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Compounded Hepatic Insults: The Convergence of Schistosomiasis, Prior Hepatitis B Exposure, and Autoimmune Hepatitis

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ABSTRACT

Schistosomiasis, a parasitic infection endemic to sub-Saharan Africa, is an uncommon but significant cause of liver disease in developed countries, particularly when compounded by additional hepatic insults. This case report describes a 71-year-old male with severe liver cirrhosis resulting from multifactorial etiologies, including chronic schistosomiasis, viral hepatitis, possible autoimmune hepatitis, and monoclonal gammopathy of undetermined significance (MGUS). Despite initial praziquantel therapy, incomplete treatment allowed for ongoing granulomatous inflammation and fibrosis. The patient's clinical presentation included portal hypertension, recurrent variceal bleeding, and profound anemia. This case highlights the complex interplay of multiple hepatic insults, the critical need for adherence to antiparasitic therapy, and the challenges of managing overlapping liver pathologies. Increased awareness of schistosomiasis and its role in liver disease is vital for timely diagnosis and intervention, particularly in patients from endemic regions.

Keywords: Parasite infection; Granulomatous inflammation; Viral hepatitis; Autoimmune hepatitis; schistosomiasis

INTRODUCTION

Liver disease is a complex and multifactorial condition with significant implications for global health. Its etiologies span a broad spectrum, including viral infections, metabolic disorders, autoimmune processes, and parasitic diseases. Among these, parasitic infections such as schistosomiasis often receive less attention despite their widespread impact. Schistosomiasis, caused by Schistosoma species, affects an estimated 200 million people globally, with over 700 million living in at-risk areas, predominantly in sub-Saharan Africa, the Middle

East, Egypt, and parts of South America and Asia.[1] Chronic infections with Schistosoma mansoni, Schistosoma haematopium, or Schistosoma japonicum frequently lead to hepatosplenic disease characterized by portal hypertension, fibrosis, and in severe cases, cirrhosis. Despite its endemicity in specific regions, the increasing movement of populations through migration and travel has brought schistosomiasis into the clinical landscape of non-endemic areas, presenting diagnostic challenges for healthcare providers unfamiliar with its presentation and progression.[2]

The pathogenesis of schistosomal liver disease is distinct from other forms of liver dysfunction, as it results primarily from the host's immune response to parasite eggs lodged in the portal venous system.[3] Over time, this granulomatous inflammation triggers periportal fibrosis, vascular remodeling, and portal hypertension. However, schistosomiasis does not occur in isolation, particularly in patients with additional hepatic insults. Coexisting conditions, such as chronic viral hepatitis or autoimmune hepatitis, can significantly accelerate liver damage by compounding inflammatory and fibrotic processes. Viral hepatitis, particularly hepatitis B and C, plays a significant role in accelerating liver damage in co-infected individuals. Even prior exposure to hepatitis B, as evidenced by serologic markers, may contribute to liver fibrosis through residual immune dysregulation and persistent inflammatory processes. This overlap creates unique diagnostic and therapeutic dilemmas, as shown in this report, as it can obscure the primary etiology of liver disease and necessitate tailored, multifaceted treatment strategies.

Despite advances in antiparasitic therapies, schistosomiasis continues to pose significant challenges in management, particularly in cases of incomplete treatment. While praziquantel remains the cornerstone of therapy, it is often insufficient to reverse established fibrosis or prevent the progression of chronic liver disease. Furthermore, gaps in patient adherence, diagnostic delays, and coexisting conditions exacerbate outcomes, making timely and comprehensive management critical. The role of public health interventions in endemic areas and among high-risk populations cannot be overstated, as early screening and treatment remain the most effective tools in preventing long-term complications.[4]

This case report presents a rare and multifaceted example of severe liver cirrhosis in a patient with chronic schistosomiasis complicated by viral hepatitis and suspected autoimmune hepatitis. The case underscores the complex interplay of parasitic, viral, and autoimmune factors in liver dysfunction and highlights the importance of recognizing less common etiologies of hepatic disease in patients with relevant travel history or epidemiologic risk factors. By examining this case, we aim to illuminate the diagnostic and therapeutic challenges posed by multifactorial liver disease and provide insights that may guide clinicians facing similar presentations in increasingly globalized healthcare settings.

