

## CASE REPORT

# Retrograde Cystic Duct Milking Before Ligation for Isolated Gallbladder Ascariasis with Acute Acalculous Hydrops: An Observational Analytic Case Study

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## ABSTRACT

**Background:** *Ascaris lumbricoides* infects an estimated 700 million to one billion people, but gallbladder involvement accounts for only about 2.1% of hepatobiliary ascariasis. A worm impacted in the cystic duct can cause acute acalculous hydrops and may be transected during cholecystectomy.

**Objective:** To describe the presentation, open surgical management with retrograde cystic duct milking, and six-month course of isolated gallbladder ascariasis with acute acalculous hydrops.

**Methods:** This study describes a 57-year-old woman with acute-on-chronic right upper quadrant pain in Indonesia, assessed clinically, biochemically, and sonographically from admission (T<sub>0</sub>) to month 6 (T<sub>6</sub>).

**Results:** At admission, pain was 8/10, leukocytes 15,460/mm<sup>3</sup>, eosinophils 8.0% (1,236/mm<sup>3</sup>), C-reactive protein (CRP) 45.0 mg/L, and alkaline phosphatase 133 U/L, with normal bilirubin (0.8 mg/dL). Ultrasonography showed a tense, stone-free gallbladder (10.5 × 4.5 cm; estimated volume 111.3 cm<sup>3</sup>; wall 4.5 mm) with the inner tube and stripe signs of a live worm impacted in the cystic duct. Under rapid sequence induction without nitrous oxide, open cholecystectomy with retrograde cystic duct milking displaced an intact 22.0-cm *A. lumbricoides* into the gallbladder before ligation. Albendazole 400 mg was given on postoperative day 1 after flatus; she was discharged on day 3. At month 6, she was asymptomatic, CRP (−96.9%) and eosinophils (−88.9%) had fallen, and liver enzymes were normal.

**Conclusion:** In this patient, retrograde cystic duct milking during open cholecystectomy, followed by albendazole after the return of bowel function, removed the impacted worm intact and was followed by full recovery.

## 1. Introduction

Soil-transmitted helminths remain one of the most persistent problems in global health and tropical medicine.<sup>1,2</sup> Among them, the giant intestinal roundworm *Ascaris lumbricoides* is the leading cause of parasitic morbidity worldwide, infecting between 700 million and one billion people in tropical and subtropical regions of Asia, sub-Saharan Africa, and Latin America.<sup>1,3</sup> This burden is sustained by the environmental resilience of fertilized *Ascaris* eggs, whose thick chitinous, ascaroside-rich shells withstand desiccation, chemical disinfectants, and temperature fluctuations for years in moist, shaded soil.<sup>4</sup> In rural agricultural districts where drinking water is drawn from untreated shallow wells and human fecal waste (night soil) is applied directly to food crops, fecal-oral transmission continues across generations.<sup>2,5</sup> Although ascariasis is classified as a neglected tropical disease, its capacity to cause life-threatening visceral, hepatobiliary, and pancreatic emergencies makes it important to digestive and hepatobiliary surgeons.<sup>3,6</sup>

In most infected hosts, adult *A. lumbricoides* live in the lumen of the jejunum and middle ileum, holding their position against peristalsis through continuous sinusoidal muscular contraction without causing peritoneal irritation.<sup>4,7</sup> However, when the worm burden rises above a critical density (classically, more than 1,000 adult worms in the intestine) or when the intestinal environment is disturbed by fever, prolonged fasting,

hyperacidity, subtherapeutic anthelmintic exposure, or volatile general anesthetic agents, the adult worms become agitated and migrate erratically.<sup>5-7</sup> Driven by overcrowding, hypoxia, and chemotactic stress, they migrate proximally against peristalsis from the jejunum into the duodenum, where their tendency to explore narrow openings directs them toward the ampulla of Vater.<sup>6,8</sup> Under physiological conditions, the sphincter of Oddi acts as a high-pressure muscular valve (basal resting pressure of 10–15 mmHg) that allows antegrade flow of bile and pancreatic juice while preventing duodenobiliary reflux.<sup>9</sup> Under the mechanical pressure of a high duodenal worm burden, during sphincter relaxation mediated by cholecystokinin or vasoactive intestinal peptide, or after previous endoscopic biliary instrumentation, this barrier can be breached, allowing the adult worm to ascend into the extrahepatic biliary tree and cause hepatobiliary and pancreatic ascariasis.<sup>8-10</sup>

Within the spectrum of hepatobiliary ascariasis, invasion of the gallbladder itself is rare, accounting for approximately 2.1% of hepatobiliary ascariasis cases in a systematic review.<sup>6</sup> This rarity is attributed to the anatomy of the extrahepatic biliary tract, in which the cystic duct is narrow and tortuous.<sup>6,11</sup> When an adult worm passes through the ampulla of Vater, the straight course and relatively wide lumen of the common bile duct and common hepatic duct offer the path of least resistance toward the hepatic confluence.<sup>4</sup> By contrast, to enter the gallbladder, an adult worm measuring 15–35 cm in length and 3–6 mm in diameter must make an acute turn at the cystic-choledochal

junction and ascend the narrow cystic duct, whose normal internal lumen measures only 2–3 mm.<sup>6,7</sup> The mucosa of the cystic duct forms a series of 2–14 crescentic spiral folds containing smooth muscle fibers, known anatomically as the valves of Heister.<sup>11,12</sup> Physiologically, the valves of Heister are thought to preserve the patency of the cystic duct, preventing its collapse or overdistension during sudden changes in biliary pressure, and may help regulate bile flow; pathologically, they form a tortuous, high-friction helical baffle that resists the entry of large parasites ascending against the outflow of concentrated gallbladder bile.<sup>11,12</sup> The gallbladder is therefore entered only when forceful cephalad movement of a viable worm coincides with transient relaxation of the cystic duct.<sup>13</sup>

Once an adult *A. lumbricoides* passes the valves of Heister and enters the gallbladder, its impaction in the cystic duct may initiate three linked processes: mechanical closed-loop obstruction, immunochemical mucosal inflammation, and enzymatic lithogenesis.<sup>6</sup> First, mechanically, the hydrated, cylindrical cuticle of the worm, supported by its hydrostatic skeleton, conforms closely to the helical contours of the cystic duct and acts as a compliant biological plug that stops bile outflow from the gallbladder while leaving the common bile duct patent.<sup>6</sup> Because the columnar epithelium of the gallbladder mucosa and the tubuloacinar glands of the infundibulum continue to secrete mucin glycoproteins, electrolytes, and water into the closed lumen, intraluminal pressure rises rapidly.<sup>14</sup> This closed-loop accumulation distends the gallbladder into a tense, cylindrical, stone-free mucocele, termed acute acalculous gallbladder hydrops, increasing wall tension in proportion to the radius according to Laplace's law and compressing intramural venous and lymphatic outflow.<sup>15,16</sup> Second, immunochemically, the live parasite is a metabolically active organism that releases nitrogenous waste, volatile organic acids, and highly immunogenic somatic cuticular antigens, including the ABA-1 nematode polypeptide allergen, directly onto the gallbladder mucosa.<sup>7</sup> These antigens activate mucosal mast cells, dendritic cells, and type 2 innate lymphoid cells to secrete interleukin-4 (IL-4), IL-5, and IL-13, producing a T-helper 2 (Th2) inflammatory response characterized by peripheral blood eosinophilia, mural eosinophilic infiltration, and raised C-reactive protein in the absence of gallstones.<sup>7,17</sup> Third, during prolonged residence in the lumen, live worms produce  $\beta$ -glucuronidase, which hydrolyzes water-soluble bilirubin diglucuronide into insoluble unconjugated bilirubin; this free bilirubin precipitates with luminal calcium ions ( $\text{Ca}^{2+}$ ) around dead cuticular fragments to form brown calcium bilirubinate pigment stones.<sup>6,18</sup>

Isolated gallbladder ascariasis complicated by acute acalculous hydrops is a diagnostic and therapeutic pitfall in emergency surgery.<sup>18,19</sup> Because the main bile duct remains unobstructed, patients typically present with severe right upper quadrant pain, localized hypochondrial fullness, and a normal serum bilirubin, a combination easily mistaken for a calculous gallbladder mucocele, Mirizzi syndrome, or idiopathic acalculous cholecystitis.<sup>19,20</sup> In addition, fecal microscopy for ova and parasites is often negative when biliary migration involves a single worm, an immature female, or a male worm.<sup>6,20</sup> Real-time B-mode transabdominal ultrasonography is the first-line diagnostic test and, unlike static computed tomography, can directly show the longitudinal echogenic stripe sign, the transverse concentric inner tube sign, and active non-directional writhing movement inside a distended gallbladder without acoustic shadowing.<sup>4,21,22</sup> Therapeutically, although uncomplicated intestinal ascariasis responds to oral benzimidazoles, giving anthelmintics while a live worm is

impacted in the cystic duct may be hazardous, because a worm killed inside a closed gallbladder becomes a decomposing foreign body that cannot pass the valves of Heister and may cause transmural gangrene, free gallbladder perforation, or choledochogastric fistula formation.<sup>6,13</sup> Moreover, standard endoscopic retrograde cholangiopancreatography (ERCP) extraction baskets cannot safely negotiate the tortuous valves of Heister,<sup>23–25</sup> and routine laparoscopic cholecystectomy carries a specific risk of crushing or transecting the impacted worm in the cystic duct because of reduced tactile feedback, leaving a devitalized parasitic remnant in the common bile duct.<sup>26–28</sup>

Although case reports of biliary ascariasis are available,<sup>7,8,13</sup> published reports are single cases that rarely document the longitudinal course of biomarkers, the volume of the hydropic gallbladder, the anesthetic precautions, or the intraoperative steps needed to clear an impacted worm from the cystic duct without transecting it.<sup>6</sup> This case report aims to describe the six-month clinicopathological course ( $T_0$  to  $T_6$ ) of acute acalculous gallbladder hydrops caused by isolated impaction of *A. lumbricoides* in the cystic duct, to describe the technique of retrograde cystic duct milking during open cholecystectomy, and to report the perioperative measures used, namely rapid sequence induction without nitrous oxide and alendazole given after the return of bowel function.

## 2. Methods

### Case design and reporting framework

We report a single patient. She was assessed clinically, in the laboratory, and by ultrasonography at seven predefined time points: on emergency admission ( $T_0$ ), during preoperative stabilization ( $T_1$ ), at open surgical decompression ( $T_2$ ), on the first three postoperative days ( $T_3$ – $T_5$ ), and at the 6-month outpatient visit ( $T_6$ ). The report follows the Surgical Case REport (SCARE) 2023 guideline.<sup>29</sup> Clinical observations, operative findings, and laboratory results were entered prospectively into the standard surgical records of the participating hospitals in South Sumatra, Indonesia.

