Diagnostic Evaluation of Feline (*Felis catus*) Cholangiohepatitis: Insights from X-ray, Ultrasonography, and Hematology

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ABSTRACT

Feline cholangiohepatitis is a significant inflammatory hepatobiliary disease causing major morbidity in cats. Its clinical relevance is underscored by substantial diagnostic challenges, primarily attributable to nonspecific clinical manifestations like jaundice, anorexia, and lethargy. Furthermore, definitive diagnosis necessitates invasive procedures, including hepatic biopsy, making noninvasive evaluation crucial. A two-year-old intact female domestic cat, weighing exactly 3 kg, was presented for veterinary evaluation due to jaundice, severe lethargy, and complete anorexia. Based on initial clinical assessment, a presumptive diagnosis of cholangiohepatitis was considered, prompting comprehensive diagnostic investigation. The diagnostic evaluation revealed significant abnormalities on imaging and biochemical tests. Abdominal radiography demonstrated marked hepatomegaly, while ultrasonography highlighted gallbladder dilation and marked wall thickening. Biochemical profiling also indicated severe hepatic and biliary involvement, with markedly elevated serum aspartate aminotransferase, alanine aminotransferase, and gamma glutamyl transferase.Diagnostic imaging revealed significant gallbladder enlargement, indicative of severe and acute biliary inflammation and concurrent hepatic involvement. Collectively, these imaging and biochemical abnormalities indicated severe hepatobiliary inflammation, strongly supporting a presumptive clinical diagnosis of cholangiohepatitis. This clinical case illustrates the complementary diagnostic benefit of combining abdominal radiography, ultrasonography, and serum biochemical testing when assessing feline hepatobiliary disease. Here, integrated radiographic findings of hepatomegaly, ultrasonographic abnormalities, and pronounced biochemical increases substantiated a clinical diagnosis of cholangiohepatitis. These findings highlight the necessity of a multimodal diagnostic strategy to accurately define the degree of hepatic and biliary involvement, offering a dependable basis for decision-making in comparable feline cases.

Keywords: Cholangiohepatitis; Feline; Hepatobiliary; Prognosis; Therapy

1. Introduction

Feline cholangiohepatitis is a complex inflammatory hepatobiliary disease characterized by the infiltration of inflammatory cells into the bile ducts and surrounding hepatic parenchyma. The pathogenesis is frequently associated with an ascending bacterial infection originating from the gastrointestinal tract or pancreas into the biliary system. Based on histopathological features, the disease is broadly classified into two main forms: neutrophilic (suppurative) cholangitis, which is typically acute and often linked to bacterial infection, and lymphocytic (lymphoplasmacytic) cholangitis, which is generally chronic and immune-mediated [1, 2]. Furthermore, cholangiohepatitis is a core component of feline triaditis, a syndrome where concurrent inflammatory bowel disease (IBD) and pancreatitis frequently co-occur, thereby complicating the clinical picture and obscuring the primary hepatobiliary pathology [3].

The clinical presentation of feline cholangiohepatitis is notoriously heterogeneous and non-specific. Affected cats commonly exhibit lethargy, anorexia, weight loss, vomiting, and occasionally icterus or hepatomegaly. Because these signs overlap significantly with other systemic feline diseases, particularly the gastrointestinal and pancreatic components of triaditis, clinical Diagnosis suspect remains highly challenging. The prognosis is highly variable and largely dependent on the underlying histopathological classification, the chronicity of the disease at the time of presentation, and the presence of concurrent conditions. While early and aggressive intervention can yield a favorable outcome in acute neutrophilic cases, chronic lymphocytic forms or those with advanced hepatic fibrosis carry a guarded to poor prognosis [2, 4].

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Establishing a definitive diagnosis of cholangiohepatitis presents substantial challenges. Currently, histopathological evaluation via hepatic biopsy is considered the gold standard, allowing for definitive classification and etiological confirmation. However, this invasive procedure carries inherent risks, particularly in critically ill or coagulopathic patients, and may not be universally available or financially feasible for all owners. Consequently, clinicians must rely on a combination of serum biochemical profiling and diagnostic imaging. While elevations in liver enzymes (such as ALT, AST, ALP, and GGT) and hyperbilirubinemia indicate hepatobiliary injury and cholestasis, these biochemical markers lack absolute specificity. Similarly, imaging modalities like ultrasonography can reveal hepatomegaly, gallbladder abnormalities, or biliary dilation, but these findings are not pathognomonic for cholangiohepatitis and must be interpreted with caution [5, 6].

