

# Bacterial Hepatitis Caused by *Staphylococcus pseudintermedius* Associated with Hepatic Copper Accumulation in a Dog: A Case Report

Amanda Regina Nadalin <sup>1,\*</sup>, Bruno Marucci Thomaz <sup>2</sup>

<sup>1</sup> School of Veterinary Medicine and Animal Science, University of São Paulo, São Paulo, Brazil.

<sup>2</sup> Veterinarian, Private Practice, São Paulo, Brazil.

\* Correspondence: amanda.nadalin@alumni.usp.br.

**Abstract:** Hepatopathies in dogs often present diagnostic challenges due to the overlap of infectious, metabolic, and inflammatory etiologies. This report describes a case of bacterial hepatitis caused by *Staphylococcus pseudintermedius* associated with hepatic copper accumulation in a dog, emphasizing the diagnostic approach, clinical progression, and therapeutic management. A female dog was presented with nonspecific clinical signs and biochemical evidence of hepatocellular injury. Serial hematological and biochemical analyses revealed progressive liver enzyme alterations, prompting further diagnostic investigation. Definitive diagnosis was established through liver biopsy, histopathological evaluation, bacterial culture with antimicrobial susceptibility testing, and hepatic copper quantification. Histopathology demonstrated inflammatory changes consistent with bacterial hepatitis, along with cholestasis and hepatocellular degeneration. Microbiological culture identified *Staphylococcus pseudintermedius* as the causative agent. Hepatic copper concentration was markedly increased, exceeding reference limits. Targeted antimicrobial therapy and supportive hepatic treatment were initiated, resulting in progressive clinical improvement and gradual normalization of laboratory parameters during follow-up. The temporal response to therapy suggests that bacterial hepatitis was the primary condition, with secondary copper accumulation likely associated with cholestasis. However, limitations in the assessment of copper distribution prevent definitive differentiation between primary and secondary hepatopathy. This case highlights the importance of comprehensive diagnostic evaluation in canine hepatopathies and underscores the potential coexistence of infectious and metabolic factors in liver disease. Recognition of such multifactorial conditions is essential for accurate diagnosis and appropriate therapeutic decision-making in small animal practice.

**Citation:** Nadalin AR, Thomaz BM. Bacterial Hepatitis Caused by *Staphylococcus pseudintermedius* Associated with Hepatic Copper Accumulation in a Dog: A Case Report. Brazilian Journal of Case Reports. 2026 Jan-Dec; 06(1):bjcr174.

<https://doi.org/10.52600/2163-583X.bjcr.2026.6.1.bjcr174>

Received: 9 March 2026

Accepted: 23 March 2026

Published: 25 March 2026

**Keywords:** Canine Hepatopathy; Bacterial Hepatitis; Hepatic Copper Accumulation; Liver Biopsy; Veterinary Case Report.

## 1. Introduction

Liver diseases are common in dogs and may arise from a variety of etiologies, including infectious, metabolic, toxic, and neoplastic conditions. Among these disorders, hepatitis represents an important cause of hepatic dysfunction and may result in significant morbidity and mortality in affected animals [1]. Bacterial hepatitis in dogs may occur through several routes, including ascending infection from the biliary tract, hematogenous dissemination, intestinal bacterial translocation, iatrogenic causes, trauma, or neoplasia [2]. This condition is frequently associated with other hepatobiliary or gastrointes-



**Copyright:** This work is licensed under a Creative Commons Attribution 4.0 International License (CC BY 4.0).

tinal disorders such as cholangitis, gallbladder mucocele, pancreatitis, intestinal dysbiosis, and cholelithiasis. Several bacterial species have been identified in hepatic infections, with *Escherichia coli* and *Enterococcus spp.* among the most isolated pathogens [3].

Copper-associated hepatitis is another recognized cause of chronic liver disease in dogs [4]. Excessive hepatic copper accumulation induces oxidative stress through the production of reactive oxygen species (ROS), leading to hepatocellular injury, inflammation, and progressive hepatic damage [5]. This condition may occur due to genetic defects affecting copper metabolism or secondary to impaired biliary copper excretion, chronic cholestasis, or excessive dietary copper intake [6]. Although copper-associated hepatopathy has been widely reported in predisposed breeds such as Bedlington Terriers, Doberman Pinschers, Labrador Retrievers, and West Highland White Terriers, copper accumulation may also occur in mixed-breed dogs. The coexistence of metabolic and infectious hepatic disorders may complicate diagnosis and therapeutic management in affected patients [7].