### Case setting and patient information

The patient was managed by the Department of Surgery, Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital, Palembang, and the Department of Surgery, Dr. H. Mohamad Rabain Regional General Hospital, Muara Enim, Indonesia. The diagnosis of isolated gallbladder ascariasis complicated by acute acalculous gallbladder hydrops rested on four clinical, sonographic, and intraoperative criteria. First, the patient presented with acute-on-chronic right upper quadrant pain with localized right hypochondrial fullness and a tensely distended gallbladder exceeding 10.0 cm in length or 4.0 cm in transverse diameter on transabdominal ultrasonography. Second, ultrasonography showed a live, motile, non-shadowing tubular echogenic parasite with both the transverse inner tube sign and the longitudinal stripe sign, with its cephalad end impacted in the cystic duct. Third, gallstones, choledocholithiasis, and dilatation of the main bile duct were absent (common bile duct internal diameter less than 6.0 mm with a normal serum total bilirubin of  $\leq 1.2$  mg/dL), indicating isolated obstruction of the cystic duct. Fourth, an intact, viable adult *A. lumbricoides* was retrieved at operation from a stone-free gallbladder and identified parasitologically. Synchronous choledochal ascariasis with obstructive jaundice, gallstone disease, ascending pyogenic cholangitis, and acute biliary pancreatitis were excluded, and the patient agreed to 6-month follow-up.

### Data collection and diagnostic assessment

The baseline assessment at emergency admission ( $T_0$ , hour 0) recorded rural environmental sanitation and groundwater exposure; the chronology of chronic and acute pain, scored on the 10-point Visual Analog Scale (VAS; 0–10); vital signs, including mean arterial pressure ( $\text{MAP} = \text{DBP} + [\text{SBP} - \text{DBP}]/3$ ); complete blood count with differential leukocyte count and absolute eosinophil count; serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), and total bilirubin; high-sensitivity C-reactive protein (CRP); direct fecal microscopy for helminth ova; and high-frequency B-mode transabdominal ultrasonography. The clinical and laboratory measurements were repeated on postoperative day (POD) 1 ( $T_3$ , hour 24), POD 2 ( $T_4$ , hour 48), and POD 3 ( $T_5$ , hour 72, at discharge, with full hematological and biochemical reassessment), and at month 6 ( $T_6$ , day 180), when a comprehensive clinical, biochemical, fecal, and sonographic reassessment was performed.

Each laboratory and morphometric value was compared with standard adult reference intervals by calculating the ratio to the upper limit of normal ( $R_{ULN} = X_t/ULN$ ), which expresses the size of the deviation across different laboratory units. The relative change from baseline at each later time point  $T_n$  was calculated as  $\Delta\% = [(T_n - T_0)/T_0] \times 100\%$ . Gallbladder volume ( $V$ ) was estimated from the sonographic dimensions with the prolate ellipsoid formula  $V = (\pi/6) \times L \times W \times H \approx 0.5236 \times L \times W^2$ , where  $L$  is the longitudinal length (10.5 cm) and  $W$  is the transverse width (4.5 cm); only two dimensions were measured, and  $H$  was assumed to equal  $W$ . Inferential statistics do not apply to a single patient and were not used. The severity of acute cholecystitis was graded with the Tokyo Guidelines 2018 (TG18).<sup>30</sup> For comparison, published reports of gallbladder ascariasis were reviewed for patient age, worm length, gallbladder findings, eosinophilia, management (medical treatment, laparoscopic cholecystectomy, or open surgery), postoperative length of stay, and complications (Table 4).<sup>5–8,13–15,31–35</sup>

### Intervention and follow-up

Preoperative optimization ( $T_1$ , hours 2–6) consisted of nil per os fasting, intravenous isotonic Ringer's lactate, and prophylactic intravenous ceftriaxone (1 g). Intraoperative assessment ( $T_2$ , hours 6–8) documented rapid sequence endotracheal induction without nitrous oxide ( $\text{N}_2\text{O}$ ), the findings at right subcostal (Kocher) laparotomy, the external dimensions of the hydropic gallbladder and common bile duct, the retrograde cystic duct milking maneuver across the valves of Heister before cystic duct ligation, and back-table measurement of the retrieved viable worm. A single oral dose of albendazole (400 mg) was given on POD 1 ( $T_3$ ) after the passage of flatus; a solid diet was started on POD 2 ( $T_4$ ), and the patient was discharged on POD 3 ( $T_5$ ). Water, sanitation, and hygiene (WASH) counseling was given and continued through the 6-month follow-up ( $T_4$ – $T_6$ ).

### Ethical approval and consent

Ethical approval was granted by CMHC Research Center Ethics Committee, Indonesia (protocol No. 2026/0418), in coordination with the surgical governance of the Department of Surgery, Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General Hospital, Palembang, and the Department of Surgery, Dr. H. Mohamad Rabain Regional General Hospital, Muara Enim, Indonesia. All diagnostic, anesthetic, operative, and follow-up procedures followed the ethical standards of the institutional research committee, the 1964 Declaration of Helsinki and its later amendments, and the International Committee of Medical Journal Editors (ICMJE) recommendations for the protection of research participants.

Written informed consent was obtained from the patient and her legal family representative before surgery and before manuscript preparation, authorizing anonymous publication of her clinical records, laboratory results, sonographic measurements, and intraoperative specimen photographs. In line with ICMJE privacy recommendations, all clinical and specimen photographs were checked to ensure that they contained no biometric identifiers, hospital registration tags, or facial features, and a permanent black bar was to be applied to any clinical image showing the periorbital region.

## 3. Results

### Demographic profile, environmental exposure, and sonographic findings

A 57-year-old woman living in a rural agricultural community in South Sumatra, Indonesia, presented to the emergency department of our tertiary surgical referral center with an acute, severe exacerbation of right upper quadrant abdominal pain. The history revealed environmental exposure to geohelminth transmission: she lived in a farming district with incomplete sewage containment, relied exclusively on an untreated shallow well for drinking water, and lived next to cultivated fields where night soil (human fecal fertilizer) was periodically applied. She denied recent travel outside South Sumatra and diarrheal illness in her household; her daily use of untreated groundwater was a likely route of fecal-oral ingestion of embryonated *A. lumbricoides* eggs.

Her symptoms followed two phases, from an indolent phase to an acute obstructive crisis. During the preceding 12 months (prodromal phase), she had intermittent, dull, gnawing epigastric and right upper quadrant discomfort rated 2–3/10 on the VAS, typically 30–60 minutes after fatty meals, which she treated herself with over-the-counter antacids for presumed functional dyspepsia. Forty-eight hours before admission ( $T_0$ ), the pain became continuous, unremitting, and severe, rated 8/10 on the VAS, an increase of +220.0% over her mean chronic baseline score of 2.5/10. The pain radiated to the inferior angle of the right scapula (Boas' sign), consistent with referral along the right phrenic and lower intercostal nerves from tense capsular distension. She denied fever, rigors, hematemesis, melena, scleral icterus, dark tea-colored urine (choloria), and pale acholic stools, suggesting that the obstruction was confined to the cystic duct and gallbladder and that the common bile duct remained patent.

On examination at admission ( $T_0$ ), the patient was alert and oriented, in acute pain, and hemodynamically compensated without signs of sepsis. Blood pressure was 120/80 mmHg (MAP 93.3 mmHg), heart rate 83 beats/min, respiratory rate 21 breaths/min, and axillary temperature 36.1°C. The sclerae were anicteric, and cardiopulmonary auscultation was unremarkable. Superficial and deep palpation of the right hypochondrium elicited marked localized tenderness and revealed a smooth, tense, pear-shaped to cylindrical mass projecting below the right costal margin in the gallbladder fossa, consistent with acute gallbladder hydrops. Murphy's sign was equivocal: deep inspiration increased right upper quadrant tenderness, but the abrupt inspiratory arrest typical of transmural bacterial pericholecystitis was absent, consistent with sterile, high-pressure mucin distension.

Real-time B-mode transabdominal ultrasonography at  $T_0$  showed a tensely distended, cylindrical gallbladder measuring 10.5 × 4.5 cm, corresponding to an estimated ellipsoid volume of 111.3 cm<sup>3</sup> (+147.3% above the upper physiological fasting limit of 45.0 cm<sup>3</sup>;  $R_{ULN} = 2.47$ ), with concentric submucosal wall edema of 4.5 mm (reference < 3.0 mm;  $R_{ULN} = 1.50$ ) and no

gallstones or acoustic shadowing. Dynamic scanning showed the transverse concentric inner tube sign, the longitudinal parallel stripe sign, and active non-directional writhing movement of a

live worm whose cephalad end was tightly wedged in the cystic duct, whereas the common bile duct was of normal caliber at 4.8 mm (< 6.0 mm) (Table 1).

**Table 1.** Baseline demographic, environmental, clinical, and sonographic profile at emergency admission (T<sub>0</sub>).

| Parameter                                                          | Observed value (T <sub>0</sub> )                                                       | Reference interval                                       | Ratio and interpretation                                                    |
|--------------------------------------------------------------------|----------------------------------------------------------------------------------------|----------------------------------------------------------|-----------------------------------------------------------------------------|
| Age, sex, and setting                                              | 57 years, female; rural farming district                                               | Adult demographic baseline                               | Endemic area for geohelminth transmission                                   |
| Drinking water and sanitation                                      | Untreated shallow well; agricultural night soil                                        | Treated potable water supply                             | Likely fecal-oral route for embryonated <i>A. lumbricoides</i> eggs         |
| Prodromal phase (12 months to 48 h before admission)               | Intermittent dull RUQ ache 30–60 min after meals (VAS 2–3/10)                          | 0/10 (asymptomatic)                                      | Free worm in the gallbladder postulated; CCK-induced colic                  |
| Acute obstructive phase (48 h before admission to T <sub>0</sub> ) | Continuous severe RUQ pain with radiation to the right scapula (Boas' sign) (VAS 8/10) | 0/10 (asymptomatic)                                      | +220.0% increase in pain; cephalad impaction of the worm in the cystic duct |
| Hemodynamics and temperature                                       | BP 120/80 mmHg (MAP 93.3 mmHg); HR 83 bpm; RR 21/min; temperature 36.1°C               | BP < 120/80 mmHg; HR 60–100 bpm; temperature 36.5–37.5°C | Hemodynamically compensated; afebrile                                       |
| Right hypochondrial findings                                       | Tense, palpable hydroptic gallbladder; equivocal Murphy's sign                         | Non-palpable gallbladder; negative Murphy's sign         | Capsular stretch from a closed-loop mucocoele; anicteric sclerae            |
| Gallbladder dimensions (ultrasonography)                           | 10.5 × 4.5 cm (tense, cylindrical)                                                     | < 10.0 × 4.0 cm (pear-shaped)                            | +5.0% length and +12.5% width above the upper normal size                   |
| Estimated ellipsoid volume                                         | 111.3 cm <sup>3</sup> ( $V = [\pi/6] \times 10.5 \times 4.5 \times 4.5$ cm)            | 30.0–45.0 cm <sup>3</sup> (fasting volume)               | $R_{ULN} = 2.47$ (+147.3% volume expansion)                                 |
| Gallbladder wall thickness                                         | 4.5 mm (concentric submucosal edema, no gas)                                           | < 3.0 mm                                                 | $R_{ULN} = 1.50$ (+50.0% wall thickening; TG18 imaging finding)             |
| Intraluminal sonographic signs                                     | Inner tube sign and stripe sign; writhing movement                                     | Anechoic bile without internal echoes                    | Live worm confirmed; no calculi or sludge                                   |
| Cystic duct and common bile duct (CBD)                             | Cephalad end of the worm impacted in the cystic duct; CBD 4.8 mm, patent               | Cystic duct patent; CBD < 6.0 mm                         | Isolated closed-loop cystic duct plug with a patent CBD                     |

