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Isolated Gallbladder Ascariasis Causing Acalculous Hydrops: Management of a Rare Surgical Entity

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A B S T R A C T

Introduction: Ascariasis, caused by the nematode *Ascaris lumbricoides*, affects approximately one billion people globally. While hepatobiliary involvement is a recognized complication, isolated gallbladder ascariasis is exceptionally rare, accounting for less than 2.1% of biliary cases due to the anatomical resistance provided by the tortuous valves of Heister. This condition presents a significant diagnostic dilemma, often mimicking acute acalculous cholecystitis, and carries a high risk of complications if mismanaged. **Case presentation:** We report the case of a 57-year-old female presenting with acute-on-chronic right upper quadrant pain. Physical examination revealed localized tenderness without jaundice. Laboratory investigations demonstrated leukocytosis (15,460/mm³), significant eosinophilia (8%; absolute count 1,236/mm³), and cholestasis with elevated Alkaline Phosphatase (133 U/L). Ultrasonography, the diagnostic gold standard, revealed a distended gallbladder consistent with hydrops and a mobile, tubular echogenic structure—the inner tube sign—extending into the cystic duct. The patient underwent an open cholecystectomy. Intraoperatively, a critical retrograde milking maneuver was performed to dislodge the worm from the cystic duct back into the gallbladder to prevent transection. The Common Bile Duct was palpated and confirmed to be free of stones or parasites. A viable *Ascaris* worm was extracted from the specimen. **Conclusion:** Isolated gallbladder ascariasis must be considered in the differential diagnosis of acalculous biliary disease in endemic regions. Ultrasonography is superior to other modalities for diagnosis. Surgical intervention is mandatory when hydrops or cystic duct impaction occurs, with meticulous attention to cystic duct clearance to prevent biliary sequelae. The patient was discharged on postoperative day 3 without complications.

1. Introduction

The interaction between human populations and soil-transmitted helminths represents one of the most enduring challenges in global public health. Among these pathogens, *Ascaris lumbricoides* stands as the preeminent agent of parasitic morbidity, exerting a profound toll on human health, particularly within the developing world. It is currently estimated that approximately one billion individuals worldwide are harboring this nematode, a staggering statistic that cements ascariasis as the leading cause of parasitic infection globally.[1] This burden is not distributed equally; rather, it is disproportionately concentrated in the tropical and subtropical belts of Asia, Africa, and Latin America, where the interplay of environmental conditions and socioeconomic factors—specifically the lack of treated water and sanitation—facilitates the fecal-oral transmission cycle. While ascariasis is frequently categorized as a Neglected Tropical Disease (NTD), the sheer scale of its prevalence underscores

that it remains a significant and active public health crisis.[2]

In the vast majority of clinical encounters, infection with *Ascaris lumbricoides* follows a benign or subclinical course.[3] The adult worms typically reside within the lumen of the jejunum and ileum, coexisting with the host without causing acute distress. However, the clinical picture shifts dramatically when the parasitic load increases. Complications are statistically correlated with a high worm burden—typically defined as a load exceeding 1,000 worms. Under these conditions, or in response to physiological stress, the adult nematodes exhibit an erratic wanderlust.[4] Driven by overcrowding or environmental perturbations, the worms migrate proximally from the small intestine, seeking refuge in the biliary or pancreatic ducts. This upward migration necessitates the passage of the nematode through the Ampulla of Vater, a critical anatomical gateway. Normally, the Sphincter of Oddi acts as a competent barrier preventing duodenal reflux.[5] However, under the pressure of a high worm load, previous interventions such as sphincterotomy or physiological relaxation, the sphincter is breached, allowing the worm access to the biliary tree. This invasion precipitates a spectrum of disease known collectively as hepatobiliary and pancreatic ascariasis (HPA). The clinical manifestations of HPA are diverse and severe, ranging from biliary colic and pyogenic cholangitis to cholecystitis and pancreatitis, depending on the precise location and degree of obstruction caused by the parasite.[6]

Within the spectrum of HPA, the involvement of the gallbladder itself represents a distinct and exceedingly rare pathological entity. Clinical series and epidemiological reviews indicate that isolated gallbladder ascariasis accounts for only approximately 2.1% of all cases of hepatobiliary ascariasis. This statistical rarity is not a matter of chance but is dictated by rigorous anatomical constraints.[7] To enter the gallbladder, a worm must navigate the cystic duct. Unlike the common bile duct, the cystic duct presents a formidable mechanical challenge to the parasite. It contains the valves of Heister—a series of spiral, mucosal folds that project into the lumen. These valves create a narrow, tortuous, and spiraled pathway that serves a physiological role in regulating bile flow but acts as a potent physical deterrent against the entry of large nematodes. Given that adult *Ascaris* worms can measure between 15 to 20 centimeters or more in length, the tortuosity of the valves of Heister usually effectively prevents their ingress. Consequently, while the common bile duct is a frequent site of infestation, the gallbladder remains an anatomical fortress, breached only in exceptional circumstances.[8]