CASE PRESENTATION

A 71-year-old male of Ethiopian origin presented with signs of decompensated cirrhosis. The patient’s height was 161 cm (5’3”), and his weight was 56.7 kg (125 lbs), corresponding to a body mass index (BMI) of 22 kg/m², within the normal range. His medical history revealed a recent 3-month stay in Ethiopia, after which he

was diagnosed with chronic schistosomiasis. A biopsy of a cecal lesion confirmed the presence of schistosome eggs surrounded by granulomatous inflammation. The histological features of the ova were used to identify them as belonging to the Schistosoma species, specifically mansoni (Table 1). The patient received a single dose of praziquantel 3400 mg, but failed to complete the recommended second dose. The dose was calculated by giving 20 mg/kg orally, given 3 times in a single day.

Schistosoma Species	Ovum Shape	Key Features	Description
Schistosoma mansoni	Oval with a large lateral spine	Prominent spine located on the side of the ovum	The ova are elongated with a noticeable spine on one side, aiding in its identification.
Schistosoma haematobium	Oval with a large terminal spine	Prominent spine located at one pole (end) of the ovum	These ova are more elongated, with a spine at the terminal end, often found in urine samples.
Schistosoma japonicum	Round to slightly oval, small lateral spine	Smaller and more subtle lateral spine compared to others	The ova are more rounded, with a small, barely visible spine, often requiring closer examination.

Table 1: Morphological Characteristics of Schistosoma Ova
This table summarizes the distinct morphological features of Schistosoma ova from the three main species: Schistosoma mansoni, Schistosoma haematobium, and Schistosoma japonicum. Each species has characteristic ovum shapes and spine placement that aid in their identification during microscopic examination.

Laboratory findings also revealed the patient had elevated anti-smooth muscle antibodies, raising suspicion for autoimmune hepatitis. Furthermore, he had positive hepatitis B core IgG antibodies, suggesting past exposure to hepatitis B. However, the patient tested negative for hepatitis B surface antigen (HBsAg), effectively ruling out active or chronic hepatitis B infection. While hepatitis B DNA PCR testing was not performed to assess for potential low-level viral replication, the absence of HBsAg makes significant ongoing viral activity unlikely. Hepatitis C serologies were negative, excluding co-infection with this virus. Based on these findings, active viral hepatitis was not present, and the patient's liver dysfunction was more likely attributable to schistosomiasis, autoimmune hepatitis, and other contributing factors.

Imaging studies, including CT scans of the abdomen and pelvis, demonstrated a nodular liver contour consistent with cirrhosis, but no focal hepatic lesions (Figures 1 and 2). Ultrasound imaging further supported these findings, showing a nodular liver contour consistent with cirrhosis and an absence of focal hepatic lesions (Figure 3). Serial colon biopsies over several years showed chronic inflammation and granulomatous changes indicative of chronic schistosomiasis.

The patient's clinical course was complicated by multiple hepatic insults, each supported by diagnostic findings. Chronic schistosomiasis was confirmed through the cecal biopsy demonstrating Schistosoma mansoni eggs with granulomatous inflammation, as well as a rectal biopsy showing similar findings. Prior exposure to hepatitis B was evidenced by positive hepatitis B core IgG antibodies, although active or chronic infection was ruled out by a negative HBsAg test. Autoimmune hepatitis was suspected based on the elevated anti-smooth muscle antibodies, indicative of an autoimmune process contributing to hepatic inflammation. The patient also had a

history of metabolic dysfunction-associated steatohepatitis (MASH), diagnosed through prior imaging studies and liver enzyme abnormalities. Additionally, monoclonal gammopathy of undetermined significance (MGUS) was identified, adding a layer of immune dysregulation that may have influenced the progression of liver damage.

On presentation, the patient exhibited complications consistent with advanced liver disease, including portal hypertension, splenomegaly, and recurrent gastrointestinal bleeding from esophageal varices and arteriovenous malformations, as confirmed by endoscopy. During his most recent hospitalization, profound anemia was noted, with hemoglobin levels as low as 5 g/dL, necessitating repeated transfusions, with a total of four units of packed red blood cells administered to stabilize his condition. The combination of biopsy findings, autoimmune serology, and imaging results supported the diagnosis of multifactorial liver disease involving chronic schistosomiasis, autoimmune hepatitis, and the sequelae of prior hepatitis B exposure.

