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RESEARCH ARTICLE

When Worm Meets Virus: Gallbladder Ascariasis with Acute Hepatitis A – A Rare Co-Infection

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Abstract: Gallbladder ascariasis is a rare manifestation of helminthic infection, primarily due to the narrow, tortuous anatomy of the cystic duct that prevents worm migration. Concurrently, acute Hepatitis A virus (HAV) infection can lead to hepatocellular injury and, less commonly, gallbladder dysfunction. We report a unique case of a 27-year-old male presenting with right hypochondrial pain, vomiting, jaundice, and elevated liver enzymes. Investigations revealed eosinophilia and positive anti-HAV IgM, suggesting acute hepatitis A. MRCP identified a tubular hypointense structure within the gallbladder, indicative of Ascaris lumbricoides. The patient was managed conservatively with albendazole and supportive care. This case underscores the importance of recognizing rare co-infections in endemic regions, where both parasitic infestations and viral hepatitis are prevalent. It highlights the diagnostic challenges and therapeutic implications of dual gallbladder pathology. To our knowledge, this is among the few reported cases documenting gallbladder ascariasis in conjunction with acute HAV infection, verified through imaging and serology.

Keywords:

INTRODUCTION

Hepatobiliary infestation by Ascaris lumbricoides is a well-recognised phenomenon in endemic regions, yet its presentation in the gallbladder remains exceptionally rare. Although the large adult worm predominantly occupies the small intestine, in some cases it migrates into the biliary tract, causing biliary colic, cholangitis, cholecystitis or even acute pancreatitis [1]. Gallbladder ascariasis is defined as worm presence within the gallbladder lumen [2]. The anatomical constraints of the cystic duct, characterised by its narrow and tortuous course, make gallbladder invasion by Ascaris particularly unlikely, further emphasising the rarity of such a diagnosis [3].

In parallel, acute infection by Hepatitis A virus (HAV) is a common cause of self-limited acute hepatitis, especially in young adults in countries with intermediate sanitation standards [4]. While the hallmark of HAV is hepatocellular inflammation and cholestasis, extra-hepatic manifestations—including gallbladder involvement such as acalculous cholecystitis or gallbladder hydrops—have been documented, albeit rarely [5]. The proposed mechanism involves bile stasis, gallbladder wall oedema and impairment of cystic duct flow in the setting of viral hepatopathy.

The coexistence of gallbladder ascariasis and acute HAV infection presents a unique diagnostic and therapeutic challenge. To our knowledge, such dual pathology is extremely uncommon in the literature, as the pathophysiologies of helminthic biliary migration and viral biliary involvement converge in a rare anatomical niche. The parasitic invasion may mimic or exacerbate viral-related biliary dysfunction, and conversely the acute viral inflammatory milieu may predispose to or complicate worm migration and biliary obstruction. In regions where both helminthiasis and viral hepatitis are endemic, clinicians must maintain a high index of suspicion for overlapping aetiologies in patients presenting with hepatobiliary symptoms.

We report here a case of a 27-year-old male who presented with right hypochondrial pain, intermittent vomiting of food particles, eosinophilia, elevated hepatic transaminases and a positive anti-HAV IgM. Imaging revealed tubular echogenic structures within the gallbladder lumen. This case underscores the importance of recognising the possibility of dual pathology—gallbladder ascariasis with concomitant acute hepatitis A—and discussing diagnostic, therapeutic and prognostic considerations in such an unusual setting.

RESULTS AND OBSERVATIONS:

A 27-year-old male presented to the outpatient department with complaints of dull, aching pain in the right hypochondrium for one week. The pain was intermittent, non-radiating, and worsened with food intake. The patient

also reported episodes of nonbilious vomiting for the past two days, with 2–3 episodes per day, usually occurring after meals.

Clinical history revealed prior episodes of similar abdominal pain and vomiting over the previous year. Additionally, the patient gave a history of high-colored urine and yellowish discoloration of the eyes for the past two days, suggestive of jaundice. On examination, icterus was present, and abdominal examination revealed tenderness in the right hypochondrium and epigastric regions. No palpable mass or organomegaly was noted.
Laboratory investigations showed elevated total and direct bilirubin levels (Total: 5.4 mg/dL, Direct: 4.98 mg/dL), along with significantly raised liver transaminases (SGOT: 1325 IU/L, SGPT: 1548 IU/L), suggesting acute hepatocellular injury. The eosinophil count was elevated (9.8%), and the Hepatitis A IgM marker tested reactive, confirming acute Hepatitis A infection.

