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SCHISTOSOMIASIS WITH PERICARDIAL EFFUSION

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ABSTRACT

Schistosomiasis, caused by parasitic flatworms of the genus *Schistosoma*, is endemic in many areas of the world. We report the case of a young male patient presenting with persistent fever, abdominal pain, vomiting and diarrhoea, with travel history to Al Baha, Saudi Arabia, an endemic area for schistosomiasis. Diagnostic imaging revealed hepatosplenomegaly and pericardial effusion. Despite negative stool and blood cultures, the clinical presentation and travel history supported the diagnosis of schistosomiasis. The patient was started on praziquantel and prednisolone therapy, resulting in significant clinical improvement. A follow-up echocardiogram one month later showed complete resolution of the pericardial effusion.

KEYWORDS

Schistosomiasis, *Schistosoma mansoni*, pericardial effusion, hepatosplenomegaly, Katayama fever

LEARNING POINTS

- Pericardial effusion is a rare complication but should be considered in a patient diagnosed with schistosomiasis who presents with tachycardia, tachypnoea and dyspnoea.
- Negative stool culture does not rule out the infection in patients with history of travel to an endemic area with high clinical suspicion.
- Early recognition and management of complications will improve patient outcomes.
- Praziquantel, with or without steroids, is the standard treatment for managing schistosomiasis and its complications.

INTRODUCTION

Schistosomiasis (bilharziasis) is a parasitic infection caused by trematode worms of the genus *Schistosoma*. There are three main species of blood flukes that can cause the infection: *Schistosoma mansoni*, *Schistosoma haematobium* and *Schistosoma japonicum*[1]. Infection occurs when the larvae penetrate the skin during contact with contaminated water[2]. If not detected early, acute schistosomiasis can cause

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systemic complications and, rarely, cardiac complications. In this report, we present a case of *S. mansoni* infection that was complicated by pericardial effusion.

CASE DESCRIPTION

A 19-year-old medically free male patient was referred from another hospital as a case of fever of unknown origin, to rule out systemic diseases and malignancy. He was in his usual state of health until he went to Al Baha and swam in a swamp. At that time, he started to have a skin rash over his hands and feet, and few days later, he developed fever which was associated with abdominal pain, nausea, vomiting and diarrhoea. He went to a hospital in Jubail, 25 days after his symptoms started. The patient was given supportive therapy, improved and was discharged.

He came back seven days after discharge with the same complaint. Further investigations were performed including blood cultures, sputum for acid-fast bacillus and tests for malaria, which were all negative; however, *Brucella* IgM was highly positive. The patient was started on streptomycin, doxycycline and ciprofloxacin to treat brucellosis, but he was not compliant with his medication. He was then referred to our hospital, which was two months after the initial presentation.

On presentation to our hospital, he complained of fever and on-and-off back pain for the previous two months associated with nausea, vomiting and diarrhoea. He had no history of sexual contact, use of alcohol or illicit drugs. Upon admission, he looked ill but was alert and conscious, and his vital signs were stable except for fever measuring 39.3°C. Cardiovascular and respiratory examination were unremarkable. Hepatosplenomegaly was noted on abdominal examination associated with severe tenderness around the umbilical region. Full septic workup was done and revealed high CRP (240.4 mg/l), high ESR (61 mm/hr), high procalcitonin (0.276 ng/ml), high LDH (243.589 U/l), eosinophilia (10.6%, 0.632 $\times 10^3$/µl), lymphocyte count (18.4%, 1.09 $\times 10^3$/µl) and low complements (C 0.36 g/l, C4 0.079 g/l) *(Fig. 1)*.

Liver function tests showed low albumin (24.2 g/l), high ALP (208.4 U/l), high GGT (87 U/l), low AST (14.3 U/l), elevated bilirubin (total: 25.4 µmol/l, conjugated: 10.36 µmol/l) and prolonged coagulation profile (APTT:37.8 sec, PT:16.5 sec, INR:1.51). Electrolytes panel showed: hypocalcaemia (2.04 mmol/l), hypomagnesaemia (0.67 mmol/l), hypokalaemia (3.3 mmol/l), hyponatraemia (133 mmol/l) and low uric acid (173 µmol/l). The viral profile was negative for COVID-19, hepatitis and HIV. Urine, blood and stool cultures were all negative. Malaria screening was negative.