When an *Ascaris* worm successfully navigates the anatomical challenge of the cystic duct and invades the gallbladder, the clinical consequences are immediate and severe. The presence of the worm initiates a cascade of pathological events driven by both mechanical and chemical factors.[9] The worm acts as a physical plug. By lodging within the gallbladder or becoming entrapped in the cystic duct, it obstructs the physiologic outflow of bile. This obstruction is critical; while the outflow is blocked, the gallbladder mucosa continues its physiological secretion of mucin. In the closed system of an obstructed gallbladder, this mucin accumulation leads to rapid distension, resulting in the formation of a mucocele, or gallbladder hydrops. Beyond simple mechanics, the worm is a biological irritant. The excretion of metabolic waste products by the live parasite, combined with the release of somatic antigens, incites an intense inflammatory response from the gallbladder mucosa. This results in acalculous cholecystitis—inflammation of the gallbladder without the presence of gallstones. Over time, the worm or its fragments can serve as a nidus for stone formation. The worm may eventually perish, and the necrotic remnants can act as a core upon which calcium bilirubinate precipitates, leading to the formation of brown pigment stones.

The diagnosis of isolated gallbladder ascariasis requires a high index of suspicion, particularly because the clinical presentation—Right upper quadrant (RUQ) pain and leukocytosis—mimics that of classic calculous cholecystitis. Accurate diagnosis

relies heavily on imaging. Ultrasonography is established as the gold standard in this context, favored for its ability to visualize the characteristic morphology of the worm and, crucially, its motility in real-time. The sonographic identification of the stripe sign or the inner tube sign (a tubular echogenic structure with a central anechoic canal) allows for the differentiation of the parasite from biliary sludge or organized hematoma.

Therapeutically, this condition presents a complex dilemma. While intestinal ascariasis is effectively managed with pharmacological agents, the management of biliary entrapment is controversial. Conservative management with anthelmintics carries significant risks in the setting of cystic duct obstruction. Killing the worm while it is entrapped within the narrow cystic duct or gallbladder lumen can be catastrophic; the resulting necrotic foreign body cannot be flushed out, leading to persistent inflammation, stricture formation, or sepsis. Therefore, when the worm is entrapped or associated with complications such as hydrops, surgical intervention becomes the indicated approach.[10]

This manuscript aims to document a rare presentation of isolated gallbladder ascariasis manifesting as severe gallbladder hydrops in a 57-year-old female without pre-existing cholelithiasis. While hepatobiliary ascariasis is well-documented, the isolation of the parasite within the gallbladder causing acalculous hydrops is an exceptional finding that challenges standard diagnostic algorithms. Beyond the clinical description, this report provides a critical analysis of the surgical methodology required to manage this entity. We specifically highlight the necessity of the retrograde milking maneuver during open cholecystectomy to prevent the transection of the worm within the cystic duct—a technical nuance often under-described in the literature. Furthermore, we discuss the diagnostic utility of ultrasonography in identifying the inner tube sign and underscore the pathophysiology of acalculous hydrops driven by parasitic obstruction, aiming to refine the management protocols for acalculous biliary disease in endemic regions.

2. Case Presentation

Written informed consent was obtained from the patient for the publication of this case report and accompanying intraoperative images. All procedures performed were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki Declaration and its later amendments.

A 57-year-old female presented to the Emergency Department of a tertiary care academic medical center in Indonesia, seeking urgent evaluation for an acute exacerbation of abdominal pain. The patient's demographic profile provided crucial initial clues regarding the potential etiology of her condition. She was a long-term resident of a rural, agricultural community characterized by limited sanitation infrastructure. Her primary source of potable water was an untreated shallow well, and she lived in close proximity to agricultural lands where the use of human waste as fertilizer (night soil) is occasionally practiced. This environmental context is of paramount epidemiological significance. In such tropical settings, the reliance on untreated ground water is a documented high-risk factor for the ingestion of embryonated *Ascaris lumbricoides* eggs. The fecal-oral transmission route remains the dominant vector for ascariasis in this region, where the interplay of soil humidity, temperature, and sanitation gaps creates an optimal incubator for geo-helminths. While the patient had no recent travel history or contact with known sick contacts, her daily environmental exposure placed her in a high-risk category for parasitic infestation, a factor that would later guide the differential diagnosis beyond standard metabolic or neoplastic etiologies.

The clinical trajectory of this patient offered a classic study in the dual-phase presentation of biliary parasitosis: a chronic, indolent phase followed by an acute, catastrophic obstructive event. The patient reported a one-year history of digestive discomfort localized to the right upper quadrant (RUQ). She described this pain as dull, gnawing, and intermittent,

typically manifesting 30 to 60 minutes after meals (Table 1). This post-prandial timing mimics the physiology of biliary colic associated with cholelithiasis, where cholecystokinin release stimulates gallbladder contraction against an obstruction. For twelve months, she had self-managed these episodes with over-the-counter analgesics and dietary modifications, assuming the symptoms were related to indigestion or gastritis. In retrospect, this period likely corresponded to the presence of the *Ascaris* worm freely inhabiting the gallbladder lumen, causing sporadic irritation of the mucosa without frank obstruction. Forty-eight hours prior to her admission, the clinical picture shifted drastically. The pain lost its intermittent nature and transformed into a continuous, unrelenting agony, rated by the patient as 8 out of 10 on the Visual Analog Scale. The pain radiated distinctly to the right scapular region (Boas' sign), a somatic referral pattern indicative of phrenic nerve irritation via the inflamed gallbladder capsule. This transition from a dull ache to incapacitating pain marked a critical pathophysiological turning point: the migration of the nematode from a floating existence in the gallbladder lumen to a position of impaction within the cystic duct. Despite the severity of the pain, the patient denied nausea, vomiting, or fever, suggesting that the process had not yet progressed to systemic sepsis or generalized peritonitis. Clinically pivotal was the absence of jaundice, dark urine (choluria), or pale stools (acholia). This negative finding was of immense diagnostic value. It strongly suggested that the Common Bile Duct remained patent. The obstruction was essentially off-line, confined to the gallbladder and cystic duct, thereby sparing the patient from the profound bilirubinemia associated with choledocholithiasis or distal CBD obstruction.