Despite the recognized limitations of non-invasive diagnostics, there is a distinct knowledge gap in the literature regarding the practical, integrated application of these accessible tools in clinical settings where biopsies are not routinely performed. Furthermore, detailed case reports documenting the step-by-step diagnostic reasoning and the complementary value of combining radiography, ultrasonography, and specific biochemical panels in the Indonesian feline practice are scarce. Therefore, this case report aims to bridge this gap by detailing the clinical, biochemical, and imaging findings of a cat with suspected cholangiohepatitis. By critically evaluating the utility and limitations of these non-invasive diagnostic modalities in this specific case, this report provides a practical framework for clinicians to optimize diagnostic accuracy and initiate timely therapeutic interventions in similar real-world clinical scenarios.

2. Case Presentation

2.1. Signalment and Clinical History

A 2-year-old, 3.0 kg, intact female Domestic cat named Puspita (Figure 1) was presented to the Surgery and Radiology laboratory of the Veterinary Medicine faculty, Universitas Wijaya Kusuma Surabaya. The history included progressive lethargy, complete anorexia, and noticeable yellowing of the skin. The cat is primarily fed a commercial dry food and has an outdoor lifestyle. The vaccination history is unknown, and the last deworming was administered at an unknown. There was no significant previous medical history, and no prior treatments or medications were administered by the owner before this presentation.

2.2. Physical Examination

Upon initial presentation, the cat exhibited a lethargic mental status and a weak body condition. The Body Condition Score (BCS) was assessed as 2/5. Vital parameters included a rectal temperature of 39.1°C, a heart rate of 245 beats/minute, and a respiratory rate of 30 breaths/minute. Hydration status was estimated at 5% dehydration, indicated by a skin turgor of <2 seconds and slightly tacky mucous membranes. The mucous membranes were distinctly icteric (jaundiced) throughout the oral cavity and sclera, with a capillary refill time (CRT) of <2 seconds. Abdominal palpation revealed marked hepatomegaly and mild to moderate discomfort/pain upon cranial abdominal palpation. No other significant abnormalities were detected on the general physical examination.

Following the diagnostic workup, the cat was returned to the shelter while still symptomatic. The responsibility for subsequent therapeutic management was transferred to the shelter management to facilitate potential referral or palliative care. As the scope of this report was strictly limited to the diagnostic evaluation, an in-hospital treatment protocol was not implemented by the authors. Furthermore, no follow-up information regarding the patient's clinical outcome has been received from the shelter to date.

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Figure 1. Puspita Cat

2.3. Laboratory investigation: Based on the symptoms, an ultrasound (Figure 2), an X-ray (Figure 3), and a blood chemistry test (Table 1) were performed. The results showed increased AST, ALP, and GGT levels. Figure 2 shows the ultrasound examination.

Diagnostic Imaging: Abdominal ultrasonography was performed using a veterinary ultrasound system (EDAN DUS 60, Edan Instruments, Inc., Shenzhen, China) equipped with a sector-type probe operating at a frequency of 5.4 MHz. Before the examination, the right lateral abdominal region was clipped and prepared with acoustic coupling gel. The patient was positioned in both dorsal and right lateral recumbency to systematically evaluate the hepatic parenchyma, biliary system, and surrounding abdominal viscera. Concurrently, abdominal radiography was conducted using a portable veterinary X-ray unit (DFX-100L, omit, Xindray Medical International Co., Limited, Nanjing, Jiangsu, China). Standard views, specifically the right lateral and ventrodorsal projections, were obtained. The radiographic exposure factors were set at 50 kVp and 70 mA. Radiographic evaluation primarily focused on assessing hepatic size, silhouette, and the presence of any abdominal effusion or mass effect.

Clinicopathological Analysis: Venous blood samples were collected via the cephalic vein into serum separator tubes. Serum biochemical analysis was performed at the Klinik Pacar Laboratory, Surabaya, Indonesia. The analytical platform used for the serum biochemical profiling was an automated biochemistry analyzer. The specific serum parameters evaluated to assess hepatic and biliary function included aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), total bilirubin, and total protein. All analyses were conducted in accordance with the manufacturer's standardized protocols.