Ultrasound of the abdomen revealed no structural abnormalities in the liver or gallbladder. However, magnetic resonance cholangiopancreatography (MRCP) demonstrated a curvilinear, tubular hypointense structure within the gallbladder lumen, indicative of gallbladder ascariasis.

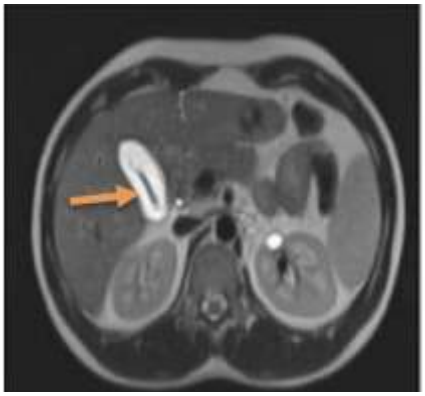

Figure 1: Curvilinear, tubular hypointense structure is noted in gall bladder lumen s/o gall bladder ascariasis

Figure 2: Ultrasound done after treatment showed empty gall bladder

The patient was managed conservatively with a single dose of Albendazole 400 mg and supportive care. Follow-up after treatment showed normalization of liver function tests and resolution of symptoms.

This case highlights a rare co-infection of gallbladder ascariasis with acute Hepatitis A, emphasizing the need for imaging modalities such as MRCP for accurate diagnosis in patients presenting with atypical hepatobiliary symptoms in endemic areas.

DISCUSSION

The presented case of gallbladder involvement in which both Ascaris lumbricoides induced gallbladder infestation and acute Hepatitis A virus (HAV) infection occurred concurrently offers a unique clinical picture, which warrants discussion on several fronts: pathophysiology, diagnosis, management, and what sets this case apart from the existing literature.

Helminthic migration of Ascaris lumbricoides into the hepatobiliary system is well described, particularly in endemic regions. The worms typically enter through the ampulla of Vater, travel up the common bile duct (CBD), and occasionally into intrahepatic ducts or the gallbladder. However, migration into the gallbladder is rare because the cystic duct is narrow, tortuous and not a usual path of least resistance. In a systematic review

of gallbladder ascariasis, only 13 case reports specifically documented worm presence in the gallbladder lumen [2]. In one larger series, 28 patients with gallbladder ascariasis were studied, and while gallbladder involvement was noted, many had worms also in the CBD; gallbladder only involvement remained uncommon [6]. Typically, clinical presentations include right upper quadrant (RUQ) pain, vomiting, biliary colic, cholangitis, or cholecystitis [7]. Imaging—especially ultrasound—shows linear, tubular echogenic structures in the gallbladder or biliary tree; active movement ("belly dance sign") has been described [8].

In parallel, acute hepatitis A is a common cause of acute viral hepatitis, particularly in regions with intermediate sanitation [9]. Beyond hepatocellular injury, it has recognized extra hepatic manifestations including gallbladder involvement. Studies of HAV infection report gallbladder wall thickening, distension (hydrops), biliary sludge, and in rare cases acalculous cholecystitis (ACC) or gallbladder hydrops [10]. For instance, one paediatric report described gallbladder hydrops due to HAV infection in a 5 year old boy [5]. The pathophysiologic mechanism postulated includes hepatocellular damage, cholestasis, bile stasis, increased intraluminal pressure, gallbladder wall oedema and sludging—all of which may impair cystic duct outflow and gallbladder motility [10].

In the presented case, the coexistence of these two processes—ascariasis of the gallbladder and acute hepatitis A infection—is noteworthy. From a pathophysiologic standpoint, HAV related bile stasis or gallbladder dysfunction may have predisposed the gallbladder to worm migration and retention (which is otherwise seldom encountered), or conversely, the presence of the worm may have aggravated biliary stasis and hepatic inflow/outflow disturbance. Either way, this interplay between parasite and virus in the gallbladder is unusual and, to our knowledge, scarcely reported.

