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Diagnostic Challenges of Advanced Hepatosplenic Bilharziasis: Regarding a Case Report at the Regional Teaching Hospital of Ouahigouya in Burkina Faso

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ABSTRACT

Introduction: *Bilharzia or hepato-splenic schistosomiasis is caused by lesions related to Schistosoma mansoni, a trematode of the genus Schistosoma. This chronic waterborne parasitic disease is a major public health problem in sub-Saharan Africa, where it is endemic. It can be complicated by gastrointestinal bleeding due to rupture of esophageal varices.The aim of this observation is to report on the diagnostic approach and challenges of advanced-stage hepatosplenic bilharzia in a resource-limited setting.*

Case History: *we report the case of a 20-year-old man with a history of swimming in fresh water, hematemesis due to rupture of varices, lost to follow up and admitted to our department for hematemesis, and in whom clinical and paraclinical examination led to a diagnosis of chronic hepatosplenic bilharzia complicated by gastrointestinal bleeding due to rupture of esophageal varices. The patient was treated for his gastrointestinal bleeding and administered anti-bilharzia treatment. The outcome was straightforward.*

Conclusion: *Hepatosplenic bilharzia remains a major public health problem in Burkina Faso. In countries with limited resources, and particularly in a medium-sized town such as Ouahigouya, diagnosis is often based on a combination of epidemiological, clinical, and, above all, radiological evidence.*

Keywords

Hepatosplenic bilharzia, Diagnosis, Advanced stage, Ouahigouya.

Introduction

Hepatosplenic bilharzia or schistosomiasis is caused by lesions related to *Schistosoma mansoni*, a trematode of the genus Schistosoma. This chronic waterborne parasitic disease is a major public health problem in sub-Saharan Africa, where it is endemic. In Burkina Faso, the average prevalence of bilharzia is 2.4%, with regional disparities [1]. It results from granulomatous

inflammatory reactions and periportal fibrosis induced by the retention of eggs in the liver, which can progress to non-cirrhotic portal hypertension, with a risk of upper gastrointestinal bleeding following their rupture.

We report a case of advanced hepatosplenic bilharzia at the Ouahigouya University Hospital Center in Burkina Faso, Sub-Saharan Africa.

The interest of this observation lies in the fact that it describes the diagnostic approach and challenges of advanced hepatosplenic bilharzia in a resource-limited setting.

Case History

A 20-year-old male gold miner was admitted to the Hepatology and Gastroenterology Department of the Regional Teaching Hospital of Ouahigouya in August 2024 for hematemesis.

The patient's medical history revealed that he had swum in fresh water during childhood and had experienced hematemesis in 2017, for which endoscopic examination had revealed grade II upper gastrointestinal bleeding with red signs and congestive fundic gastropathy due to portal hypertension (PHT). The patient was lost to follow-up. There was no mention of epigastric pain or the use of gastrotoxic medications.

Clinical examination on admission revealed the patient's general condition to be WHO 1, weight 68 kg, estimated height 170 cm, giving a BMI of 23.5. The conjunctivae were moderately colored, anicteric, with moderate signs of shock and Hackett type III splenomegaly.

The biological assessment revealed anemia with hemoglobin at 5.7 g/dl, normochromic normocytic, thrombocytopenia at 102·000/ mm^3, and normal serum transaminases. Tests for HBs antigen, anti-HBc antibody, and anti-HCV antibody were negative. Bilharzia serology could not be performed due to its unavailability in our practice setting. Parasitological examination of stool samples did not reveal any schistosome eggs. Thick smear testing for *Plasmodium falciparum* malaria was negative.

The rectal mucosa biopsy (RMB) revealed chronic nonspecific proctitis (fibrosis with eosinophils but no schistosome eggs).

Abdominal ultrasound showed that the liver was of normal size, the portal vein was of normal size (12 mm) and showed parietal thickening just before the bifurcation; the walls of the portal branches were also thickened, especially in the hilar region, with a diffuse peri-portal hyperechoic appearance resembling a "pipe." There was a portal back-and-forth flow on pulsed Doppler. The splenic vein was of normal caliber (08 mm). Collateral venous circulation was noted at the hepatic and splenic hilum. The gallbladder was alithisic, with a slightly thickened wall (05 mm), and significant splenomegaly measuring 187 mm x 179 mm, homogeneous and with regular contours; there was a small amount of peritoneal fluid effusion. Overall, the ultrasound findings were suggestive of **Homeida stage 3** periportal fibrosis with portal hypertension strongly suggestive of hepatosplenic bilharzia.