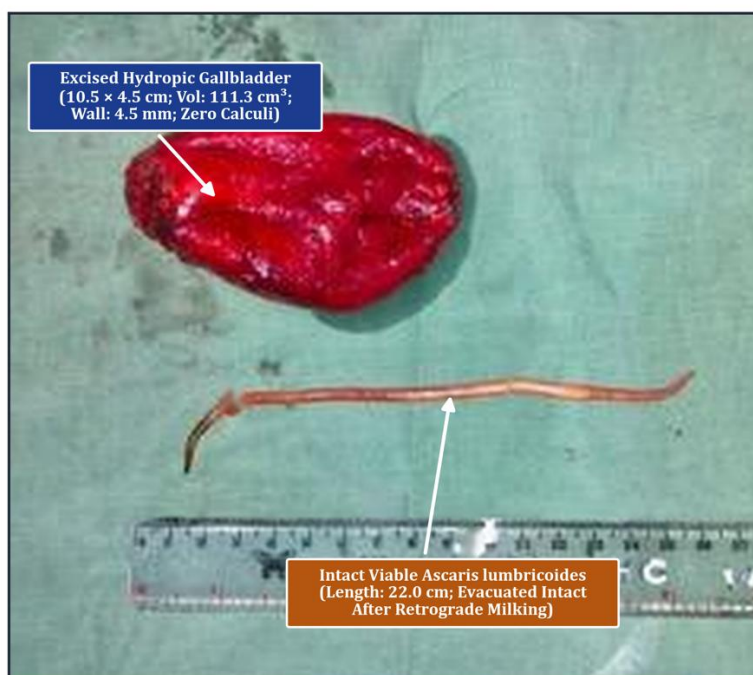
Abbreviations: BP, blood pressure; CBD, common bile duct; CCK, cholecystokinin; HR, heart rate; MAP, mean arterial pressure; RR, respiratory rate; RUQ, right upper quadrant;  $R_{ULN}$ , ratio to the upper limit of normal; TG18, Tokyo Guidelines 2018; ULN, upper limit of normal; VAS, Visual Analog Scale.

### Intraoperative retrograde cystic duct milking and macroscopic findings

After the diagnosis of acute acalculous gallbladder hydrops caused by a live worm in the cystic duct, the patient underwent preoperative optimization (T<sub>1</sub>, hours 2–6) with nil per os fasting, intravenous isotonic Ringer's lactate, and intravenous ceftriaxone (1 g) prophylaxis. At induction (T<sub>2</sub>, hour 6), general endotracheal anesthesia was secured by rapid sequence induction with intravenous propofol (2.0 mg/kg), fentanyl (2.0 µg/kg), and rocuronium (1.0 mg/kg) to avoid excitatory migration of the worm from the duodenum to the pharynx,<sup>36</sup> and nitrous oxide (N<sub>2</sub>O) was excluded to avoid diffusion of this highly soluble gas into the worm's pseudocoelomic cavity. Open cholecystectomy through a right subcostal (Kocher) incision exposed a tensely distended, edematous, hyperemic gallbladder (10.5 × 4.5 cm) without dense omental adhesions. Bidigital

palpation of Calot's triangle identified a firm, cylindrical, rubbery, mobile structure in the dilated cystic duct.

Before any clamp or ligature was applied, the lead surgeon performed a retrograde cystic duct milking maneuver: while Hartmann's pouch was stabilized with an atraumatic bowel grasper, sequential retrograde digital pressure was applied from the juxta-choledochal segment of the cystic duct toward the gallbladder body, dislodging the impacted cephalad segment of the worm from the valves of Heister back into the gallbladder lumen before double suture ligation. Supraduodenal palpation confirmed a soft, unobstructed 4.8-mm common bile duct. Longitudinal back-table incision of the excised gallbladder released clear mucinous fluid and a single intact, actively motile adult *A. lumbricoides* measuring 22.0 cm in length, with no gallstones or biliary sludge (Figure 1).

(a) Ex Vivo Macroscopic Specimen: Hydropic Gallbladder & 22.0-cm Viable *A. lumbricoides*

(b) Biomechanical Schematic of Intraoperative Retrograde Cystic Duct Milking Maneuver

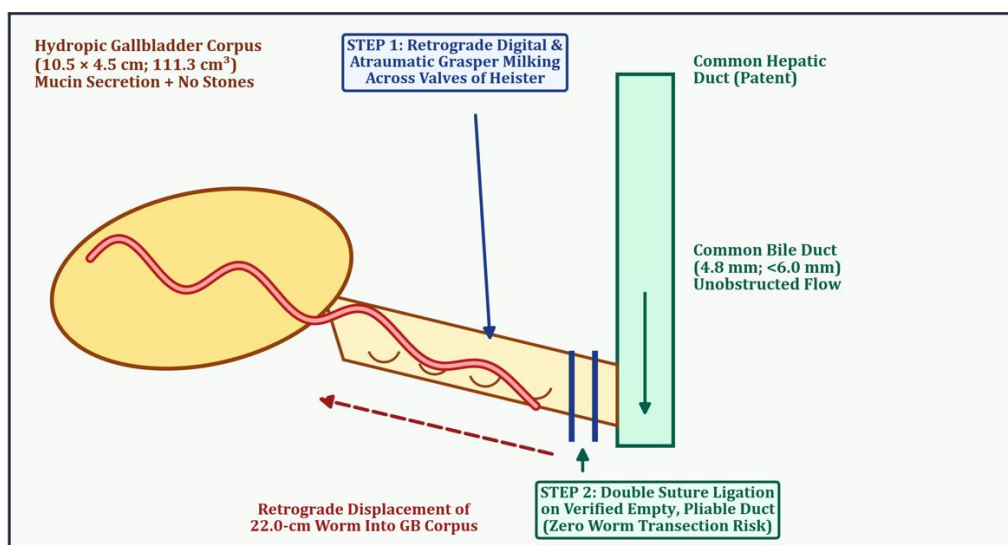


**Figure 1.** Macroscopic specimen and intraoperative retrograde cystic duct milking maneuver. (a) Excised, tensely distended, hyperemic gallbladder (10.5 × 4.5 cm; estimated ellipsoid volume 111.3 cm<sup>3</sup>; wall thickness 4.5 mm) with the intact, viable 22.0-cm adult *A. lumbricoides* and a metric ruler after longitudinal back-table incision, which released clear mucinous fluid and showed no gallstones. (b) Schematic (not to scale) of the intraoperative retrograde digital and atraumatic bowel-grasper milking maneuver, which displaced the impacted cephalad segment of the worm from the valves of Heister back into the gallbladder body before double suture ligation of the empty cystic duct, preserving the patency of the common bile duct (4.8 mm).

#### Longitudinal hematological, eosinophilic, and hepatobiliary course (T<sub>0</sub>–T<sub>6</sub>)

Laboratory tests at admission (T<sub>0</sub>) supported a helminthic cause with localized cystic duct obstruction, and serial monitoring through month 6 (T<sub>6</sub>) showed a steady decline in all inflammatory and hepatobiliary markers. At T<sub>0</sub>, hemoglobin was normal (12.5 g/dL); the total leukocyte count was raised at 15,460/mm<sup>3</sup> (R<sub>ULN</sub> = 1.41; +40.5% above ULN); the eosinophil fraction was 8.0% (R<sub>ULN</sub> = 2.67) and the absolute eosinophil count (AEC) 1,236/mm<sup>3</sup> (reference < 500/mm<sup>3</sup>; R<sub>ULN</sub> = 2.47; +147.2% above ULN); and CRP was raised ninefold to 45.0 mg/L (reference < 5.0 mg/L; R<sub>ULN</sub> = 9.00). Liver biochemistry showed

dissociated anicteric cholestasis: ALP was raised at 133 U/L (reference 35–105 U/L; R<sub>ULN</sub> = 1.27; +26.7% above ULN), with mild transaminase elevation (AST 59 U/L, R<sub>ULN</sub> = 1.69; ALT 73 U/L, R<sub>ULN</sub> = 1.62), whereas total bilirubin was normal at 0.8 mg/dL (R<sub>ULN</sub> = 0.67), consistent with a patent common bile duct.

Albendazole (a single oral dose of 400 mg) was given on POD 1 (T<sub>3</sub>, hour 24), once flatus had passed. Pain had eased to 3/10 on the VAS by then (Δ% = –62.5%). CRP had almost halved, to 24.0 mg/L (Δ% = –46.7%), whereas leukocytes (11,200/mm<sup>3</sup>; Δ% = –27.6%) and the AEC (890/mm<sup>3</sup>; Δ% = –28.0%) had fallen less. When she went home on POD 3 (T<sub>5</sub>, hour 72), she rated her pain 1/10 (Δ% = –87.5%) and CRP was 6.2 mg/L (Δ% = –86.2%). Her leukocyte count of 8,420/mm<sup>3</sup> (Δ% = –45.5%) and

AEC of  $354/\text{mm}^3$  ( $\Delta\% = -71.4\%$ ; eosinophils  $4.2\%$ ) were back within the reference range. So were AST, ALT, and ALP, at  $34 \text{ U/L}$  ( $\Delta\% = -42.4\%$ ),  $41 \text{ U/L}$  ( $\Delta\% = -43.8\%$ ), and  $96 \text{ U/L}$  ( $\Delta\% = -27.8\%$ ); total bilirubin was  $0.6 \text{ mg/dL}$  ( $\Delta\% = -25.0\%$ ).

Six months later ( $T_6$ , day 180), the patient had no symptoms (VAS  $0/10$ ;  $\Delta\% = -100.0\%$ ), and every value was normal:

leukocytes  $6,850/\text{mm}^3$  ( $\Delta\% = -55.7\%$ ), eosinophils  $2.0\%$  with an AEC of  $137/\text{mm}^3$  ( $\Delta\% = -88.9\%$ ), AST  $22 \text{ U/L}$  ( $\Delta\% = -62.7\%$ ), ALT  $24 \text{ U/L}$  ( $\Delta\% = -67.1\%$ ), ALP  $68 \text{ U/L}$  ( $\Delta\% = -48.9\%$ ), total bilirubin  $0.5 \text{ mg/dL}$  ( $\Delta\% = -37.5\%$ ), and CRP  $1.4 \text{ mg/L}$  ( $\Delta\% = -96.9\%$ ) (Table 2; Figure 2).