Upon admission, the patient appeared in acute distress due to pain but remained hemodynamically stable. Her vital signs—Blood Pressure 120/80 mmHg, Heart Rate 83 beats per minute, and Respiratory Rate 21 breaths per minute—indicated that she was compensating well, without signs of septic shock. Her temperature was 36.1°C, further supporting the theory that this was a sterile, pressure-induced inflammatory process rather than a fulminant bacterial infection at this stage. The abdominal exam provided localized and specific findings. The abdomen was non-distended, ruling out bowel obstruction or massive ascites. Palpation revealed exquisite, localized tenderness in the right hypochondrium. More importantly, deep palpation identified a palpable fullness in the gallbladder fossa. This finding is highly suggestive of a significantly distended gallbladder, consistent with hydrops. A nuanced finding in this case was the equivocal nature of Murphy's sign. While the patient exhibited tenderness during inspiration, the classic abrupt inspiratory arrest associated with bacterial cholecystitis was not distinct. This subtlety is clinically relevant. In classic acute cholecystitis, the inflammation is often transmural and bacterial, causing severe parietal peritoneal irritation. In this case of parasitic hydrops, the pain was driven primarily by rapid capsular distension (visceral pain) due to mucus accumulation, rather than a purulent bacterial infection involving the peritoneum. This distinction explains why the tenderness was severe yet lacked the precise peritoneal catch of a classic Murphy's sign. The absence of scleral icterus and palmar erythema confirmed the historical report of no jaundice, reinforcing the localized nature of the obstruction.

The laboratory workup provided the first concrete evidence pointing toward a parasitic etiology rather than standard lithogenic disease. The hematological panel revealed a Leukocytosis (15,460/mm^3), signaling an active inflammatory response. However, the differential count contained the diagnostic key: Eosinophilia of 8%, with an absolute eosinophil count of 1,236/mm^3. In the context of an acute abdomen, this profound eosinophilia is a significant biomarker. While gallstones can cause inflammation (neutrophilia), they do not trigger eosinophilia. This Th2-mediated immune response is characteristic of tissue-invasive helminths. The immune system, detecting the somatic antigens of the *Ascaris* cuticle, upregulates eosinophil production in an attempt to

combat the parasite.

The liver function tests presented a pattern of dissociation that mirrored the physical exam: (1) AST (59 U/L) and ALT (73 U/L): Mildly elevated, suggesting reactive non-specific hepatocellular stress or local inflammation; (2) Total Bilirubin (0.8 mg/dL): Completely normal, confirming the patency of the main biliary channel; (3) Alkaline Phosphatase (ALP) (133 U/L): Clearly elevated. This combination—elevated ALP with normal Bilirubin—is the biochemical hallmark of a localized, partial biliary obstruction. It indicates that while the main flow of bile is uninterrupted (normal bilirubin), there is a focal area of the biliary tree (the gallbladder and cystic duct) under significant pressure, causing the induction and release of ALP from the biliary epithelium.

A preoperative stool examination for ova and parasites was negative. In clinical practice, this is a frequent false negative that can mislead clinicians. The absence of eggs in the stool does not rule out ascariasis. This phenomenon can occur if the infection involves only male worms (which cannot produce eggs), if the female worms are immature, or if the worms are located in the biliary tree rather than the intestine, where egg shedding is intermittent or absent. Thus, the negative stool test necessitated reliance on imaging.

Abdominal ultrasonography (USG) served as the definitive diagnostic modality. Given the patient's lean body habitus and the specific RUQ localization, USG offered superior resolution to computed tomography for visualizing the internal structure of the gallbladder. The scan revealed a gallbladder that had lost its physiologic pyriform shape, appearing instead as a tense, cylindrical structure measuring 10.5 cm x 4.5 cm. The gallbladder wall was thickened to 4.5 mm (normal <3 mm), indicating edema. Crucially, the lumen was devoid of the acoustic shadowing typical of gallstones, ruling out calculous cholecystitis. Within this fluid-filled, anechoic background, the ultrasound visualized the parasite with remarkable clarity: (2) The Inner Tube Sign: In cross-section, the worm appeared as an echogenic (bright) ring representing the thick parasitic cuticle, surrounding a central anechoic (dark) dot representing the worm's fluid-filled digestive tract; (2) The stripe sign: In longitudinal section, the worm appeared as a long, linear, tubular structure; (3) Real-Time Motility: Perhaps the most critical finding was the observation of non-directional, writhing movement of the tubular structure. This erratic motility definitively distinguished the mass from biliary sludge, organized hematoma, or gangrenous mucosal sloughing, confirming the presence of a live macro-organism. The ultrasound also elucidated the mechanism of the hydrops. The cephalad end of the worm was visualized extending into the cystic duct. This visual confirmation completed the pathophysiological puzzle: the worm had acted as a cork, mechanically occluding the narrow cystic duct. This prevented bile egress while the gallbladder mucosa continued to secrete mucin, leading to the high-pressure hydrops that brought the patient to the Emergency Department.