Definitive diagnosis of chronic hepatitis requires histopathological examination of liver tissue, microbiological culture with antimicrobial susceptibility testing, and quantification of hepatic copper concentrations. Liver biopsy remains the gold standard diagnostic method and may be obtained through laparoscopic, surgical, or ultrasound-guided techniques, depending on patient condition and available resources [8, 9, 10].

Appropriate therapeutic management depends on accurate etiological identification. Antimicrobial therapy should ideally be guided by bacterial culture and susceptibility testing, particularly considering the increasing prevalence of multidrug-resistant pathogens in hepatobiliary infections [11]. A retrospective study reported that approximately 40% of bacterial isolates from hepatobiliary infections were multidrug-resistant, highlighting the importance of targeted therapy [12]. In cases of copper-associated hepatitis, treatment typically includes lifelong dietary copper restriction and copper-chelating therapy when indicated [5]. Therefore, the objective of this report is to describe a case of bacterial hepatitis caused by *Staphylococcus pseudintermedius* associated with hepatic copper accumulation in a dog, emphasizing the diagnostic approach and clinical management of this complex hepatopathy.

## 2. Case Report

A six-year-old female Dachshund was presented to a private veterinary hospital on August 6, 2025. The dog had been treated at another veterinary clinic for approximately three months due to progressive weight loss, hyporexia, intermittent vomiting, jaundice, and dark yellow urine. During that period, the patient received antibiotic therapy, hepatoprotective medications, and anti-inflammatory drugs, resulting in temporary clinical stabilization. Initial differential diagnoses reportedly included diabetes mellitus, leptospirosis, and acute kidney injury based on clinical and laboratory findings.

Clinical signs later recurred and progressed to hematemesis, requiring hospitalization. Due to the worsening clinical condition, the patient was referred to for further diagnostic investigation and intensive monitoring. At admission, physical examination revealed an alert patient with jaundiced mucous membranes and approximately 7% dehydration. Peripheral lymph nodes were not enlarged, abdominal palpation revealed no abnormalities, and cardiopulmonary auscultation was unremarkable. Rectal temperature was 38.5°C, heart rate was 138 beats per minute, respiratory rate was 40 breaths per minute, and peripheral pulses were strong. Congenital atrophy of the left ocular globe was also observed.

At admission, venous blood gas analysis, complete blood count, serum biochemical profile, and abdominal ultrasonography were performed. Hematological and biochemical findings obtained throughout the diagnostic and treatment period are summarized in Table 1 and Table 2, respectively. Venous blood gas analysis revealed hypokalemia and hyperlactatemia, while mild anemia was detected on the complete blood count. Abdominal ultrasonography was performed at admission and repeated four days later. On the first

examination, the liver appeared mildly reduced in size, with regular contours, heterogeneous echotexture, and decreased echogenicity (Figure 1). The hepatic lymph node was enlarged and hypoechoic, measuring  $2.3 \times 1.5$  cm. The gallbladder showed thickened and regular walls (0.22 cm) and absence of anechoic content, findings suggestive of cholestasis. A small amount of free abdominal fluid was also observed but was not drainable at the time of examination.

**Table 1:** Comparative hematological parameters obtained during different stages of treatment in a dog diagnosed with bacterial hepatitis and hepatic copper accumulation.

