistosomiasis should be considered in differential diagnosis in immigrants or patients with travel history to endemic areas who present with clinical features as described above.

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