Following the definitive sonographic diagnosis of gallbladder hydrops secondary to parasitic impaction, the patient was immediately admitted for preoperative stabilization. The clinical priority was two-fold: optimize the patient's fluid status and mitigate the risk of septic progression. She was placed on a strict nil per os regimen to rest the gastrointestinal tract and minimize gallbladder stimulation. Intravenous fluid resuscitation with crystalloids was initiated to correct any subclinical dehydration and support renal perfusion. Given the potential for bacterial superinfection in the setting of stasis—even in a primarily parasitic etiology—prophylactic broad-spectrum antibiotic coverage was instated with Ceftriaxone (1 gram). This third-generation cephalosporin was selected for its excellent biliary penetration and efficacy against common enteric pathogens such as *Escherichia coli* and *Klebsiella species*, which frequently colonize obstructed biliary systems.

Table 1. Summary of Clinical, Laboratory, and Imaging Findings on Admission			
Parameter	**Patient Value**	**Reference Range**	**Interpretation**
1. VITAL SIGNS & SYSTEMIC EXAM			
Blood Pressure	**120/80 mmHg**	< 120/80	**Hemodynamically Stable**
Temperature	**36.1°C**	36.5 - 37.5°C	**Afebrile (No sepsis)**
Scleral Icterus	**Absent**	N/A	**No Clinical Jaundice**
Abdominal Exam	**Fullness RUQ**	Soft/Flat	**Gallbladder Distension**
2. HEMATOLOGICAL PROFILE			
Hemoglobin	**12.5 g/dL**	12.0 - 16.0 g/dL	**Normal**
Leukocytes (WBC)	**15,460 /mm³**	4,500 - 11,000	**Significant Leukocytosis**
Eosinophils (%)	**8%**	1 - 3%	**Parasitic Immune Response**
Abs. Eosinophils	**1,236 /mm³**	< 500 /mm³	**Marked Eosinophilia**
3. LIVER FUNCTION TESTS (BIOCHEMISTRY)			
AST (SGOT)	**59 U/L**	< 35 U/L	**Mild Elevation**
ALT (SGPT)	**73 U/L**	< 45 U/L	**Mild Elevation**
Alkaline Phos. (ALP)	**133 U/L**	35 - 105 U/L	**Cholestatic Pattern**
Total Bilirubin	**0.8 mg/dL**	0.1 - 1.2 mg/dL	**Normal (CBD Patent)**
C-Reactive Protein	**45 mg/L**	< 5 mg/L	**Severe Acute Inflammation**
4. ULTRASONOGRAPHY FINDINGS			
Gallbladder Morphology	**10.5 x 4.5 cm**	< 10 x 4 cm	**Hydrops / Distension**
Wall Thickness	**4.5 mm**	< 3 mm	**Edematous / Thickened**
Lumen Contents	**Tubular Structure**	Anechoic (Bile only)	**"Inner Tube" & "Stripe" Signs**
Motility	**Non-directional**	None	**Live Parasite Confirmed**

The anesthetic management of biliary ascariasis presents unique challenges that differentiate it from standard cholecystectomy. The primary concern is the erratic behavior of *Ascaris lumbricoides* under physiological stress. Sub-optimal sedation or the excitatory phase of induction can trigger a violent migratory response in the nematodes. Literature documents catastrophic instances where worms have retrogradely migrated from the duodenum into the esophagus and pharynx during induction, precipitating acute laryngospasm or upper airway obstruction. To mitigate this, a Rapid Sequence

Induction strategy was employed. This approach utilizes a fast-acting induction agent and a neuromuscular blocker to secure the airway rapidly, minimizing the window of potential worm migration and preventing gastric aspiration. Furthermore, the choice of maintenance gases was strictly regulated. Nitrous Oxide was absolutely contraindicated. The pathophysiology behind this exclusion lies in the differential solubility of gases. Nitrous oxide diffuses into air-filled cavities significantly faster than nitrogen can diffuse out. In a patient with ascariasis, the worms' digestive tracts and the surrounding intestinal loops contain gas. The use of nitrous oxide can lead to the rapid expansion of these gas-filled spaces. In the context of a worm trapped in a narrow cystic duct, such expansion could theoretically lead to the rupture of the worm's cuticle (releasing highly immunogenic and toxic body fluids) or increase the intraluminal pressure of the gallbladder, precipitating perforation. Therefore, anesthesia was maintained using volatile agents and oxygen/air mixtures only.