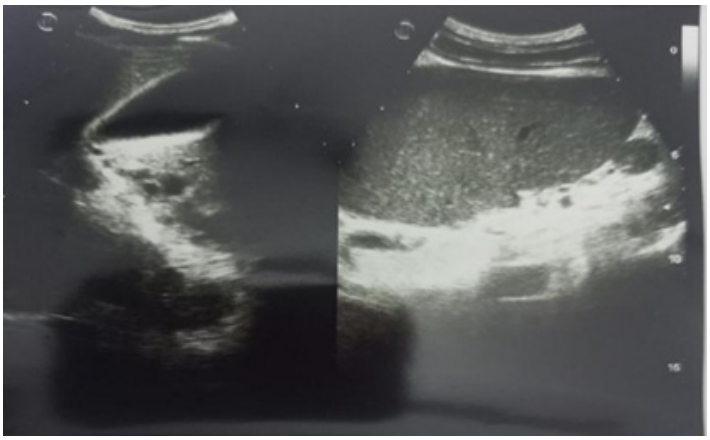

Figure 1: Peri-portal fibrosis and splenomegaly.

Treatment consisted of emergency measures to combat shock and a transfusion of iso-group iso-Rhesus blood.
After ruling out any contraindications to beta blockers, the patient was placed on propanolol at a dose of one 40 mg tablet every 8 hours.

Antibilharzia treatment was initiated with praziquantel at a dosage of 75 mg/kg in 3 doses. Since his discharge, the patient has been undergoing clinical, biological, and endoscopic monitoring to ensure that any new decompensation is detected.

Discussion

Human infection occurs during bathing in fresh water, even for a short period (less than 10 minutes). The cercariae actively penetrate the integument of any part of the human body that is immersed in the water (transcutaneous route). These freshwater environments are home to mollusk intermediate hosts specific to the parasite. For *Schistosoma mansoni*, this intermediate host is a mollusk of the genus Biomphalaria. The species *Biomphalaria pfeifferi* is the most widespread in Africa. The definitive host, humans, excrete the eggs in their feces. Once in fresh water, they release micardia that infect mollusks. Bilharzia is prevalent in tropical and intertropical areas where the temperature varies between 26 and 30°C. The disease mainly attacks the colon, then the liver and spleen. It causes reactive hyperplasia of the spleen, the formation of peri-ovular granulomas, and hepatic fibrosis at the onset of the disease. Later, portal hypertension without cirrhosis occurs, which is often overlooked in rural areas [2]. Sudden, life-threatening hemorrhage can occur as a result of the rupture of gastroesophageal varices. This was the case with our patient, who was hospitalized following hematemesis due to the rupture of esophageal varices, as confirmed by upper digestive endoscopy.

Epidemiologically, the WHO [3] estimates that approximately 240 million people worldwide are infected with various species of schistosomes. The prevalence of the hepato-splenic form *(Schistosoma mansoni*) is between 4% and 8% of people affected

by schistosomiasis [4].

The geographical distribution of schistosomiasis follows the distribution of its intermediate host. Burkina Faso is an endemic area for bilharzia, where access to drinking water and sanitation are still not a reality. We found in the patient's medical history that he had a history of swimming in fresh water, a factor that promotes contamination by *Schistosoma mansoni*.

Clinically, the patient presented with moderate hematemesis with moderate hemodynamic repercussions, portal hypertension syndrome (PHS), and no hepatomegaly. Hepatosplenic schistosomiasis manifests as signs of portal hypertension in the absence of cirrhosis [5]. For direct diagnosis, parasitological examination of the stool did not reveal any bilharzia eggs. In advanced forms of bilharzia, eggs are rarely found [6].

Bilharzia serology was not performed because it was unavailable in our practice setting, limiting indirect diagnosis. Bilharzia serology is not always available in referral hospitals [7].

In this patient, abdominal ultrasound revealed signs of periportal fibrosis classified as stage 3 according to Homeida with portal hypertension. The main lesion caused by *Schistosoma mansoni* in the liver is pipe-like fibrosis or "Symmers' fibrosis," which is highly suggestive of hepatosplenic bilharzia [8]. Several other ultrasound classifications are mentioned in the literature, including those of Cairo, Niger, and Abdel-Wahab, all of which have been harmonized by the WHO as part of the Niamey protocol [8].

However, hepatomegaly was absent, as was dilation of the portal vein and splenic vein. In advanced stages, periportal fibrosis can reduce the diameter of the portal vein, and the hepatomegaly often present at the onset is no longer found.

Liver damage occurs within a variable time frame depending on the severity of the infection.

Anti-schistosomiasis treatment was initiated with praziquantel: 75 mg/kg in three doses per day. Treatment at any stage of the disease is based on praziquantel, even if the lesions appear irreversible in the late stages [9]. The patient has also been receiving beta-blocker treatment to prevent recurrent bleeding associated with ruptured esophageal varices. The patient is monitored regularly and has not experienced any further bleeding.

Conclusion

Hepatosplenic schistosomiasis remains a major public health problem in endemic areas such as Burkina Faso. In countries with limited resources, and particularly in a medium-sized town such as Ouahigouya, diagnosis is often based on a combination of epidemiological, clinical, and radiological evidence.

The case presented illustrates the limitations of biological and anatomopathological examinations, especially for advanced forms, and highlights the role of imaging as a diagnostic tool in our context.

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