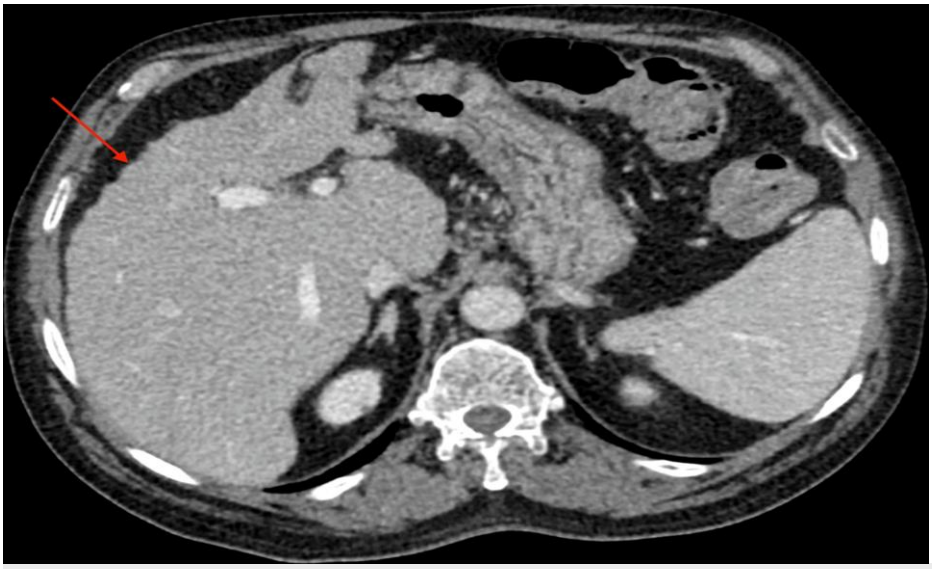

FIGURE 1: Axial CT of the abdomen and pelvis with IV contrast

CT of the abdomen and pelvis with IV contrast, in axial reformats, demonstrates a nodular liver contour consistent with cirrhosis. No focal hepatic lesion is visualized.

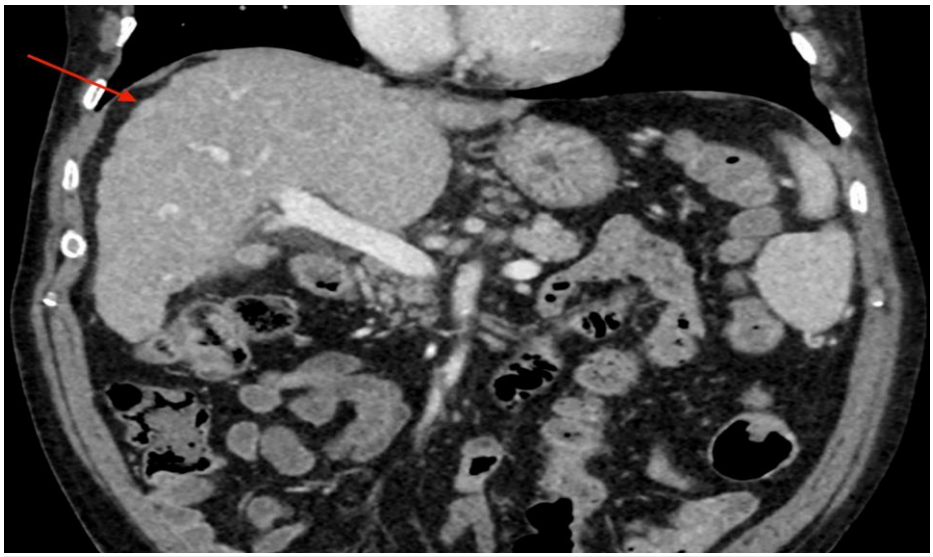

FIGURE 2: Coronal CT of the abdomen and pelvis with IV contrast

CT of the abdomen and pelvis with IV contrast, in coronal reformats, demonstrates a nodular liver contour consistent with cirrhosis. No focal hepatic lesion is visualized.