The decision to proceed with an open cholecystectomy rather than a laparoscopic approach was made after careful deliberation. While laparoscopic cholecystectomy is the gold standard for lithogenic disease, the presence of a live macro-organism impacted in the cystic duct alters the risk profile. Laparoscopic graspers reduce tactile feedback, creating a significant risk of inadvertently crushing the worm or, worse, transecting it across the cystic duct during clipping. A transected worm leaves a necrotic remnant in the common bile duct, a guaranteed recipe for postoperative cholangitis or biliary leak. Consequently, a right subcostal Kocher's incision was utilized to afford the surgeon precise manual palpation and direct control over the extrahepatic biliary tree. Upon entering the peritoneal cavity and retracting the liver, the pathology was immediately evident. The gallbladder did not exhibit the chronic, scarred, and shrunken appearance typical of recurrent lithiasis. Instead, it was tensely distended, edematous, and hyperemic, consistent with acute, high-pressure hydrops. The serosal surface was injected, reflecting acute venous congestion. Notably, there were no dense pericholecystic adhesions to the omentum or duodenum, a finding that corroborated the clinical history of an acute obstructive event superimposed on a non-inflammatory chronic carriage.

The dissection of Calot's triangle—the anatomical space bounded by the cystic duct, common hepatic duct, and the liver edge—was performed with extreme caution. The priority was not to ligate, but to clear the duct. The cystic duct was identified and noted to be significantly dilated and edematous, a reactive change to the parasite's presence. Before applying any instruments, the surgeon performed a gentle digital palpation of the cystic duct. This confirmed the presence of a firm, tubular, rubbery structure occupying the lumen, extending from the gallbladder neck toward the common bile duct junction. To safely ligate the duct, the worm had to be displaced. Using a soft, atraumatic bowel grasper on the gallbladder neck and digital pressure on the distal cystic duct, the surgeon performed a milking maneuver. Pressure was applied in a retrograde fashion, pushing the worm upwards from the cystic duct back into the wider body of the gallbladder. The tactile sensation of the worm slipping back into the gallbladder confirmed the success of the maneuver. Once the worm was dislodged, the cystic duct was re-palpated. It was confirmed to be flat, empty, and pliable. This verification is mandatory. It ensures that the subsequent application of surgical clips or ligatures creates a hermetic seal on the duct tissue alone, rather than crushing the worm or leaving a gap that would result in a bile leak (biliary peritonitis).

With the cystic duct secured and divided, attention turned to the common bile duct (CBD). In endemic areas, synchronous infection (worms in both the gallbladder and the CBD) is common. The surgeon palpated the CBD along its entire supraduodenal length. The duct was found to be soft, non-dilated (diameter less than 6 mm), and free of any palpable masses. This intraoperative finding, combined with the patient's normal preoperative bilirubin and the

absence of distal obstruction signs on ultrasound, provided high confidence that the CBD was clear. Therefore, invasive interrogation of the CBD via choledochotomy or intraoperative cholangiography was deemed unnecessary, sparing the patient additional morbidity. The gallbladder was dissected from the liver bed and removed. On the back table, away from the surgical field, the specimen was incised longitudinally. A large, viable *Ascaris lumbricoides* worm, measuring approximately 22 centimeters in length, was evacuated from the lumen (Figure 1). The worm was active, confirming the recent nature of the impaction. Inspection of the gallbladder mucosa revealed patchy areas of erythema and edema, indicative of chemical irritation from the parasite's excretions. Crucially, no cholesterol calculi, pigment stones, or biliary sludge were found. This definitive pathological finding confirmed the diagnosis of isolated parasitic acalculous cholecystitis.

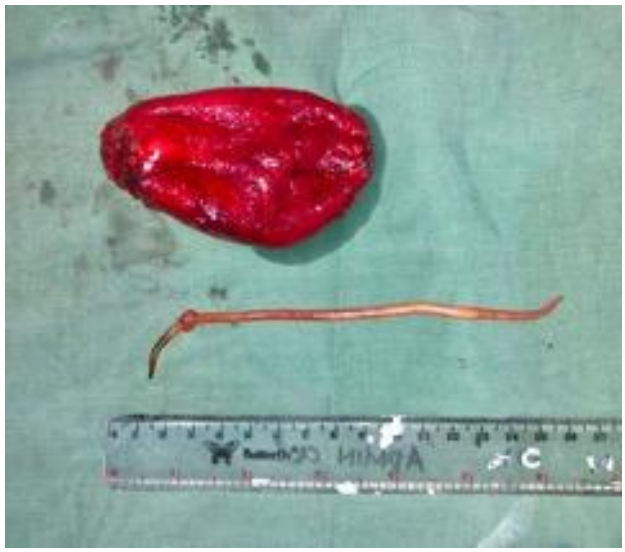

Figure 1. The gallbladder extraction and the evacuation of *Ascaris lumbricoides*.

The patient's recovery was rapid and uncomplicated, validating the decision for immediate surgical intervention. A critical component of the postoperative care was the timing of anthelmintic therapy. While the offending worm was removed, the patient undoubtedly harbored an intestinal reservoir of worms. Administering anthelmintics immediately before or during surgery is generally avoided, as dying worms in the intestine can release toxins or become agitated, potentially migrating into the biliary tree through the sphincter of Oddi during the stress of surgery. Therapy was delayed until the return of normal bowel function. On postoperative Day 1, following the passage of flatus, the patient received a single 400 mg dose of albendazole. This timing ensures the paralysis and expulsion of intestinal worms occur when peristalsis is active, preventing intestinal obstruction. The patient tolerated a solid diet on Postoperative Day 2 and was discharged on Postoperative Day 3. At the 6-month follow-up, the patient remained asymptomatic. A repeat ultrasonography was performed to ensure no late sequelae, such as stricture formation or recurrent stone disease (post-cholecystectomy syndrome). The scan showed a normal caliber CBD with no residual pathology. Liver enzymes had completely normalized, confirming the resolution of the cholestatic injury.