Several aspects of this case deserve emphasis: While both gallbladder ascariasis and HAV associated gallbladder involvement have individually been described, the simultaneous occurrence of both is exceedingly rare. The Spanish series by Montiel Jarquín et al. (2003) described two cases of gallbladder ascariasis associated with acute hepatitis (non viral) but did not document viral hepatitis A specific serology [11]. To the best of our review, we found very limited or no published cases of gallbladder ascariasis occurring with acute hepatitis A viral infection. This makes the present case unique in bridging parasitic migration and viral hepatopathy in the gallbladder context. The detection of a tubular hypointense structure within the gallbladder on MRCP (as per the case details) represents a higher resolution modality than standard ultrasound, thereby enhancing diagnostic confidence in gallbladder ascariasis. Many prior reports relied solely on ultrasound [8].

In a setting where parasite infestation and viral hepatitis are both prevalent (as in many tropical/sub tropical zones), the presence of jaundice, elevated transaminases and eosinophilia might initially be attributed to one cause only. The recognition that both entities co exist underscores the need for a high index of suspicion. Because gallbladder ascariasis in many cases resolves with conservative therapy (anthelmintic drugs, observation) rather than immediate surgical intervention, recognition of the dual mechanisms may influence management. Without identification of the parasitic component, one might erroneously attribute gallbladder involvement solely to the viral process, potentially delaying targeted anthelmintic therapy.

Demonstrating that in regions where both helminthic infestations and viral hepatitis A are endemic, clinicians should consider the possibility of simultaneous parasitic and viral gallbladder involvement rather than attributing all biliary symptoms to one entity alone. In a patient presenting with RUQ pain, elevated hepatic enzymes, eosinophilia and positive HAV IgM—as in our case—the presence of parasitic gallbladder involvement should still be entertained. Imaging beyond routine ultrasound—such as MRCP—is justified when gallbladder findings or parasite suspicion exist. While HAV related gallbladder changes are typically self limiting and managed conservatively, the addition of anthelmintic therapy becomes relevant when gallbladder ascariasis is documented. Early recognition may avoid unnecessary surgical intervention.

Although gallbladder ascariasis is rare, systematic review finds only about 13 confirmed case reports worldwide up to 2022 [2]. Each additional well documented case expands understanding of its varied presentation, imaging features, natural course, and treatment outcomes. The case suggests potential synergistic pathogenic mechanisms—viral hepatopathy causing biliary stasis, which may facilitate parasitic invasion; or parasitic obstruction provoking hepatocellular stress in the setting of viral infection. While speculative, this interplay opens avenues for further research.

Despite the novelty, some caveats remain. First, the exact chronology of parasitic migration versus viral infection onset remains uncertain; whether the worm preceded viral hepatitis or vice versa cannot be definitively established. Second, while MRCP documented the worm in the gallbladder lumen, stool ova/parasite confirmation and other parasitic investigations may have limitations. Third, the single case nature limits generalisability. From a research standpoint, future case series or registries in endemic regions might help determine how frequently such dual pathology occurs, and whether certain risk factors (such

as heavy worm load, poor sanitation, or cholecystic bile stasis) predispose to this overlap.

Mechanistically, studies could explore whether HAV infection alters bile composition, impairs gallbladder motility, or changes cystic duct resistance, thereby permitting worm lodgment. Conversely, investigations might evaluate whether gallbladder worm presence alters hepatobiliary dynamics and exacerbates viral injury. Practically, clinicians in tropical settings should maintain a low threshold for ultrasound/MRCP when gallbladder findings accompany hepatitis A and should consider empiric anthelmintic therapy when imaging suggests worm migration.

CONCLUSION

The present case illustrates a rare but clinically important scenario of gallbladder ascariasis co existing with acute hepatitis A infection. Its uniqueness lies in the dual mechanism of gallbladder pathology, the imaging confirmation of worm migration into the gallbladder, and the implications this has for diagnosis and management. Awareness of such overlap is critical, particularly in endemic regions, to enable prompt recognition, appropriate imaging, targeted therapy, and avoidance of misattribution of all findings to one pathology alone. As the literature on gallbladder ascariasis remains limited, each additional case such as this contributes valuably to the evolving understanding of this unusual clinical entity.

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