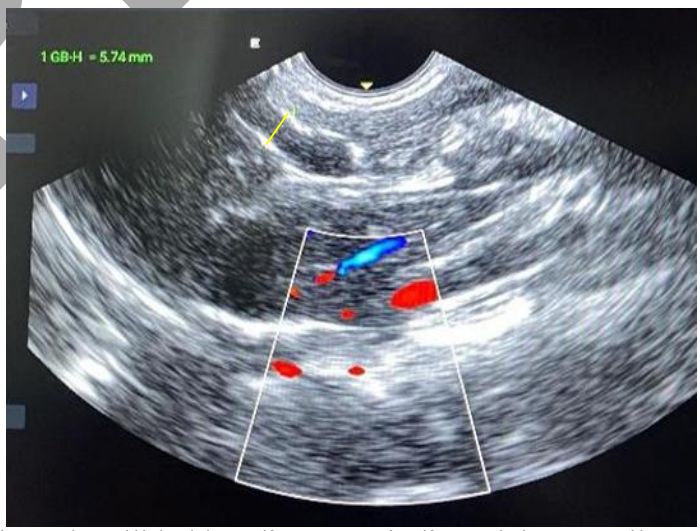


Figure 2. An enlarged gallbladder diameter, indicated by a yellow line, is identified on ultrasonography using a sector-type probe.

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Ultrasound findings

Abdominal ultrasonography revealed distinct hepatobiliary abnormalities consistent with hepatobiliary inflammation. The hepatic parenchyma exhibited heterogeneous echotexture with diffuse hyperechogenicity compared to the normal echogenicity of the adjacent spleen. The gallbladder was markedly distended, measuring approximately 5.74 mm in diameter (Figure 2), which exceeds the normal reference range for feline patients. The gallbladder wall appeared thickened and irregular. Color Doppler evaluation demonstrated preserved vascular flow within the hepatic parenchyma. The intrahepatic and extrahepatic biliary ducts appeared mildly dilated. No evidence of abdominal effusion, focal hepatic masses, or significant lymphadenopathy was detected during the examination.

Radiographic Findings

Abdominal radiographs obtained in right lateral and ventrodorsal views demonstrated marked hepatomegaly. The hepatic margins extended significantly beyond the costochondral junctions with prominent caudal displacement (indicated by red arrows, Figure 3). The enlarged liver lobes caused a dorsal displacement of the gastric axis, as evidenced by the altered position of the gastric silhouette on the lateral view. The hepatic margins appeared rounded and smoothly lobulated. There was mild loss of serosal detail in the cranial abdomen, suggestive of periportal edema or mild inflammatory changes. The remaining abdominal organs appeared unremarkable, and no abnormal soft tissue masses were identified.