**Table 2.** Clinical, hematological, and hepatobiliary values from  $T_0$  to  $T_6$  with relative change from baseline ( $\Delta\%$ ).

| Parameter (unit; reference interval)                  | $T_0$ admission (hour 0) | $T_3$ POD 1 (hour 24; $\Delta\%$ ) | $T_5$ POD 3, discharge (hour 72; $\Delta\%$ ) | $T_6$ month 6 (day 180; $\Delta\%$ ) | Comment                           |
|-------------------------------------------------------|--------------------------|------------------------------------|-----------------------------------------------|--------------------------------------|-----------------------------------|
| VAS pain score (0–10; ref 0)                          | 8                        | 3 (–62.5%)                         | 1 (–87.5%)                                    | 0 (–100.0%)                          | —                                 |
| Temperature ( $^{\circ}\text{C}$ ; ref 36.5–37.5)     | 36.1                     | 36.7                               | 36.6                                          | 36.6                                 | Normothermic throughout           |
| Mean arterial pressure (mmHg; ref 70–100)             | 93.3 (120/80)            | 90.0 (118/76; –3.5%)               | 90.0 (118/76; –3.5%)                          | 88.0 (116/74; –5.7%)                 | Hemodynamically stable throughout |
| Hemoglobin (g/dL; ref 12.0–16.0)                      | 12.5                     | 12.3 (–1.6%)                       | 12.4 (–0.8%)                                  | 13.1 (+4.8%)                         | Intraoperative blood loss < 50 mL |
| Total leukocytes ( $/\text{mm}^3$ ; ref 4,500–11,000) | 15,460 (1.41× ULN)       | 11,200 (–27.6%)                    | 8,420 (–45.5%)                                | 6,850 (–55.7%)                       | —                                 |
| Eosinophil fraction (%; ref 1.0–3.0)                  | 8.0 (2.67× ULN)          | 7.9 (–1.3%)                        | 4.2 (–47.5%)                                  | 2.0 (–75.0%)                         | —                                 |
| Absolute eosinophils ( $/\text{mm}^3$ ; ref < 500)    | 1,236 (2.47× ULN)        | 890 (–28.0%)                       | 354 (–71.4%)                                  | 137 (–88.9%)                         | —                                 |
| AST (U/L; ref < 35)                                   | 59 (1.69× ULN)           | 46 (–22.0%)                        | 34 (–42.4%)                                   | 22 (–62.7%)                          | —                                 |
| ALT (U/L; ref < 45)                                   | 73 (1.62× ULN)           | 58 (–20.5%)                        | 41 (–43.8%)                                   | 24 (–67.1%)                          | —                                 |
| Alkaline phosphatase (U/L; ref 35–105)                | 133 (1.27× ULN)          | 118 (–11.3%)                       | 96 (–27.8%)                                   | 68 (–48.9%)                          | —                                 |
| Total bilirubin (mg/dL; ref 0.1–1.2)                  | 0.8 (0.67× ULN)          | 0.7 (–12.5%)                       | 0.6 (–25.0%)                                  | 0.5 (–37.5%)                         | CBD unobstructed                  |
| C-reactive protein (mg/L; ref < 5.0)                  | 45.0 (9.00× ULN)         | 24.0 (–46.7%)                      | 6.2 (–86.2%)                                  | 1.4 (–96.9%)                         | —                                 |

$\Delta\% = [(T_n - T_0)/T_0] \times 100\%$ ;  $\Delta\%$  is not given for temperature in  $^{\circ}\text{C}$ . At  $T_4$  (POD 2, hour 48), a solid diet was tolerated, and the VAS pain score was  $2/10$  ( $\Delta\% = -75.0\%$ ).

Abbreviations: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CBD, common bile duct; POD, postoperative day; ULN, upper limit of normal; VAS, Visual Analog Scale.

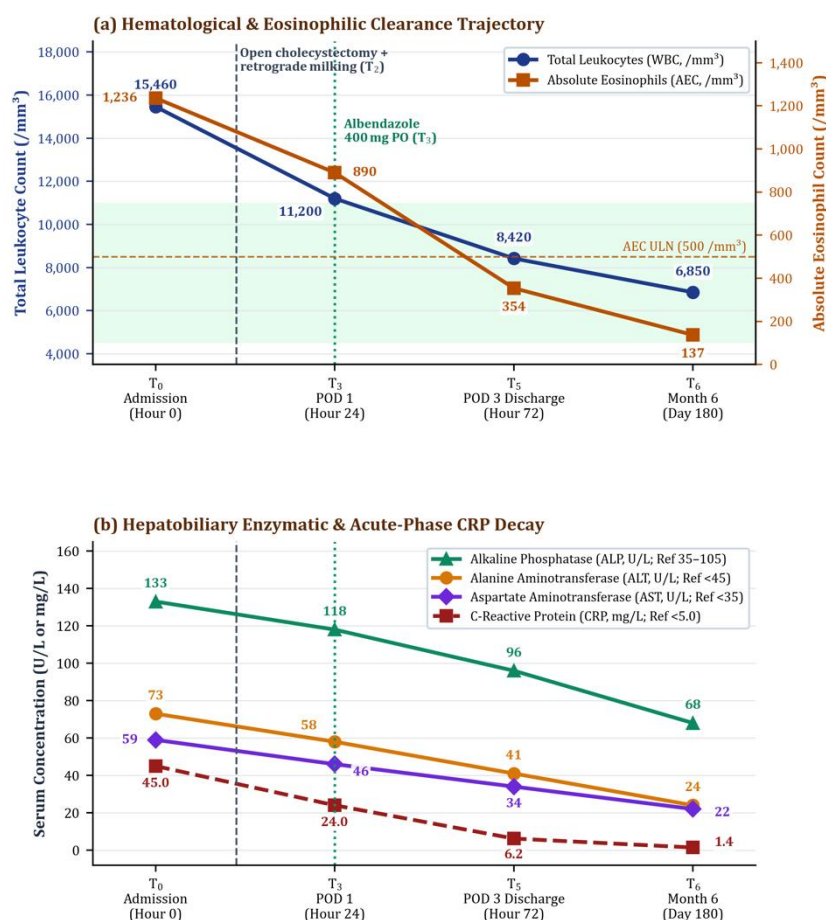


**Figure 2.** Hematological, eosinophilic, and hepatobiliary values from  $T_0$  to  $T_6$ . (a) Total leukocyte count (WBC:  $15,460 \rightarrow 11,200 \rightarrow 8,420 \rightarrow 6,850/\text{mm}^3$ ) and absolute eosinophil count (AEC:  $1,236 \rightarrow 890 \rightarrow 354 \rightarrow 137/\text{mm}^3$ ) after open cholecystectomy with retrograde cystic duct milking ( $T_2$ ) and oral albendazole 400 mg after the return of flatus ( $T_3$ ); the shaded band is the leukocyte reference range, and the dashed line is the AEC upper limit of normal. (b) Alkaline phosphatase (ALP:  $133 \rightarrow 118 \rightarrow 96 \rightarrow 68 \text{ U/L}$ ), alanine aminotransferase (ALT:  $73 \rightarrow 58 \rightarrow 41 \rightarrow 24 \text{ U/L}$ ), aspartate aminotransferase (AST:  $59 \rightarrow 46 \rightarrow 34 \rightarrow 22 \text{ U/L}$ ), and C-reactive protein (CRP:  $45.0 \rightarrow 24.0 \rightarrow 6.2 \rightarrow 1.4 \text{ mg/L}$ ).

The cumulative percentage reductions at discharge ( $T_5$ ) and at 6 months ( $T_6$ ) showed two patterns of resolution (Figure 3). Variables driven directly by acute capsular stretch and by antigen stimulation from the live parasite — VAS pain ( $\Delta\%$  = -87.5% at  $T_5$  and -100.0% at  $T_6$ ), CRP ( $\Delta\%$  = -86.2% at  $T_5$  and -96.9% at  $T_6$ ), and the AEC ( $\Delta\%$  = -71.4% at  $T_5$  and -88.9% at

$T_6$ ) — fell rapidly within 72 hours of open surgical removal of the 22.0-cm worm. The liver enzymes declined more gradually, with ALT ( $\Delta\%$  = -67.1% at  $T_6$ ), AST ( $\Delta\%$  = -62.7% at  $T_6$ ), and ALP ( $\Delta\%$  = -48.9% at  $T_6$ ) returning to the mid-normal range without secondary postoperative rises.

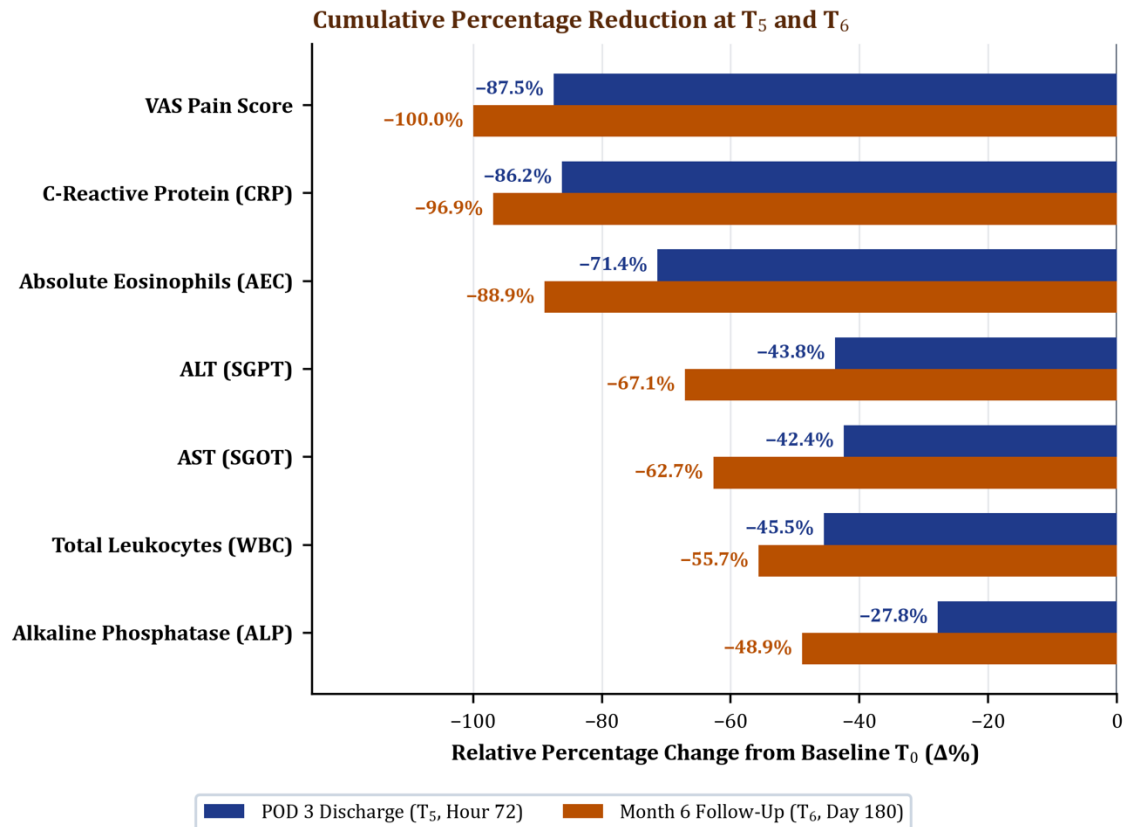


**Figure 3.** Relative percentage change ( $\Delta\%$ ) from baseline ( $T_0$ ) at POD 3 discharge ( $T_5$ ) and month 6 ( $T_6$ ): VAS pain (-87.5% and -100.0%), CRP (-86.2% and -96.9%), AEC (-71.4% and -88.9%), ALT (-43.8% and -67.1%), AST (-42.4% and -62.7%), WBC (-45.5% and -55.7%), and ALP (-27.8% and -48.9%).