Table 2. Diagnosis, Therapeutic Intervention, and Clinical Outcome

Category	Details & Findings	Clinical Rationale / Remarks
I. DIAGNOSTIC ASSESSMENT		
Final Diagnosis	**Isolated Gallbladder Ascariasis** with secondary Acalculous Hydrops.	*Differentiated from calculous cholecystitis by lack of shadowing stones.*
Imaging (USG)	Inner Tube Sign Echogenic ring with central canal. Stripe Sign Linear tubular structure. **Cystic Duct:** Worm extension visualized.	*Real-time motility confirmed live parasite versus biliary sludge.*
II. THERAPEUTIC MANAGEMENT		
Preoperative	NPO status, IV Fluids. **Antibiotics:** Ceftriaxone 1g IV.	*Biliary prophylaxis against Enterobacteriaceae.*
Anesthesia Protocol	Rapid Sequence Induction **Contraindication:** Nitrous Oxide (N_2O) strictly avoided.	*Prevent erratic worm migration and bowel/worm distension due to gas diffusion.*
Surgical Approach	**Open Cholecystectomy** via Right Subcostal (Kocher's) incision.	*Open approach selected for superior tactile control of the impacted cystic duct.*
Critical Maneuver	Retrograde Milking Manual displacement of worm from cystic duct back into gallbladder *before* ligation.	***Prevents transection of the live worm and subsequent biliary leak or stump remnant.***
Intraoperative Findings	Gallbladder: Tense, edematous (Hydrops). CBD: Soft, non-dilated (<6mm). **Specimen:** 22 cm live *Ascaris lumbricoides*.	*Normal CBD palpation + normal bilirubin obviated need for CBD exploration.*
Pharmacotherapy	**Albendazole 400 mg** (Single dose). Administered on Post-operative Day 1.	*Delayed until return of bowel function to prevent obstruction by dying worms.*
III. OUTCOME AND FOLLOW-UP		
Hospital Course	Diet tolerated POD 2. Discharged on POD 3 .	*Uneventful postoperative recovery.*
Long-term (6 Months)	**Symptom Free:** No biliary colic. **USG:** Normal CBD, no residual pathology. **Labs:** Normal Liver Enzymes.	*No evidence of Post-Cholecystectomy Syndrome or recurrent infestation.*

3. Discussion

The pathogenesis of isolated gallbladder ascariasis represents a distinct and biologically complex deviation from the more common presentation of choledochal (Common Bile Duct) ascariasis.[11] To understand the rarity of this condition, one must first appreciate the anatomical gauntlet the parasite must navigate. In the physiological baseline, the Sphincter of Oddi functions as the primary gatekeeper, regulating the flow of bile into the duodenum and

preventing the reflux of duodenal contents. However, the integrity of this barrier is compromised by specific risk factors: a high parasitic load (which creates mechanical pressure at the orifice), previous endoscopic interventions such as sphincterotomy (which permanently disrupts the sphincter mechanism), or physiological relaxation induced by hormonal fluctuations or pregnancy. Once this barrier is breached, the adult *Ascaris* worm enters the biliary tree.[12]

Here, the worm faces the anatomical challenge of the cystic duct. Unlike the relatively wide and linear common bile duct, the cystic duct is guarded by the valves of Heister—a series of spiraling mucosal folds that create a tortuous, narrow channel. These valves function physiologically to prevent the collapse of the duct and regulate bile viscosity, but pathologically, they serve as a formidable deterrent to the entry of large nematodes. This anatomical resistance explains the statistical scarcity of the condition; extensive clinical series indicate that less than 2.1% of all biliary ascariasis cases involve the gallbladder itself. The worm essentially has to thread the needle against the flow of bile to enter the gallbladder.[13]