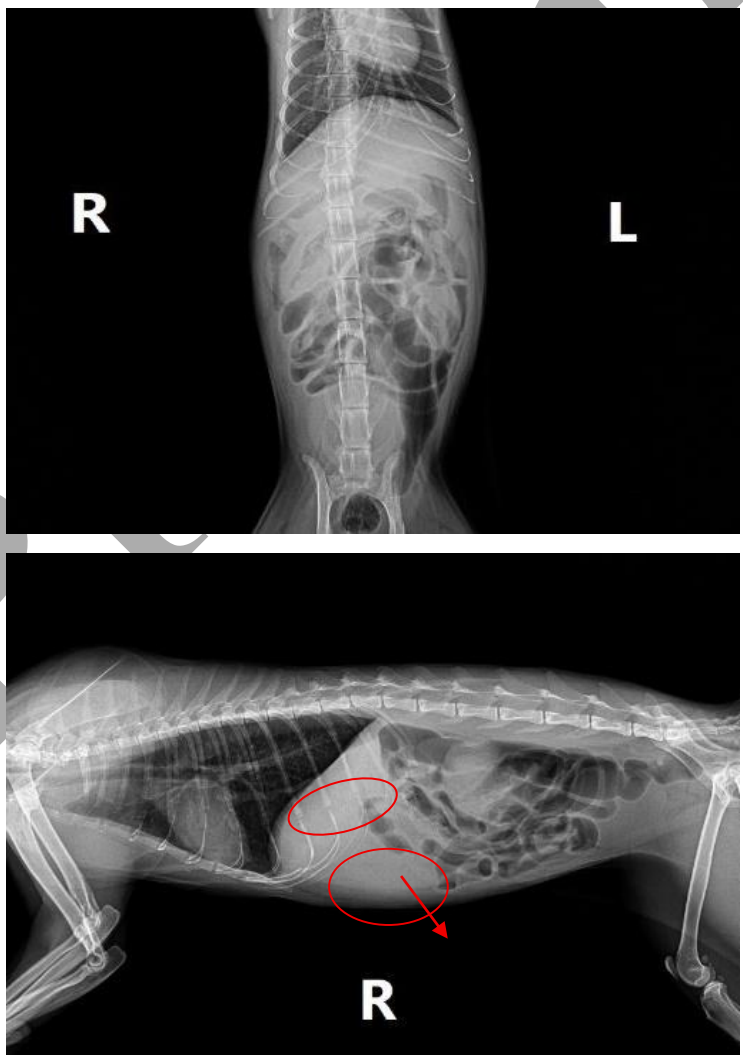


Figure 3. The X-ray examination reveals hepatomegaly (or an) enlarged liver, indicated by a rounded area in the apex (red arrow).

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Table 1. Results of Hematology Examination of the Liver Organ: an increase in AST, ALT, and Gamma GT levels

No	Examination	Result	Normal value
1	Aspartate aminotransferase (AST)	63	8.9- 48.5 U/L
2	Alanine aminotransferase (ALT)	79	8.2- 57.3 U/L
3	Alkaline Phosphatase (ALP)	13	10.6- 100.7 U/L
4	Gamma-Glutamyl Transferase (GGT)	19	1.0- 8.7 U/L

A notable limitation of this case report is the reliance on a targeted serum biochemical panel (ALT, AST, ALP, and GGT) rather than a comprehensive laboratory evaluation. The absence of a complete blood count (CBC), serum bilirubin, albumin, and total protein measurements restricts the ability to fully assess systemic inflammation, hepatic synthetic function, and the complete extent of cholestasis. This targeted diagnostic approach was necessitated by the practical and financial constraints inherent to the shelter medicine setting in which this case was evaluated [7]. Future case reports in similar settings would benefit from advocating for more comprehensive baseline diagnostics whenever feasible

3. Results and Discussion

Results:

Clinical and Diagnostic Findings

A 2-year-old, 3.0 kg domestic cat presented with a history of progressive lethargy, complete anorexia, and icterus. Physical examination revealed a rectal temperature of 39.1°C, heart rate of 245 beats/minute, respiratory rate of 30 breaths/minute, and generalized icterus of the mucous membranes and sclera. The cat exhibited a weak body condition (BCS 3/9) and dehydration estimated at 5-8%. Abdominal palpation revealed hepatomegaly with mild discomfort upon cranial abdominal palpation.

Abdominal ultrasonography demonstrated hepatomegaly with heterogeneous hepatic parenchymal echogenicity. The gallbladder was distended, measuring 5.74 mm in diameter (reference range: 0.84-4.50 mm), with gallbladder wall thickening approximately 2.0 mm (reference: <1.0 mm). Hyperechoic biliary sludge was present within the gallbladder lumen. The common bile duct appeared mildly dilated. Color Doppler evaluation revealed preserved vascular flow within the hepatic parenchyma.

Abdominal radiographs in right lateral and ventrodorsal projections confirmed marked hepatomegaly, with hepatic margins extending beyond the costochondral junctions and causing dorsal displacement of the gastric axis. The hepatic silhouette appeared enlarged with increased soft tissue opacity and rounded margins.

Serum biochemical analysis revealed elevated activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT), indicating combined hepatocellular injury and cholestasis (Table 1).

4. Discussion

Interpretation of Clinical Findings

The clinical presentation of this case—characterized by icterus, lethargy, anorexia, and fever—is consistent with the classic clinical spectrum of feline cholangiohepatitis reported in previous studies [1, 8]. The presence of visible icterus suggests significant hepatobiliary dysfunction, as approximately 60-70% of cats with neutrophilic cholangitis present with

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jaundice due to impaired bilirubin metabolism and excretion [1, 5]. The acute onset and severity of clinical signs in this patient are more suggestive of neutrophilic (suppurative) cholangitis rather than the chronic, insidious course typically associated with lymphocytic cholangitis [1, 4].