The patient was initially treated for brucellosis, based on the previous hospital report. Two days after admission, he still looked ill and was spiking fever. Brucella titres in our hospital were negative; he was started on meropenem injection 1g q8. Abdominal X-ray showed dilated gas-filled bowel loops. A chest X-ray showed cardiomegaly, perihilar congestion and hepatomegaly *(Fig. 2)*. A high-resolution

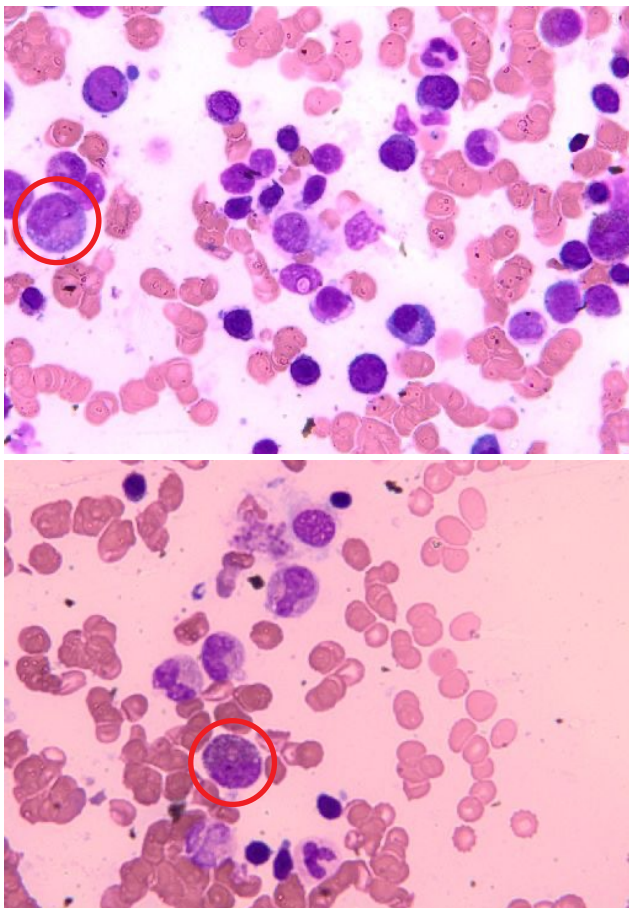


Figure 1. Peripheral blood smear showing leukocytosis with eosinophilia (red circles).

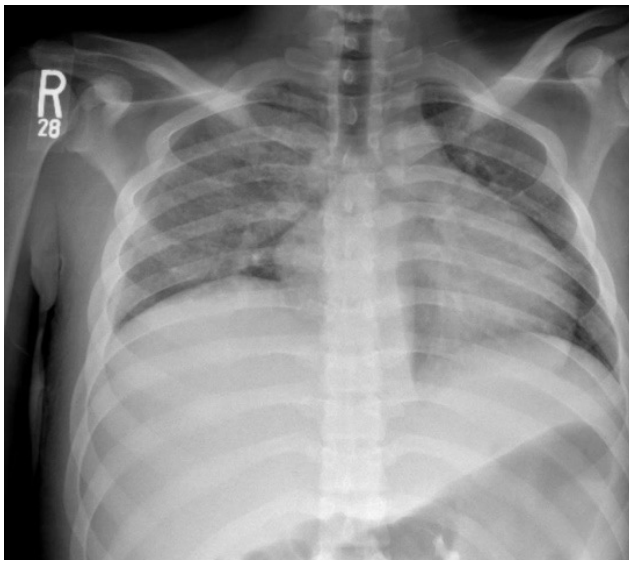


Figure 2. Chest X-ray showing cardiomegaly.

computed tomography scan of the chest showed bilateral pleural effusion with perilymphatic micronodules in the subpleural region, bronchovascular bundles and pericardial effusion *(Fig. 3)*. Computed tomography of the abdomen showed hepatosplenomegaly, transmural thickening with submucosal oedema and increased mucosal enhancement in distal ileal loops, distal part of descending colon, sigmoid and rectosigmoid as well as the proximal rectum with sizeable ascites. The patient developed tachycardia, and

an echocardiogram showed moderate circumferential pericardial effusion with no significant increase in intrapericardial pressure, and no wall motion abnormality *(Fig. 4)*.

Despite three stool cultures and analysis that were negative for ova and parasites, Katayama fever was strongly suspected due to the patient's presentation and his travel history to an endemic area, and his hepatosplenomegaly and eosinophilia. Therefore, the patient was started on prednisolone 20 mg PO OD and praziquantel 600 mg PO four tablets, then another four tablets after 4 hours. The symptoms started to improve after 48 hours, and the patient became haemodynamically and vitally stable. Eventually, the fourth stool culture turned positive for *Schistosoma mansoni* ova *(Fig. 5)*. Schistosoma serology was not readily available at that time due to logistic constraints but eventually came back positive for *S. mansoni*. The patient underwent therapeutic ascitic tap, with a total of approximately 1 litre drained, which showed low serum-ascites albumin gradient of 0.5 g/dl, and high mixed cellularity with predominance of eosinophils count at 55%. Sigmoidoscopy showed inflamed oedematous rectosigmoid mucosa; biopsies were taken and histopathology showed moderate chronic colitis with *S. mansoni* ova. Bronchoscopy was unremarkable and bronchial wash cytology was negative for *Schistosoma* ova and malignant cells. The patient started to improve on the treatment, and he was discharged after 17 days of admission on a tapering dose of prednisolone. Two weeks after discharge, he came for follow up. The patient was vitally and clinically stable, had gained 3 kg, however complained of mild generalised weakness. One month later, an echocardiogram was done and showed resolved pericardial effusion with normal systolic function of both ventricles. A repeat ultrasound of the liver at 6 months showed a normal liver.