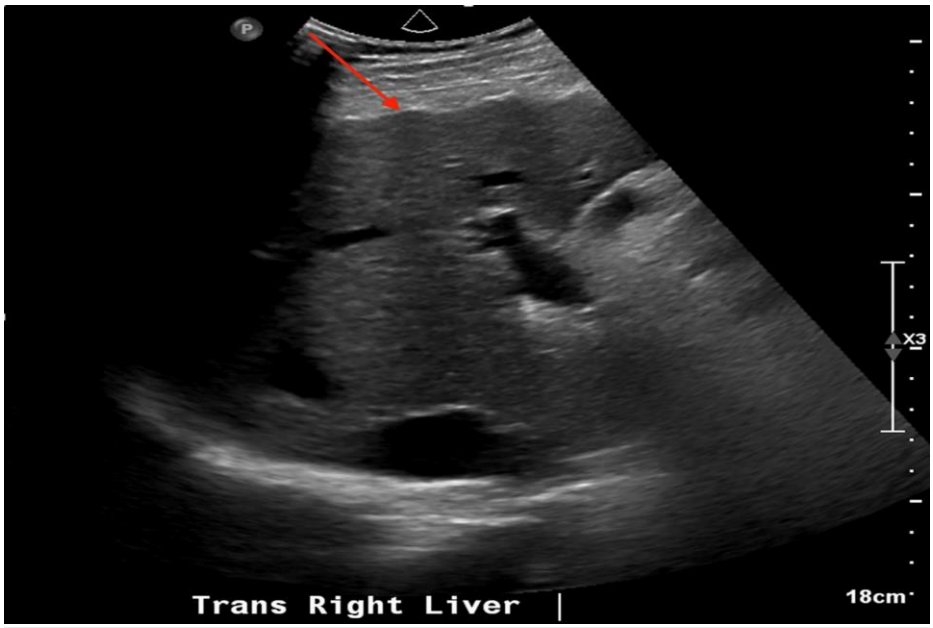

FIGURE 3: Ultrasound of the abdomen

Ultrasound of the abdomen demonstrates a nodular contour of the liver consistent with cirrhosis. No focal hepatic lesion is identified.

DISCUSSION

This case underscores the prolonged and multifaceted hepatic complications of inadequately treated schistosomiasis. Schistosomal liver disease typically arises from granulomatous inflammation in response to parasite eggs lodged in the portal venous system. This chronic inflammatory process can lead to periportal fibrosis, commonly referred to as "pipe-stem" fibrosis in advanced cases.[4] Interestingly, this patient's

presentation deviated from the classic fibrosis pattern and instead reflected a spectrum of hepatic dysfunction influenced by additional factors.

Schistosomiasis-related liver damage results primarily from the host immune response to schistosome eggs trapped in the portal system. Granulomas form as part of this immune reaction, leading to chronic inflammation and fibrosis. Over time, this process may cause significant vascular remodeling, including portal hypertension, as seen in this patient. The absence of ascites, a common finding in schistosomal cirrhosis, highlights the variability in clinical manifestations. This atypical presentation underscores the need for heightened clinical suspicion, particularly in patients from endemic areas.

The presence of MGUS in this patient further complicates the clinical picture. MGUS, a premalignant plasma cell disorder, is associated with an altered immune landscape that could influence the progression of hepatic inflammation and fibrosis. Although not directly linked to schistosomiasis or hepatitis, MGUS may impair hepatic microcirculation and promote a pro-inflammatory state, potentially exacerbating liver dysfunction. While there was no direct evidence from imaging or histology to suggest that MGUS independently affected the liver, its presence likely amplified the immune dysregulation associated with concurrent schistosomiasis and autoimmune hepatitis. This highlights the complex interplay of multiple factors contributing to the progression of the patient's liver disease.

The previous exposure to hepatitis B virus may have played a role in exacerbating the patient's liver damage, despite the absence of an active infection at the time of presentation. Hepatitis B virus is a known cause of cirrhosis and hepatocellular carcinoma, and its long-term effects, even in resolved infections, can include residual immune activation and subclinical fibrosis. In cases of prior exposure, the interaction between lingering immune dysregulation from hepatitis B and schistosomal granulomas may potentiate inflammation and fibrosis. [5] Studies have demonstrated that individuals with a history of coinfection with schistosomiasis and hepatitis B are at risk of accelerated liver fibrosis progression compared to either condition alone. The immune alterations associated with previous hepatitis B infection, including changes in cytokine profiles and T-cell function, may have contributed to amplifying the granulomatous response in this patient, compounding liver damage.[6]

A comprehensive review by Abruzzi et al. highlights the synergistic effects of coinfections involving schistosomiasis and viral hepatitis (HBV or HCV). While schistosomiasis does not inherently increase susceptibility to HBV or HCV, it significantly worsens the clinical trajectory when coinfection occurs, even in cases of prior exposure to viral hepatitis. Notably, patients with a history of Schistosoma-HBV coinfection may experience prolonged immune activation and residual fibrosis, increasing the likelihood of accelerated liver damage compared to those with mono-infection. Similar findings have been observed in Schistosoma- HCV coinfections, where reduced viral clearance and rapid progression of fibrosis were noted.[7] These observations highlight the importance of recognizing prior viral hepatitis exposure as a potential contributor to liver disease progression, particularly in patients from endemic regions or those presenting with multifactorial hepatic insults.