### Perioperative management and severity grading

The perioperative management from  $T_1$  to  $T_6$  — fasting to reduce cholecystokinin release and intravenous ceftriaxone prophylaxis ( $T_1$ ); rapid sequence endotracheal induction without nitrous oxide ( $T_2$ ); open right subcostal

cholecystectomy with bidigital palpation and the retrograde cystic duct milking maneuver ( $T_2$ ); albendazole 400 mg on POD 1 after the return of flatus ( $T_3$ ); and water, sanitation, and hygiene (WASH) counseling through month 6 ( $T_4$ - $T_6$ ) — is summarized in Table 3.

**Table 3.** Perioperative anesthetic, surgical, and postoperative management from  $T_1$  to  $T_6$ .

| Perioperative phase                                     | Intervention and technique                                                                                | Rationale                                                                                                               | Observed outcome                                                                      |
|---------------------------------------------------------|-----------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------|
| $T_1$ preoperative stabilization (hours 2–6)            | Nil per os; IV isotonic Ringer's lactate; IV ceftriaxone 1 g                                              | Reduce endogenous CCK release; achieve bactericidal biliary antibiotic concentration                                    | No gallbladder perforation or ascending infection                                     |
| $T_2$ anesthetic induction (hour 6)                     | Rapid sequence induction (propofol 2.0 mg/kg, fentanyl 2.0 $\mu$ g/kg, rocuronium 1.0 mg/kg)              | Immediate airway control, bypassing the excitatory induction stage                                                      | No pharyngeal worm migration or laryngospasm                                          |
| $T_2$ anesthetic maintenance (hours 6–8)                | Sevoflurane in $O_2$ /medical air ( $FiO_2$ 0.40); nitrous oxide ( $N_2O$ ) excluded                      | Avoid diffusion of $N_2O$ (blood:gas partition coefficient 0.47) into gas-containing spaces and the worm's pseudocoelom | Worm retrieved intact; no gallbladder rupture                                         |
| $T_2$ surgical access and exploration                   | Open laparotomy through a right subcostal (Kocher) incision; bidigital palpation of Calot's triangle      | Preserve three-dimensional tactile feedback across the dilated cystic duct and supraduodenal CBD (4.8 mm)               | Impacted worm confirmed in the cystic duct; clear CBD verified without choledochotomy |
| $T_2$ critical intraoperative maneuver                  | Retrograde cystic duct milking by digital pressure and an atraumatic bowel grasper                        | Displace the 22.0-cm viable <i>A. lumbricoides</i> from the valves of Heister into the gallbladder body                 | Worm removed intact without transection; no bile leak                                 |
| $T_3$ postoperative eradication (POD 1, hour 24)        | Single oral dose of albendazole 400 mg after the passage of flatus                                        | Inhibit parasitic $\beta$ -tubulin polymerization once aboral bowel propulsion has returned                             | No postoperative bowel obstruction; no ova in the stool at month 6                    |
| $T_4$ - $T_6$ recovery and follow-up (POD 2 to month 6) | Solid diet on POD 2; discharge on POD 3; WASH counseling; ultrasonography and laboratory tests at month 6 | Restore hepatobiliary function and interrupt fecal-oral reinfection                                                     | 3-day hospital stay; no complications; CBD 4.2 mm and no ova in the stool at month 6  |

Abbreviations: CBD, common bile duct; CCK, cholecystokinin;  $FiO_2$ , fraction of inspired oxygen; IV, intravenous;  $N_2O$ , nitrous oxide; POD, postoperative day; WASH, water, sanitation, and hygiene.

According to TG18, the patient fulfilled all three diagnostic domains (local sign A: palpable, tender right upper quadrant gallbladder mass; systemic sign B: CRP 45.0 mg/L and leukocytes 15,460/mm<sup>3</sup>; imaging sign C: distended 10.5 × 4.5-cm gallbladder with 4.5-mm wall edema) and met the criteria for grade II (moderate) severity, which supported urgent surgical decompression of the gallbladder rather than observation.<sup>30</sup>

#### Standardized ratios and comparison with published cases

Standardized ratios to the upper limit of normal are shown in Figure 4, and published comparator cases are summarized in

Table 4. At T<sub>0</sub>, CRP was 9.00× ULN, the AEC 2.47× ULN, and the estimated gallbladder volume 2.47× ULN (111.3 cm<sup>3</sup>), with preserved patency of the common bile duct (total bilirubin 0.67× ULN; common bile duct 4.8 mm at T<sub>0</sub> and 4.2 mm at T<sub>6</sub>). The hospital stay was 3 days, there were no surgical complications, and no ova were found in the stool at 6 months. The published cases differ in presentation and management (medical treatment, laparoscopic cholecystectomy, or laparotomy with bile duct exploration), and most do not report the length of stay.<sup>5-8,13,35</sup>

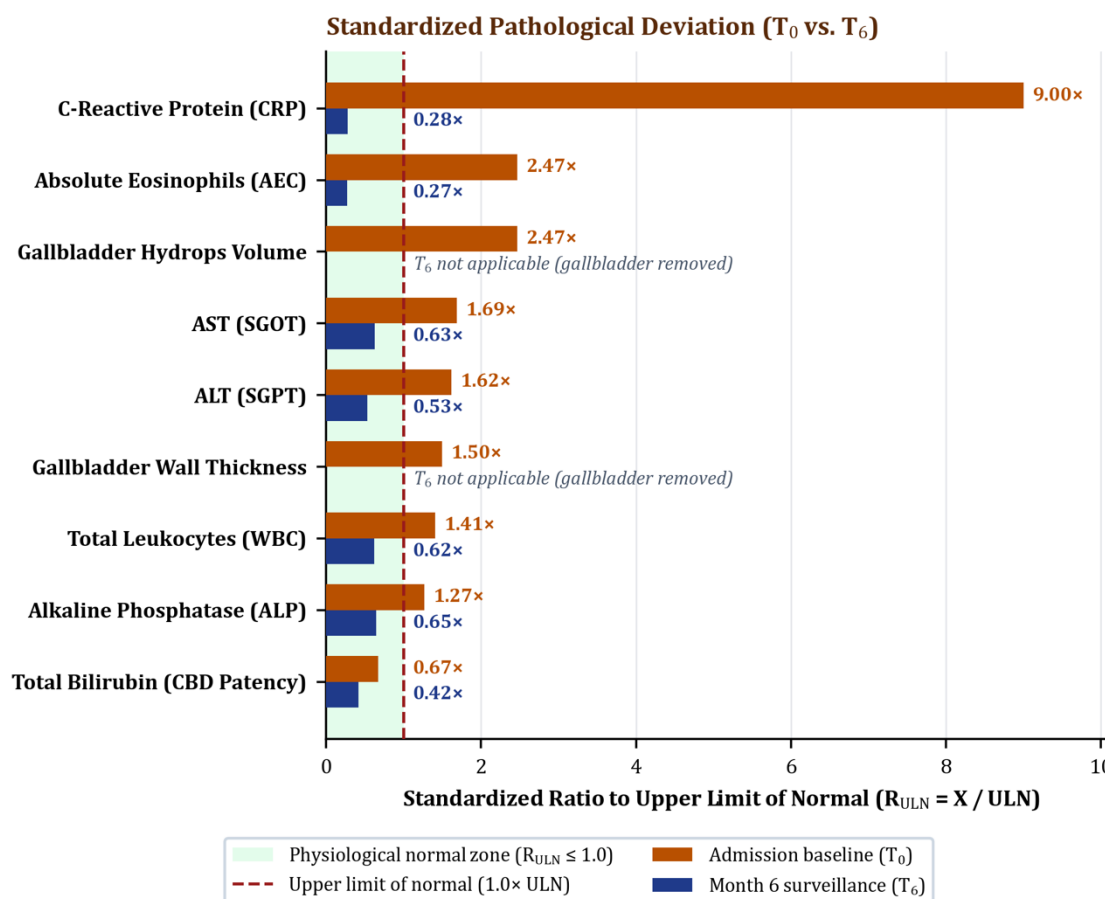


**Figure 4.** Ratio to the upper limit of normal (R<sub>ULN</sub> = X/ULN) at admission (T<sub>0</sub>) and month 6 (T<sub>6</sub>). Baseline peaks were seen for CRP (9.00× ULN), AEC (2.47× ULN), and estimated gallbladder volume (2.47× ULN; 111.3 cm<sup>3</sup>), with a normal total bilirubin (0.67× ULN); all laboratory values were below the ULN at T<sub>6</sub> (gallbladder volume and wall thickness are not applicable after cholecystectomy). Published comparator cases are summarized in Table 4.