Figure 3. CT chest showing nodular infiltrates along with pleural and pericardial effusion.

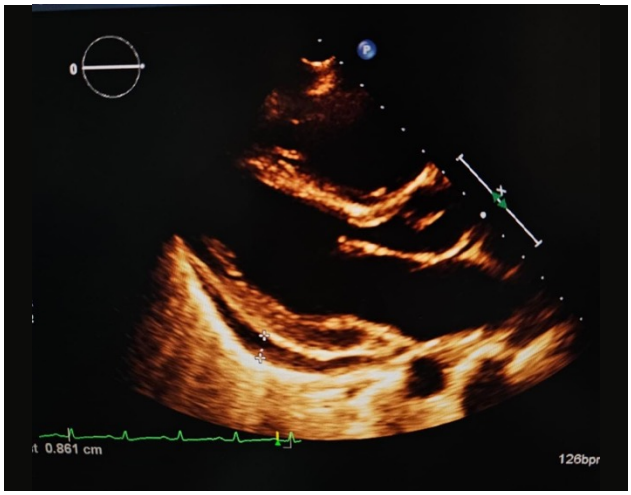


Figure 4. Echocardiogram showing pericardial effusion.



Figure 5. Stool culture showing Schistosoma mansoni egg.

DISCUSSION

Schistosomiasis is a common parasitic disease which can present as acute or chronic infection caused by different species, most commonly *S. mansoni*, *S. haematobium* and *S. japonicum*[1,2]. The infection occurs when the host's skin is penetrated by larvae during contact with contaminated water[2].

Although schistosomiasis has significantly decreased in Saudi Arabia due to proper elimination programmes, it remains prevalent in some areas. Moreover, our data reveal that both clinical forms of schistosomiasis (intestinal and urinary) are reported in Saudi Arabia. Al Baha city, to where our patient had travelled, is considered to be one of the affected western areas, along with Mecca and Aseer. Meanwhile, fewer cases were reported in the central, northern and eastern regions. As in our case, *S. mansoni* is the most reported species in the western region[3].

Cardiopulmonary complications of bilharziasis are considered rare and were first described in the early 1900s. It was mostly linked to the hepatosplenic form of the infection, in which portal hypertension and portosystemic shunting results from presinusoidal obstruction caused by embolised worm eggs[4]. Most of the cardiac manifestations reported include pulmonary hypertension and cor pulmonale, myocarditis, pericarditis and myocardial ischaemia[5,6]. One study had reported pericardial tamponade secondary to chronic schistosomiasis[7]. We believe that the limited literature on pericardial effusion is likely attributable to significant underreporting, due to low clinical suspicion, lack of essential diagnostic method or the fact that the effusion improves after starting the patient on proper management of schistosomiasis. Literature has shown that

pulmonary nodules could be found among patients with schistosomiasis[8], which is the case in our patient.

Acute *S. mansoni* infection should be considered in a patient who has come from an endemic area and presents with persistent fever, abdominal pain, vomiting, diarrhoea and eosinophilia, even if stool culture results are negative. Early recognition of systemic complications is important as it allows for timely intervention, prevents further deterioration and improves patient outcomes. Pericardial effusion should be suspected in a patient diagnosed with schistosomiasis presenting with tachycardia, tachypnoea and dyspnoea. An echocardiogram should be performed when there is clinical suspicion of cardiac complications.

Katayama syndrome is a hypersensitivity reaction to *Schistosoma* antigen and circulating immune complexes. This coincides with *Schistosoma* egg production; hence it starts two to twelve weeks after exposure to *Schistosoma* for the first time or heavy reinfection. Symptoms include nocturnal fever, cough, myalgia, headache and abdominal tenderness. Hepatosplenomegaly and diffuse pulmonary infiltrates and nodules are found radiologically, and most cases have eosinophilia and a history of water contact before onset of clinical symptoms; praziquantel shows effectiveness in treating patients with or without steroids[9].

CONCLUSION

This case highlights the importance of considering schistosomiasis in the differential diagnosis of travellers to endemic areas, presenting with fever, abdominal symptoms and systemic complications. We report here pericardial effusion as a complication of systemic disease. Treatment with praziquantel and prednisolone led to complete resolution of symptoms, including pericardial effusion. The initial stool tests were negative, but this does not rule out schistosomiasis. Travel to endemic area, with fever and abdominal symptoms, hepatosplenomegaly and eosinophilia, as in our case, should raise the possibility of Katayama fever.

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