Additional evidence of the complexities arising from coinfections is provided by Ranalli et al., who documented an unexpected finding of liver schistosomiasis in a patient undergoing evaluation for HBV- related chronic hepatitis. The case highlights how pathological findings, such as granulomas and eosinophilic infiltration in liver biopsies, can reveal previously undiagnosed Schistosoma infections in patients with established viral hepatitis. This co-infection resulted in enhanced inflammatory responses localized around schistosomal eggs, compounding hepatic injury. The authors emphasize the diagnostic challenges posed by migratory populations and the importance of a multidisciplinary approach to liver disease evaluation, particularly in non-endemic areas.[8]

Furthermore, the possibility of autoimmune hepatitis, suggested by elevated anti-smooth muscle antibodies, adds another layer of complexity to the immune-mediated damage observed in this patient. Autoimmune hepatitis, characterized by a loss of tolerance to liver antigens, can result in chronic inflammation and progressive fibrosis. In this case, the overlap of autoimmune and parasitic liver disease highlights the challenges in distinguishing and managing concurrent hepatic insults.

Incomplete treatment of schistosomiasis in this patient likely allowed for continued egg deposition and granulomatous inflammation, driving progressive hepatic damage. Praziquantel, the standard treatment for schistosomiasis, effectively targets adult worms but has limited efficacy in reversing established fibrosis.
This highlights the importance of early diagnosis and adherence to the full treatment regimen. Delayed or incomplete treatment increases the risk of chronic complications, as seen in this case.

The management of schistosomiasis-related liver disease requires a multidisciplinary approach, particularly in cases complicated by coexisting conditions. Antiparasitic therapy remains the cornerstone of treatment, but adjunctive strategies, such as immunomodulatory therapies or antifibrotic agents, may be beneficial in reducing long-term hepatic damage.[9] For patients with advanced fibrosis or cirrhosis, liver transplantation may be the only definitive treatment. However, transplantation in the context of active parasitic infection poses unique challenges, including the risk of disease recurrence and postoperative complications.

This case exemplifies the complex interplay of schistosomiasis, previous viral hepatitis, autoimmune processes, and MGUS in the progression of liver disease. By incorporating findings from Abruzzi et al. and Ranalli et al., the broader implications of coinfections in exacerbating liver damage are made clear. This underscores the importance of comprehensive evaluation and tailored management strategies, particularly in patients with multifactorial hepatic insults. Awareness of schistosomiasis as a potential etiology for liver disease, particularly in individuals with pertinent travel history, is essential for improving outcomes and mitigating long-term complications.

CONCLUSIONS

This case highlights the complex interplay of multiple hepatic insults, including schistosomiasis, prior hepatitis B exposure, autoimmune hepatitis, and MGUS, contributing to progressive liver dysfunction. While the patient

did not have active viral hepatitis, past exposure to hepatitis B likely played a role in exacerbating liver damage through residual immune dysregulation. The pathogenesis of liver disease in this case underscores the importance of detailed diagnostic evaluation, including serology, biopsy, and imaging, to unravel multifactorial hepatic conditions. The challenges posed by incomplete schistosomiasis treatment, compounded by overlapping pathologies, emphasize the need for timely and comprehensive management strategies. Recognizing the impact of coexisting or prior hepatic conditions, even when not active, is critical for tailoring treatment and improving outcomes. Increased awareness of these interactions, particularly in patients from endemic regions, is essential in addressing the global burden of liver disease.

Prevention at the community level remains a cornerstone in mitigating the impact of schistosomiasis and other related conditions. Strategies such as mass screening and treatment programs, ensuring safe water supplies for all populations, and promoting early diagnosis and treatment of at-risk children and adults are vital. Health education is central to these interventions, raising awareness about disease prevention and fostering community engagement in controlling the spread of parasitic and viral infections.

This case serves as a reminder of the diagnostic and therapeutic complexities associated with multifactorial hepatic insults and the importance of considering all contributing factors in a patient's clinical history.
Future research should focus on the long-term impact of past viral hepatitis infections and their interaction with parasitic and autoimmune liver diseases to guide more effective management and prevention strategies.

ADDITIONAL INFORMATION

DISCLOSURES

Human subjects: All authors have confirmed that this study did not involve human participants or tissue. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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