Ultrasonographic Findings and Diagnostic Interpretation

The ultrasonographic findings in this case demonstrate several characteristic features that support the presumptive diagnosis of cholangiohepatitis. The gallbladder diameter of 5.74 mm exceeds the normal reference range for feline patients (0.84-4.50 mm), indicating gallbladder distension [3, 6]. This finding, combined with gallbladder wall thickening (approximately 2.0 mm; reference <1.0 mm) and the presence of hyperechoic biliary sludge, is consistent with inflammatory changes of the biliary system [3, 11]. Previous studies have reported gallbladder abnormalities, including wall thickening and sludge accumulation, in 70-80% of cats with cholangiohepatitis [3, 6].

The heterogeneous hepatic parenchymal echogenicity observed in this case reflects the inflammatory infiltration and architectural disruption characteristic of cholangiohepatitis [1, 5]. This pattern differs from the diffuse hyperechogenicity typically seen in hepatic lipidosis or the hypoechoic nodules associated with hepatic neoplasia [3, 6]. The mild dilation of the common bile duct further supports biliary tract involvement, as extrahepatic bile duct dilation >4 mm is considered abnormal in cats and suggests obstruction or inflammation [3, 11].

Radiographic Findings and Diagnostic Limitations

The radiographic evidence of marked hepatomegaly, with hepatic margins extending beyond the costochondral junctions and causing dorsal displacement of the gastric axis, provides supportive evidence for significant hepatic pathology [3]. The increased soft tissue opacity and rounded hepatic margins are consistent with hepatic enlargement secondary to inflammation [3, 6]. However, it is important to acknowledge the diagnostic limitations of radiography compared to ultrasonography in evaluating hepatobiliary disease.

While radiography effectively demonstrates hepatomegaly and gross changes in hepatic size and position, it lacks the sensitivity to detect subtle parenchymal changes, biliary tract abnormalities, or gallbladder pathology that are readily visualized with ultrasonography [3, 6]. Radiography cannot differentiate between various causes of hepatomegaly (e.g., inflammation, neoplasia, lipidosis, or congestion) and provides no information about biliary duct dilation, gallbladder wall thickness, or the presence of biliary sludge [3, 6]. Therefore, while radiographic findings in this case support the presence of hepatobiliary disease, ultrasonography provided more specific and diagnostically valuable information regarding the nature and extent of the pathology.

Biochemical Findings and Pathophysiological Significance

The biochemical profile in this case revealed elevations in all four hepatic enzymes (ALT, AST, ALP, and GGT), indicating a mixed pattern of hepatocellular injury and cholestasis. This combined enzyme elevation pattern is characteristic of cholangiohepatitis, where inflammation affects both hepatocytes and the biliary epithelium [1, 7].

Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) are cytosolic enzymes released into circulation following hepatocellular membrane damage [2, 7]. In cats with neutrophilic cholangitis, ALT and AST elevations are typically pronounced (often >5-10 times the upper reference limit), reflecting active hepatocellular necrosis [1, 7]. The magnitude of ALT elevation in this case is consistent with reports of acute hepatocellular injury associated with cholangiohepatitis [2, 7].

Alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) are membrane-bound enzymes associated with the biliary epithelium, and their elevation indicates cholestasis or biliary tract inflammation [2, 7]. In cats, GGT is considered a more sensitive marker of hepatobiliary disease than ALP, as feline ALP has a shorter half-life and is less readily induced

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compared to dogs [2, 7]. The concurrent elevation of both ALP and GGT in this case strongly supports the presence of cholestasis secondary to biliary tract inflammation [1, 7].

The biochemical pattern observed in this case aligns with recent retrospective studies demonstrating that cats with cholangiohepatitis typically exhibit combined elevations of hepatocellular and cholestatic enzymes, reflecting the dual nature of the disease process [2, 7]. However, the absence of bilirubin measurement in this case limits our ability to fully assess the severity of cholestasis, as hyperbilirubinemia is reported in up to 80% of icteric cats with cholangiohepatitis [1, 5].