**Table 4.** Published cases and series of gallbladder ascariasis used for comparison (2022–2025).

| Study, year (country)                          | Age and sex      | Gallbladder findings and worm                                                                                                                           | Management                                                                                                       | Length of stay and outcome                                                             |
|------------------------------------------------|------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------------|
| Present case, 2026 (Indonesia)                 | 57 years, female | Acute acalculous hydrops (10.5 × 4.5 cm; 111.3 cm <sup>3</sup> ; wall 4.5 mm); live 22.0-cm worm impacted in the cystic duct; AEC 1,236/mm <sup>3</sup> | N <sub>2</sub> O-free RSI; open cholecystectomy with retrograde cystic duct milking; albendazole 400 mg on POD 1 | 3 days; no complications; clinical, biochemical, and sonographic resolution at month 6 |
| Soh et al., 2025 (Senegal) <sup>13</sup>       | 41 years, female | Live worm moving freely in the gallbladder; eosinophils 1.3%; CRP 7.23 mg/L; worm length not reported                                                   | Albendazole 400 mg and amoxicillin-clavulanate; no surgery                                                       | Monitored for 3 days in hospital; no recurrence at 1 month                             |
| Saad et al., 2025 (Egypt) <sup>7</sup>         | 1 year, male     | Chronic eosinophilic cholecystitis; degenerated worm ≈ 15 mm                                                                                            | Oral albendazole, then laparoscopic cholecystectomy                                                              | Discharged on POD 3; no recurrence on ultrasonography at 1, 3, and 6 months            |
| Mohamud et al., 2025 (Somalia) <sup>8</sup>    | 38 years, female | Acalculous cholecystitis with a mobile intraluminal worm; dead worm 13.5 cm; chronic cholecystitis on histology                                         | Intravenous antibiotics for 3 days, laparoscopic cholecystectomy, albendazole 400 mg daily for 3 days            | Length of stay not reported; no residual worm on ultrasonography on POD 5              |
| Demessie et al., 2024 (Ethiopia) <sup>35</sup> | 28 years, female | Sealed gallbladder perforation with gallstones; choledocholithiasis and a dead worm in a 2-cm CBD; cholangitis                                          | Laparotomy, cholecystectomy, CBD exploration with T-tube drainage; anthelmintic therapy                          | Length of stay not reported; uneventful recovery                                       |
| Ramos et al., 2024 (Ecuador) <sup>5</sup>      | 43 years, female | Cholangitis with a 12 × 1.5-cm worm obstructing the CBD                                                                                                 | Laparoscopy, choledochotomy, and worm extraction; antibiotics and anthelmintics                                  | Drain removed on POD 5, then discharged; full recovery                                 |

| Study, year (country)                                | Age and sex                               | Gallbladder findings and worm                                                            | Management                                                                                  | Length of stay and outcome         |
|------------------------------------------------------|-------------------------------------------|------------------------------------------------------------------------------------------|---------------------------------------------------------------------------------------------|------------------------------------|
| Inyang et al., 2022 (systematic review) <sup>6</sup> | Pooled case reports (children and adults) | Gallbladder involvement in ≈ 2.1% of hepatobiliary ascariasis; adult worms 15–35 cm long | Initial vermifuge therapy; endoscopic or surgical treatment when conservative therapy fails | Pooled length of stay not reported |

Values were taken from the cited articles. Abbreviations: AEC, absolute eosinophil count; CBD, common bile duct; CRP, C-reactive protein; N<sub>2</sub>O, nitrous oxide; POD, postoperative day; RSI, rapid sequence induction.

#### 4. Discussion

This case illustrates the biomechanical, immunochemical, and surgical aspects of isolated gallbladder ascariasis complicated by acute acalculous hydrops.<sup>6,13,32</sup> Soil-transmitted helminthiasis caused by *A. lumbricoides* remains entrenched in rural agricultural communities where untreated shallow groundwater and night-soil crop fertilization sustain fecal-oral transmission.<sup>1,2,4</sup> Yet even in hyperendemic tropical areas, where hepatobiliary ascariasis is a well-recognized cause of acute pyogenic cholangitis and biliary pancreatitis,<sup>4,5,37</sup> gallbladder involvement accounts for only about 2.1% of hepatobiliary cases.<sup>6</sup> This difference reflects the anatomy of the extrahepatic biliary tree.<sup>6,11</sup> Once an adult worm passes the sphincter of Oddi through the ampulla of Vater, the common bile duct offers a straight, low-resistance route to the hepatic hilum.<sup>5,17</sup> By contrast, to enter the gallbladder, a worm such as the 22.0-cm specimen in this patient must negotiate the acute angle of the cystic-choledochal junction and pass along the

narrow cystic duct against the spiral mucosal folds of the valves of Heister.<sup>11–13</sup>

The two-phase course in this patient — 12 months of intermittent postprandial right upper quadrant discomfort (VAS 2–3/10) followed by an abrupt 48-hour escalation to unremitting severe pain (VAS 8/10) with radiation to the right scapula (Boas' sign) — is compatible with a two-stage natural history of gallbladder ascariasis (Figure 5).<sup>6</sup> We postulate that during the 12-month prodrome, the worm lay free in the gallbladder body and that meal-stimulated release of cholecystokinin triggered gallbladder contraction against the mobile parasite, producing transient, self-limiting biliary colic that mimicked functional dyspepsia.<sup>31,32</sup> Forty-eight hours before admission, the cephalad end of the 22.0-cm worm probably migrated back into the infundibulum and became tightly wedged within the valves of Heister, converting a partially patent gallbladder into a completely obstructed closed-loop cavity.<sup>14,28,33</sup>

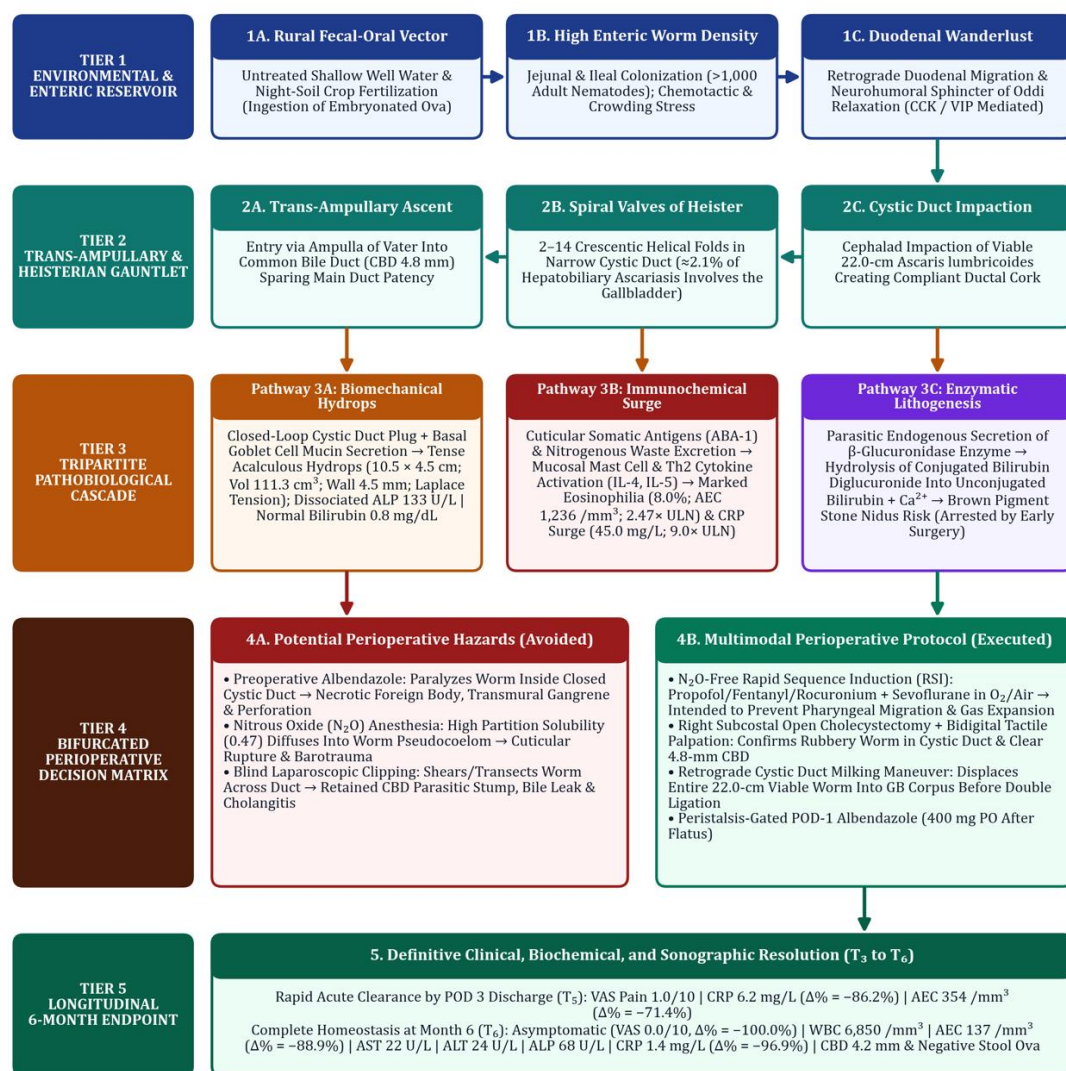


**Figure 5.** Pathophysiological cascade and perioperative decision pathway in isolated gallbladder ascariasis. Tier 1: fecal-oral transmission from untreated shallow-well water and night-soil agriculture, leading to a high jejunal worm burden (> 1,000 worms) and duodenal migration. Tier 2: trans-ampullary ascent and passage through the valves of Heister (gallbladder involvement in approximately 2.1% of hepatobiliary ascariasis), with cephalad impaction in the cystic duct. Tier 3: three parallel processes — closed-loop mucin hydrops (111.3

cm<sup>3</sup>) with dissociated ALP elevation (133 U/L); cuticular antigen-driven Th2/IL-5 eosinophilia (1,236/mm<sup>3</sup>) and CRP rise (45.0 mg/L); and  $\beta$ -glucuronidase-mediated bilirubin deconjugation. Tier 4: potential perioperative hazards that were avoided, contrasted with the protocol used (N<sub>2</sub>O-free rapid sequence induction, open cholecystectomy with retrograde cystic duct milking, and albendazole 400 mg on POD 1). Tier 5: clinical, biochemical, and sonographic resolution by month 6.

The rapid development of tense acalculous gallbladder hydrops measuring 10.5 × 4.5 cm (estimated ellipsoid volume 111.3 cm<sup>3</sup>; +147.3% above the upper physiological fasting limit of 45.0 cm<sup>3</sup>) within 48 hours of cystic duct impaction can be explained by compliant ductal plugging and Laplace's law of wall stress.<sup>16,33</sup> Unlike a rigid, faceted cholesterol gallstone, which may allow intermittent leakage of bile around its irregular surface, the cylindrical, fluid-pressurized pseudocoelomic cuticle of a viable *A. lumbricoides* may act as a soft hydraulic gasket that conforms closely to the spiral mucosal folds of the valves of Heister.<sup>13,14</sup> Once bile outflow is blocked, the gallbladder epithelium and the tubuloacinar glands of the neck continue their constitutive secretion of mucin glycoproteins and water into the closed lumen.<sup>14,38</sup> As the intraluminal volume expands to 111.3 cm<sup>3</sup> and the radius of the gallbladder reaches 2.25 cm, Laplace's law for a cylindrical viscus (wall stress =  $[P \times r]/w$ ) predicts a rise in circumferential wall stress.<sup>15,16</sup> This wall stress compresses intramural venous and lymphatic outflow, producing the 4.5-mm concentric submucosal wall edema seen on ultrasonography, and stretches capsular nociceptors supplied by the right phrenic nerve, accounting for the severe visceral pain (VAS 8/10), the radiation to the right scapula, and the equivocal Murphy's sign before transmural bacterial suppuration.<sup>27,34,39</sup> If surgical decompression is delayed, rising wall tension can exceed the cystic arterial perfusion pressure at the watershed fundus, leading to ischemic gangrene, free perforation into the peritoneal cavity, or fistulation into adjacent viscera, as reported in delayed surgical series.<sup>15,35,40,41</sup>