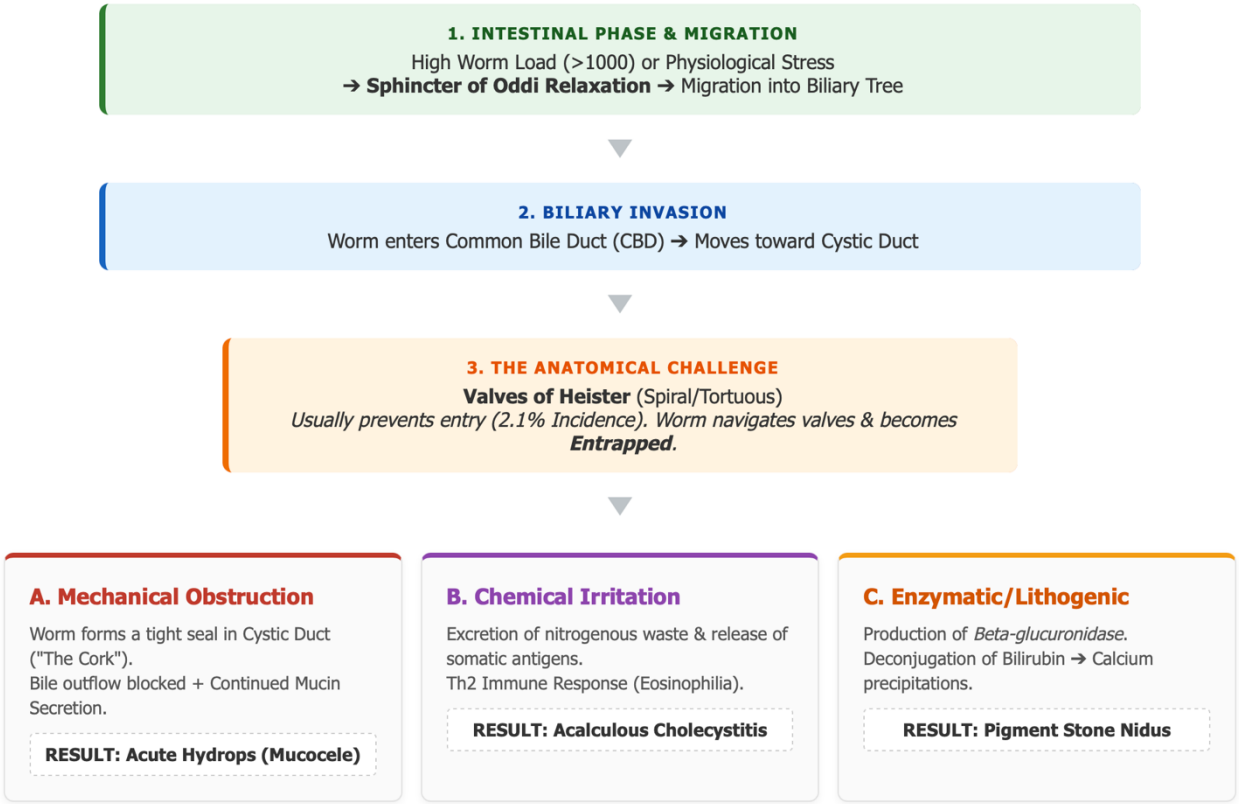

Figure 2. Pathophysiology of gallbladder ascariasis and hydrops.

In the case of this patient, the worm successfully navigated this tortuosity but subsequently became entrapped. This entrapment is not merely a positional finding; it precipitates a catastrophic cascade of pathology driven by three distinct mechanisms: mechanical obstruction, chemical irritation, enzymatic activity, and lithogenesis (Figure 2).[14] The primary driver of the acute presentation is mechanical. The worm, having entered the cystic duct, acts as a biological plug. Unlike a gallstone, which is rigid and

often irregular—allowing for intermittent ball-valve obstruction or partial flow—the nematode is soft, cylindrical, and capable of conforming to the ductal lumen to form a tight, complete seal. This obstruction creates a closed-loop obstruction. While bile outflow is halted, the gallbladder mucosa is not quiescent; it continues its physiological secretion of mucin and glycoproteins. In the absence of an outlet, this accumulation of fluid leads to a rapid rise in intraluminal pressure. The result is the distension of the gallbladder wall and the thinning of the mucosa, clinically manifesting as a mucocele or hydrops. The acute nature of this distension is what generates the intense visceral pain and the palpable mass observed on examination.[15]

The second mechanism is biochemical. The *Ascaris* worm is not an inert foreign body; it is a metabolically active organism.[16] The excretion of waste products (nitrogenous waste) and the shedding of somatic antigens from the worm's cuticle incite a profound inflammatory response from the gallbladder mucosa. This reaction is characterized as acalculous cholecystitis—inflammation in the absence of stones. This mechanism elucidates the diagnostic paradox observed in this patient: the presence of significantly elevated inflammatory markers (C-Reactive Protein of 45 mg/L) and Leukocytosis, despite the initial absence of bacterial superinfection. The eosinophilia further corroborates that this is a specific Th2-mediated immune response to parasitic antigens, rather than a standard pyogenic response to bacteria.

The third mechanism represents the long-term consequence of untreated infestation: the potential for stone formation. *Ascaris lumbricoides* produces significant quantities of the enzyme beta-glucuronidase. In the biliary tree, this enzyme catalyzes the deconjugation of bilirubin diglucuronide into free, unconjugated bilirubin. This insoluble free bilirubin precipitates with calcium ions to form calcium bilirubinate—the primary component of brown pigment stones. While our patient presented in the acute phase of hydrops, the chronic presence of the worm (suggested by the one-year history) placed her at high risk for this transformation. Early surgical intervention in this case not only resolved the hydrops but also preemptively arrested this lithogenic phase.[17]

The diagnostic journey in hepatobiliary ascariasis frequently highlights the limitations of cross-sectional imaging (CT/MRI) and the distinct superiority of transabdominal ultrasonography. While Computed Tomography is the modality of choice for staging malignancies or defining complex anatomy, its static nature is a liability when diagnosing parasitic infections. A worm on CT may appear merely as a non-specific filling defect, indistinguishable from mucus strands or sludge. In contrast, ultrasonography provides high-frequency, real-time imaging resolution. This case reinforces the status of ultrasound as the gold standard, capable of visualizing the definitive signs of infestation. In the longitudinal axis, the worm appears as a non-shadowing, linear, echogenic strip. This lack of acoustic shadowing is the key differentiator from gallstones, which almost always cast a posterior acoustic shadow. In the transverse axis, the worm presents as an echogenic ring (the cuticle) with a central anechoic dot (the fluid-filled digestive tract). This multilayered appearance is pathognomonic. The *sine qua non* of diagnosis is movement. Ultrasound allows the clinician to observe the erratic, non-directional writhing of the worm. This motility distinguishes the living pathology from static mimics like organized hematoma, arterial walls, or gangrenous mucosal flaps. In this case, the visualization of motility not only confirmed the diagnosis but also dictated the urgency of the intervention, as a mobile worm implies an active, rather than calcified, process.[18]