Differential Diagnosis Considerations

Several differential diagnoses were considered based on the clinical presentation and diagnostic findings. Hepatic lipidosis, a common cause of hepatomegaly and icterus in cats, was considered less likely given the absence of severe obesity or a prolonged history of anorexia preceding clinical signs, and the ultrasonographic finding of heterogeneous rather than diffusely hyperechoic hepatic parenchyma [3, 6]. Hepatic neoplasia (particularly lymphoma) typically presents with focal or multifocal hepatic masses or diffuse nodularity on ultrasonography, which were not observed in this case [3, 6].

Feline infectious peritonitis (FIP) can cause hepatobiliary involvement, but the absence of characteristic findings such as abdominal effusion, pyogranulomatous inflammation on cytology, or typical hematological abnormalities (e.g., lymphopenia, non-regenerative anemia) makes this diagnosis less likely [1, 5]. Acute hepatocellular necrosis due to toxin exposure would typically present with more dramatic enzyme elevations (>20-50 times reference limits) and a history of toxin exposure, which was not reported in this case [1, 5].

The combination of gallbladder wall thickening, biliary sludge, bile duct dilation, and combined hepatocellular-cholestatic enzyme elevations most strongly supports cholangiohepatitis over other differential diagnoses [3, 6, 11]. However, it must be acknowledged that without histopathological confirmation, bacterial culture, or bile cytology, the diagnosis remains presumptive [1, 4].

Etiological Considerations and Diagnostic Limitations

While the clinical and diagnostic findings in this case are consistent with cholangiohepatitis, it is important to acknowledge that the etiology cannot be definitively determined without bacteriological culture, bile cytology, or histopathological examination. The traditional teaching suggests that neutrophilic cholangitis is often associated with ascending bacterial infection from the gastrointestinal tract or pancreas [1, 4, 8]. However, in the absence of positive bacterial culture or cytological evidence of bacteria, attributing the lesions to a bacterial etiology in this case would be speculative.

Recent studies have emphasized that while bacterial infection is a common cause of neutrophilic cholangitis, not all cases have demonstrable bacterial organisms, and some may have immune-mediated or other inflammatory etiologies [1, 2]. Therefore, the discussion of etiology in this case must remain cautious and acknowledge the limitations imposed by the absence of definitive diagnostic procedures.

Limitations

Several limitations of this case report must be acknowledged. The absence of histopathological evaluation via hepatic biopsy precludes definitive classification of the disease as neutrophilic versus lymphocytic cholangitis and prevents identification of specific pathological changes [1, 4]. Furthermore, the lack of bacterial culture and bile cytology limits our ability to confirm or exclude bacterial etiology and to guide targeted antimicrobial therapy [1, 4]. The diagnostic workup was restricted to a targeted biochemical panel (ALT, AST, ALP, GGT) rather than a comprehensive profile including bilirubin, albumin, total protein, and

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complete blood count. This limitation restricts our ability to fully assess hepatic synthetic function, the severity of cholestasis, and the presence of systemic inflammation [2, 7]. Additionally, the absence of therapeutic intervention and follow-up prevents assessment of treatment response and clinical outcome, which would have provided valuable prognostic information. These limitations reflect the practical and financial constraints inherent to the shelter medicine setting in which this case was evaluated, as well as the primary objective of this report, which focused on the diagnostic utility of non-invasive modalities. Future case reports in similar resource-limited environments should endeavor to incorporate comprehensive diagnostics whenever feasible to validate non-invasive predictive models [1, 4].

Furthermore, subsequent follow-up in July 2026 confirmed that the patient had died. The absence of a post-mortem examination (necropsy) precludes the definitive determination of the exact cause of death, although it is presumed to be secondary to the progression of the underlying hepatobiliary pathology.

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Author Contributions

Study concept and design: RW, DL
Acquisition of data: DL, FS, MU, RMWS, JWAP
Drafting of the manuscript: RW, DL
Interpretation analysis: RW, DL, WM
Administrative, technical, and material support: DL, FS, MU, RMWS, JWAP
All authors read and approved the final manuscript.

Conflicts of Interest

All authors have stated that there is no conflict of interest in carrying out this research.

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Data Availability

The data supporting the findings of this study are available in the research records conducted by the researchers.

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