Beyond mechanical hydrops, the serial laboratory findings show the immunochemical and enzymatic features that distinguish parasitic acalculous hydrops from lithogenic cholecystitis or acalculous cholecystitis of critical illness.<sup>7,34,42,43</sup> Whereas calculous cholecystitis usually causes a neutrophilic leukocytosis, our patient had a pronounced peripheral eosinophilia of 8.0%, with an AEC of 1,236/mm<sup>3</sup> ( $R_{ULN} = 2.47$ ), together with a ninefold rise in CRP (45.0 mg/L;  $R_{ULN} = 9.00$ ). This profile is attributed to continuous shedding of immunogenic somatic cuticular antigens and nitrogenous metabolic waste by the viable worm in the gallbladder lumen, which activate mucosal mast cells and Th2 lymphocytes to secrete IL-4 and IL-5, stimulating eosinophil release from the bone marrow and eosinophilic infiltration of the gallbladder wall.<sup>7,43,44</sup> At the same time, the patient showed a dissociated biochemical pattern: ALP was raised at 133 U/L ( $R_{ULN} = 1.27$ ) with mild transaminase elevation (AST 59 U/L; ALT 73 U/L), whereas total bilirubin was normal at 0.8 mg/dL ( $R_{ULN} = 0.67$ ). In hepatobiliary surgery, a raised ALP with a normal serum bilirubin is consistent with localized biliary hypertension in a duct that does not drain the liver: because the common bile duct was unobstructed (4.8 mm), hepatic bilirubin excretion continued normally, whereas high pressure within the obstructed cystic duct and hydropic gallbladder may have induced local release of ALP from stressed biliary ductular epithelium and adjacent pericholecystic hepatocytes.<sup>19,37,45,46</sup>

The postoperative course is consistent with prompt surgical source control. After open cholecystectomy and removal of the 22.0-cm worm at T<sub>2</sub>, CRP fell from 45.0 to 6.2 mg/L by POD 3 ( $\Delta\% = -86.2\%$ ), a decline consistent with the short plasma half-life of human CRP (approximately 19–24 hours)<sup>47</sup> and with cessation of the inflammatory stimulus after removal of the hydropic gallbladder and the parasite. Similarly, the AEC fell by

71.4% by POD 3 and by 88.9% by month 6, while AST, ALT, and ALP returned to the reference range by discharge and remained normal at 6 months. Early surgery also pre-empted the third pathological mechanism of biliary ascariasis, enzymatic lithogenesis driven by  $\beta$ -glucuronidase, which deconjugates bilirubin diglucuronide and promotes the formation of brown calcium bilirubinate stones around dead worm fragments.<sup>18,21</sup>

From a diagnostic imaging standpoint, this case shows why real-time transabdominal ultrasonography is preferred to computed tomography (CT) for suspected hepatobiliary parasitosis.<sup>4,17,20</sup> On static cross-sectional CT, an uncalcified worm in the lumen may have an attenuation similar to that of inspissated mucinous sludge, fibrinous exudate, or clot from a ruptured cystic artery pseudoaneurysm.<sup>4,48</sup> In contrast, high-frequency B-mode ultrasonography provided high spatial resolution in real time, showing the transverse concentric inner tube sign, the longitudinal parallel stripe sign, the absence of posterior acoustic shadowing, and live non-directional writhing movement while localizing the cephalad impaction in the cystic duct.<sup>18,21,22</sup>

The main clinical contribution of this report is the description of perioperative measures that address the specific anesthetic, intraoperative, and pharmacological pitfalls of a worm impacted in the cystic duct.<sup>13,22</sup> First, in anesthesia, rapid sequence induction (RSI) was combined with avoidance of nitrous oxide (N<sub>2</sub>O). Because *A. lumbricoides* may respond to light anesthesia or volatile irritation with vigorous retrograde migration, RSI rapidly secures the airway and may reduce the risk of pharyngeal worm migration and laryngospasm, airway complications that have been reported with ascariasis during anesthesia.<sup>36</sup> Avoiding N<sub>2</sub>O was also considered important: because of its high blood:gas partition coefficient (0.47, compared with 0.015 for nitrogen), N<sub>2</sub>O diffuses rapidly into gas-containing spaces, including the bowel lumen;<sup>49</sup> the authors also considered its diffusion into the pseudocoelomic digestive tract of the worm, with a risk of intraluminal expansion, cuticular rupture with release of the anaphylactogenic ABA-1 allergen, or acute perforation of the tense hydropic gallbladder.<sup>35,50</sup>

Second, in the operative domain, open cholecystectomy through a right subcostal (Kocher) incision with retrograde cystic duct milking allowed the live worm impacted in the cystic duct to be cleared before ligation in this patient.<sup>8,13,33</sup> Although conservative anthelmintic therapy can succeed when a worm lies free in an unobstructed gallbladder,<sup>32,51</sup> paralysis of a worm wedged within the valves of Heister may trap a necrotic foreign body inside a tense, closed mucocoele, leading to gangrenous cholecystitis or perforation.<sup>6,35,52</sup> Similarly, although ERCP is effective for extracting worms from the main bile duct,<sup>24,25</sup> endoscopic baskets cannot traverse the tortuous valves of Heister.<sup>22,24</sup> During cholecystectomy, standard laparoscopic instruments do not provide direct bidigital tactile feedback; applying a surgical clip across an edematous cystic duct that contains an unrecognized segment of a 22.0-cm worm risks either incomplete clip closure with bile leakage or mechanical transection of the worm, leaving a necrotic caudal remnant in the common bile duct that may cause postoperative cholangitis or stricture.<sup>6</sup> Open tactile palpation identified the rubbery worm in the cystic duct, and gentle retrograde milking with finger pressure and an atraumatic bowel grasper displaced the entire 22.0-cm viable worm back into the gallbladder body before

suture ligation, leaving a flat, empty cystic duct and a clear 4.8-mm common bile duct without the need for choledochotomy. Third, delaying the single 400-mg oral dose of albendazole until POD 1 (T<sub>3</sub>), after the passage of flatus had confirmed active intestinal peristalsis, allowed any jejunal worms to be expelled without the risk of a paralytic bolus obstruction in the ileum,<sup>3,50</sup> and the hospital stay was 3 days, with no recurrence during 6 months of follow-up.

The strengths of this report include the prospective seven-point observation schedule (T<sub>0</sub>–T<sub>6</sub>), the complete serial laboratory and sonographic documentation over 6 months, the explicit avoidance of inferential statistics, which do not apply to a single patient, and reporting in accordance with SCARE 2023.<sup>29</sup> Its limitations include the single patient, inherent to an uncommon surgical condition (approximately 2.1% of hepatobiliary ascariasis), so that the laboratory trends describe this case rather than a population; reliance on a normal bilirubin (0.8 mg/dL), preoperative ultrasonography (common bile duct 4.8 mm), and intraoperative bidigital palpation rather than routine intraoperative cholangiography to confirm clearance of the common bile duct; and the inference, rather than direct observation, of the prodromal course. Nevertheless, the normalization of all liver enzymes and a 4.2-mm common bile duct on ultrasonography at 6 months support the outcome of the operative approach in this patient.

From a pathophysiological and histomorphological perspective, the mucosal environment of an obstructed, hydropic gallbladder containing a viable *A. lumbricoides* differs fundamentally from that of acute calculous cholecystitis.<sup>7,16,34</sup> In lithogenic cholecystitis, impaction of a cholesterol or mixed pigment stone in Hartmann's pouch causes mechanical decubitus ulceration of the mucosa, while supersaturated biliary cholesterol and hydrophobic bile salts, chiefly chenodeoxycholic and deoxycholic acid, solubilize epithelial membrane phospholipids and activate phospholipase A<sub>2</sub>, converting membrane lecithin into cytotoxic lysolecithin.<sup>16,34,42</sup> Secondary bacterial translocation by enteric Gram-negative rods then converts the stagnant bile into a suppurative empyema with transmural neutrophilic infiltration, mucosal sloughing, and micro-abscess formation.<sup>15,41,42</sup> By contrast, in isolated gallbladder ascariasis with acute acalculous hydrops, the luminal fluid is sterile, clear-to-opalescent mucin secreted by hyperplastic goblet cells against a compliant cuticular plug, and the cellular infiltrate is dominated by degranulating eosinophils and mast cells recruited by parasitic cuticular allergens and Th2 cytokine signaling (IL-4, IL-5, and IL-13).<sup>7,43,44</sup> This distinction may explain why our patient remained afebrile (36.1°C), with an equivocal Murphy's sign at admission, despite a tense 10.5 × 4.5-cm gallbladder (111.3 cm<sup>3</sup>), a ninefold rise in CRP (45.0 mg/L), and an AEC of 1,236/mm<sup>3</sup>.<sup>7,14</sup>

In addition, the comparative performance of hepatobiliary imaging in resource-limited endemic hospitals has practical implications for surgical triage.<sup>4,17,20</sup> Although magnetic resonance cholangiopancreatography (MRCP) and endoscopic ultrasound (EUS) provide high contrast resolution of the extrahepatic biliary tree, showing an intraluminal worm as a linear T2-hypointense filling defect within hyperintense bile, both are expensive, not portable, and rarely available as emergency examinations in rural agricultural districts where ascariasis is hyperendemic.<sup>4,51</sup> Multidetector computed tomography (MDCT) is more widely available in regional hospitals, yet uncalcified worms within mucinous gallbladder fluid have attenuation values (20–40 HU) that overlap substantially with inspissated biliary sludge, fibrinous purulent exudate, or organizing hemobilia.<sup>4,34,48</sup> Consequently, real-time

B-mode ultrasonography at the bedside is a rapid, inexpensive, and specific test: by scanning the gallbladder in orthogonal planes, the sonographer can measure the dimensions for the estimated volume (111.3 cm<sup>3</sup>), quantify concentric submucosal wall edema (4.5 mm), confirm the absence of shadowing calculi, observe the characteristic inner tube and stripe signs, and confirm worm viability by its writhing movement while assessing the caliber of the common bile duct (4.8 mm).<sup>4,17,21</sup>

A further surgical consideration concerns the management of the common bile duct during open cholecystectomy for gallbladder ascariasis.<sup>5,22</sup> When a patient presents with synchronous choledochal ascariasis, manifested by obstructive jaundice (total bilirubin > 2.0–18.0 mg/dL), ascending cholangitis (Charcot's triad), and sonographic dilatation of the common bile duct (> 6.0–20.0 mm) containing linear echogenic structures, preoperative endoscopic extraction by ERCP or intraoperative supraduodenal choledochotomy with T-tube drainage is indicated to decompress the main biliary tree.<sup>5,22,24</sup> However, an unnecessary choledochotomy in a patient with a common bile duct of normal caliber (< 6.0 mm) carries substantial iatrogenic morbidity, including bile leakage, ampullary edema, and late stricture.<sup>13,22,33</sup> In this patient, three findings allowed routine choledochotomy to be omitted: (1) a normal preoperative serum total bilirubin (0.8 mg/dL; R<sub>ULN</sub> = 0.67); (2) a normal preoperative sonographic diameter of the common bile duct (4.8 mm) without intraluminal echoes; and (3) normal intraoperative bidigital palpation of the entire supraduodenal common bile duct after retrograde milking of the cystic duct worm into the gallbladder body. Recovery was accordingly rapid, and the patient was discharged on POD 3.<sup>8</sup>