The management of biliary ascariasis is multimodal, often involving a combination of pharmacological, endoscopic, and surgical approaches.[19] However, the isolation of the parasite within the gallbladder fundamentally shifts the therapeutic paradigm toward surgery. Why is surgery necessary if anthelmintics (deworming drugs) are effective? The answer lies in the physics of clearance. Anthelmintics such as Albendazole function by

paralyzing the worm's neuromuscular system. In the intestinal tract, the paralyzed worm is expelled via peristalsis. In the common bile duct, a paralyzed worm *may* be flushed into the duodenum by bile flow. However, a worm paralyzed inside the gallbladder or cystic duct has no exit route. It becomes a trapped, dying foreign body. The decomposition of the worm releases highly toxic byproducts, leading to severe granulomatous inflammation, necrosis of the gallbladder wall, and eventually, perforation. Furthermore, the calcified remnants of a dead worm act as a permanent nidus for recurrent stone formation. Therefore, mechanical extraction is mandatory.

Endoscopic retrograde cholangiopancreatography (ERCP) is the gold standard for clearing the common bile duct.[20] However, its utility in *isolated gallbladder* ascariasis is severely limited. Standard endoscopic accessories (Dormia baskets or balloons) cannot be easily maneuvered through the tortuous valves of Heister to capture a worm inside the gallbladder. Attempts to do so carry a high risk of cystic duct perforation. Moreover, ERCP addresses only the parasite, not the underlying complication of hydrops or the ischemic threat to the gallbladder wall.

In an era dominated by minimally invasive surgery, the decision to perform an open cholecystectomy requires justification. While laparoscopic cholecystectomy is the undisputed standard for cholelithiasis, this case presented a specific trap for the laparoscopic surgeon: the impaction of the worm in the cystic duct. Laparoscopy relies on visual cues but lacks tactile feedback. A surgeon using laparoscopic graspers cannot feel the worm inside the duct. There is a substantial risk that the application of a surgical clip to the cystic duct could crush the worm or, worse, transect it. A transected worm leaves a viable segment in the common bile duct, guaranteeing postoperative complications (cholangitis or leak). The open approach (right subcostal incision) was chosen to prioritize tactile safety. It allowed for the manual palpation of the duct and the execution of the milking maneuver, ensuring the duct was empty before ligation. However, it is acknowledged that in centers with advanced laparoscopic expertise, this procedure can be performed safely via laparoscopy, provided the surgeon is vigilant and exercises the same principles of ductal clearance prior to clipping.

The surgical removal of the gallbladder resolves the acute crisis, but it does not cure the patient of ascariasis. The patient remains at risk for post-cholecystectomy syndrome driven by the intestinal reservoir. The millions of eggs and adult worms residing in the jejunum represent a persistent threat. Without systemic treatment, new worms can migrate through the Ampulla of Vater into the biliary stump (cystic duct remnant) or the hepatic ducts, causing recurrent biliary colic, cholangitis, or hepatic abscesses. This reality underscores the absolute necessity of the postoperative anthelmintic course (Albendazole). Surgery treats the *complication*, but medicine treats the *disease*. Long-term management must also extend beyond the hospital to the community level, focusing on sanitation improvements (WASH protocols) to break the fecal-oral transmission cycle and prevent reinfection.[19,20]

This report is limited by its design as a single case study, which prevents the generalization of findings to all cases of biliary ascariasis. Additionally, the assessment of common bile duct clearance relied on intraoperative palpation rather than cholangiography. While the clinical outcome was excellent, reliance on tactile sensitivity alone carries a theoretical risk of missing small worm fragments or debris. However, the normalization of the biochemical profile post-operatively validates the clinical decision-making in this specific context.

4. Conclusion

Isolated gallbladder ascariasis is a rare but clinically significant entity that challenges the diagnostic acumen of the surgeon and radiologist. It presents a masquerade, mimicking the clinical and laboratory features of acute acalculous cholecystitis, yet requiring a fundamentally different management strategy. This case serves as a critical reminder that in

endemic tropical regions, the differential diagnosis for biliary colic must extend beyond gallstones to include parasitic etiologies. Abdominal ultrasonography is the diagnostic modality of choice. The clinician should actively search for the inner tube sign and, crucially, verify worm viability by observing motility. This real-time assessment guides the urgency of intervention. When hydrops or cystic duct impaction is present, surgical intervention is mandatory to prevent gangrene and perforation. The critical technical step is the retrograde milking of the worm from the cystic duct prior to ligation. Neglecting this step risks transecting the parasite, leading to biliary leaks or retained foreign bodies. The scalpel alone is insufficient. Surgery must be paired with rigorous anthelmintic therapy to eradicate the intestinal reservoir. Long-term success depends on preventing the second wave of migration through systemic deworming and sanitation education. Ultimately, the successful management of this rare entity requires a synthesis of epidemiological awareness, precise sonographic interpretation, and detailed surgical technique tailored to the unique behavior of the parasite.

5. References

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