From a clinical pharmacology and parasitological perspective, the interaction between benzimidazole carbamates and intestinal motility after abdominal surgery deserves attention.<sup>2,22,31</sup> Albendazole binds selectively and with high affinity to parasitic  $\beta$ -tubulin, inhibiting microtubule polymerization in the intestinal cells of *A. lumbricoides*, impairing glucose uptake, depleting glycogen stores, and reducing adenosine triphosphate synthesis until the worm is immobilized and dies.<sup>31</sup> After oral administration, albendazole is poorly absorbed in the fasting state but undergoes rapid first-pass hepatic oxidation by flavin monooxygenases and cytochrome P450 enzymes to its active metabolite, albendazole sulfoxide, which reaches peak plasma concentrations within 2–5 hours and has an elimination half-life of 8.5 hours.<sup>53</sup> If albendazole is given preoperatively or during the first 12–24 hours after laparotomy, while intestinal myoelectric activity is inhibited by postoperative ileus, paralyzed jejunal worms cannot be propelled aborally and may coalesce into a tangled bolus at the ileocecal valve, causing secondary mechanical small bowel obstruction.<sup>3,50</sup> By timing the single 400-mg oral dose of albendazole to the clinical return of peristalsis (passage of flatus at hour 24 on POD 1), the treatment ensured that drug-induced immobilization of the worms coincided with active propulsive peristalsis; there were no gastrointestinal or hepatotoxic adverse events, and no ova were found in the stool at 6 months.<sup>3,50</sup>

To appreciate the mechanics of the retrograde cystic duct milking maneuver, it is useful to consider the micro-anatomy of the cystic-choledochal confluence and the biomechanical properties of the worm's cuticle.<sup>11,12</sup> The adult female *A. lumbricoides* has a multilayered extracellular cuticle composed of cross-linked collagenous fibers arranged in opposing helical arrays at approximately 70 degrees to the longitudinal body axis, enclosing a high-pressure pseudocoelomic hydrostatic skeleton maintained at an internal turgor pressure of 70–120

mmHg.<sup>4,13,31</sup> This internal pressure gives the living worm a firm, rubbery, incompressible cylindrical consistency that resists external crushing while enabling powerful sinusoidal movement.<sup>5,10</sup> When the cephalad end of a 22.0-cm adult worm enters the 2–3-mm lumen of the cystic duct, the crescentic mucosal folds of the valves of Heister may act as unidirectional mechanical ratchets: forward undulatory propulsion wedges the tapered cephalad end deeper between successive spiral folds, whereas passive backward recoil is resisted by cuticular friction against the mucosal folds.<sup>11,12</sup>

Once wedged across the valves of Heister, the turgid worm converts the gallbladder into an isolated, compliant hydraulic chamber.<sup>14,38</sup> Under normal fasting conditions, the gallbladder stores 30.0–45.0 cm<sup>3</sup> of hepatic bile while the mucosal epithelium absorbs sodium, chloride, and water through apical Na<sup>+</sup>/H<sup>+</sup> exchangers, maintaining a low resting intraluminal pressure of 5–10 mmHg.<sup>42</sup> However, when cystic duct outflow is sealed by an impacted worm and the mucosa is stimulated by parasitic excretory-secretory products and local prostaglandin E<sub>2</sub> release, net fluid transport reverses from absorption to active secretion of mucin and electrolytes.<sup>38</sup> Within 48 hours of complete ductal impaction, our patient's gallbladder had distended to 10.5 × 4.5 cm, corresponding to an estimated ellipsoid volume of 111.3 cm<sup>3</sup>, a 2.47-fold expansion above the upper physiological limit.<sup>27,33,39</sup> Applying Laplace's law for a cylindrical viscus (wall stress =  $[P \times r]/w$ ), with a radius of 2.25 cm and an intraluminal pressure that rose above normal venous capillary pressure (> 25–30 mmHg), circumferential wall stress would have increased markedly despite the reactive 4.5-mm submucosal edema.<sup>40</sup> This sustained wall stress may explain why patients with acute acalculous gallbladder hydrops have unremitting, severe visceral pain (VAS 8/10 in this patient) and phrenic nerve referral to the right scapula (Boas' sign) even in the absence of bacterial empyema.<sup>27</sup>

The distinction between open tactile decompression and laparoscopic clipping without tactile feedback in cystic duct ascariasis also has implications for hepatobiliary patient safety.<sup>13,26,33</sup> In contemporary surgical practice, laparoscopic cholecystectomy is rightly regarded as the standard of care for uncomplicated calculous cholecystitis and long-standing calculous hydrops.<sup>33,39,45</sup> However, laparoscopic graspers and clip applicators transmit less than 15% of the spatial haptic resolution of the human fingertips.<sup>26,45</sup> When an edematous gallbladder neck with a 4.5-mm wall and a dilated cystic duct are inspected laparoscopically, a viable 22.0-cm worm extending across the lumen of the cystic duct cannot be seen through the opaque duct wall. If clips are applied across the cystic duct without recognition of the intraluminal parasite, the clip jaws may either fail to lock securely across the thick collagenous cuticle, causing a high-output postoperative bile leak from the cystic duct stump, or shear the worm transversely.<sup>6,13,21</sup> Transection of a live *Ascaris* at the cystic-choledochal junction leaves a devitalized, antigen-rich caudal segment in the common bile duct and spills pseudocoelomic fluid into Calot's triangle, which may trigger eosinophilic peritonitis, necrotizing cholangitis, or late biliary stricture.<sup>35</sup>

By contrast, the right subcostal (Kocher) laparotomy allowed direct bidigital palpation of Calot's triangle between the thumb and index finger, immediately identifying the firm, rubbery worm in the cystic duct and confirming that the supraduodenal common bile duct (4.8 mm) was free of worms and calculi.<sup>8,13,22</sup> The retrograde cystic duct milking maneuver — stabilizing Hartmann's pouch with an atraumatic bowel grasper while applying sequential, gentle digital compression from the juxta-choledochal cystic duct toward the gallbladder infundibulum —

overcame the frictional resistance of the valves of Heister without rupturing the parasitic cuticle.<sup>13,33</sup> Once the entire 22.0-cm worm had slipped back into the gallbladder body, repeat palpation confirmed a flat, empty, pliable cystic duct that could be doubly ligated with absorbable sutures without tension, securing the duct and allowing a 3-day postoperative recovery.<sup>6,8,13</sup>

Equally important to the long-term success of surgical treatment in endemic rural areas is the integration of perioperative anesthetic gas physics with postoperative public health measures.<sup>1,2,51</sup> During general anesthesia, avoidance of nitrous oxide (N<sub>2</sub>O) is an important but often overlooked precaution in patients with hepatobiliary or intestinal ascariasis.<sup>49</sup> Because the blood:gas partition coefficient of N<sub>2</sub>O (0.47) is more than thirty times that of nitrogen (0.015), N<sub>2</sub>O enters gas-containing spaces about thirty times faster than nitrogen can diffuse out.<sup>49</sup> Within the pseudocoelomic digestive tract of an impacted *Ascaris* and within the surrounding small bowel, N<sub>2</sub>O diffusion could cause rapid volume expansion that might rupture the cuticle of the worm or perforate an ischemic, hydropic gallbladder wall.<sup>15,35</sup> Combining N<sub>2</sub>O-free sevoflurane maintenance with rapid sequence endotracheal induction (propofol, fentanyl, and rocuronium) aimed both to prevent excitatory pharyngeal worm migration during induction and to avoid gas expansion during laparotomy.<sup>36,49</sup> Finally, coupling albendazole (400 mg on POD 1 after the return of flatus) with structured household water, sanitation, and hygiene (WASH) measures, specifically boiling shallow-well water for at least one minute and ending exposure to night soil, aimed to interrupt the rural fecal-oral reinfection cycle, and the patient remained clinically, biochemically, and sonographically well at 6 months.<sup>1,2,51</sup>

## 5. Conclusion

Isolated gallbladder ascariasis causing acute acalculous gallbladder hydrops is an uncommon surgical emergency (approximately 2.1% of hepatobiliary ascariasis) caused by migration of a viable adult *A. lumbricoides* through the valves of Heister and its impaction in the cystic duct, which closes the gallbladder outlet. In endemic tropical regions, surgeons and radiologists should suspect this condition in patients with acute-on-chronic right upper quadrant pain, a palpable hydropic gallbladder, marked peripheral eosinophilia (AEC 1,236/mm<sup>3</sup> in this patient), and dissociated anicteric cholestasis (raised ALP of 133 U/L with a normal total bilirubin of 0.8 mg/dL). Real-time transabdominal ultrasonography can establish the diagnosis by showing the inner tube sign, the stripe sign, and live writhing movement inside a hydropic gallbladder without acoustic shadowing. Cholecystectomy should be considered when a worm is impacted in the cystic duct with hydrops. In this patient, retrograde cystic duct milking to displace the impacted worm into the gallbladder before ligation of the cystic duct, combined with rapid sequence induction without nitrous oxide and oral albendazole (400 mg on POD 1) after the return of bowel function, allowed the worm to be removed intact without transection or bile leakage and was followed by full clinical and laboratory recovery at 6 months.

## 6. Declarations

### Ethics Approval and Consent to Participate

Ethical approval was granted by CMHC Research Center Ethics Committee, Indonesia (protocol No. 2026/0418). All procedures were performed in accordance with the ethical standards of the Department of Surgery, Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin General

Hospital, Palembang, and Dr. H. Mohamad Rabain Regional General Hospital, Muara Enim, Indonesia, and with the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from the patient and her legal family representative before participation.

### Consent for Publication

Written informed consent was obtained from the patient for publication of this case report, the accompanying laboratory and sonographic data, and the anonymized intraoperative specimen photograph, in accordance with ICMJE privacy recommendations.

### Availability of Data and Materials

All longitudinal data generated and analyzed for this report are included in the article, its tables, and its figures.

### Artificial Intelligence Use Statement

No artificial intelligence tools were used in the preparation of this article.

### Author Contribution Statement

M.A.D.P.: Conceptualization, Methodology, Investigation, Data curation, Formal analysis, Writing – original draft. A.F.H.: Investigation, Data curation, Validation. A.H.: Investigation, Methodology, Writing – review & editing. A.A.: Supervision, Validation, Writing – review & editing. All authors reviewed and approved the final version of the manuscript.

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### Conflict of Interest / Competing Interests

The authors declare that they have no competing financial or non-financial interests.

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