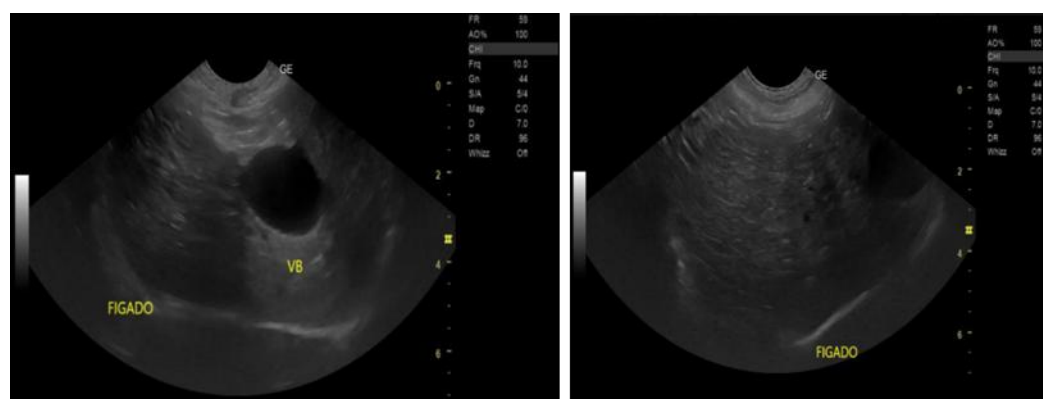
Parameter	Admission	10 days post-biopsy	30 days post-biopsy	90 days post-biopsy	Reference range	Unit
RBC	5.09	6.59	7.37	8.05	5.5–8.5	milhões/mm <sup>3</sup>
Hemoglobin	14	17.20	20	22.70	12–18	g/dL
Hematocrit	36	46	51	56	37–55	%
MCV	70.73	69.80	69.20	69.57	60–77	u <sup>3</sup>
MCHC	38.89	37.39	39.22	40.45	30–36	g/dL

**Table 2:** Serum biochemical parameters evaluated throughout the diagnostic investigation and treatment period.

Parameter	Admission	10 days post-biopsy <sup>1</sup>	30 days post-biopsy <sup>2</sup>	90 days post-biopsy <sup>2</sup>	Reference range <sup>1</sup>	Reference range <sup>2</sup>	Unit
ALT	303	721.2	421.3	422.3	7–92	10–100	UI/L
AST	105	181.2	105.8	90.6	10–88	0–50	UI/L
ALP	1299	5378	2340	294	10–156	23–212	UI/L
Albumin	3	3.54	3.97	3.83	2,3–3,8	2,3–3,8	g/dL
Urea	17	52.8	28	39.9	10–60	7–54	mg/dL
Glucose	99	82.29	98.29	81.4	60–118	70–143	g/dL

Note: Biochemical analyses were performed in different laboratories. A marked increase in ALP activity was observed 10 days after biopsy. This elevation may be attributed to a combination of factors, including postoperative cholestasis, disease progression, and possible induction associated with corticosteroid administration. Laboratory<sup>1</sup>, did not provide bibliographic sources for reference values, whereas Laboratory<sup>2</sup> used the reference intervals Weiss, DJ; Wardrop, Schalm's Veterinary Hematology. Iowa. 6ed, 2010, 1206p. Thrall, MA et al. Gradual changes in biochemical parameters were observed over the course of treatment.

**Figure 1:** Abdominal ultrasonography of the liver in a dog with hepatitis. Ultrasonographic image demonstrates diffuse hepatic parenchymal heterogeneity with increased echogenicity and irregular echotexture. The hepatic margins appear slightly rounded, and mild biliary sludge is observed within the gallbladder lumen, suggesting hepatobiliary involvement and cholestasis.

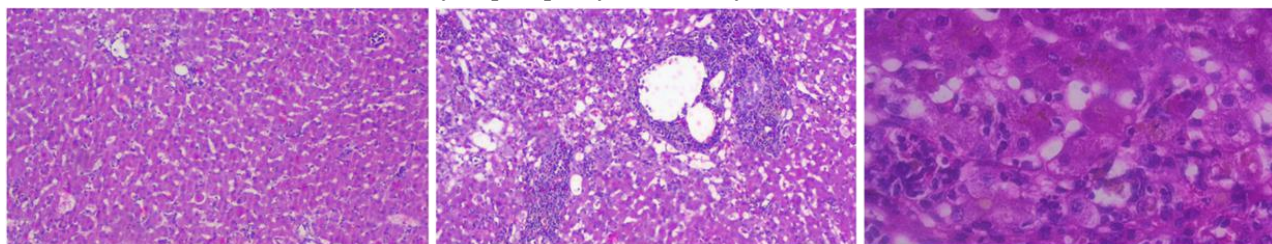


On the follow-up examination performed four days later, the liver remained reduced in size and showed irregular and coarse contours with mixed echogenicity, indicating a heterogeneous parenchymal pattern. The gallbladder and hepatic lymph node maintained similar abnormalities. Based on these findings, hepatic biopsy was recommended for histopathological evaluation, bacterial culture with antimicrobial susceptibility testing, fungal culture, and hepatic copper quantification. Prior to surgery, electrocardiography, echocardiography, and coagulation testing (PT and aPTT) were performed. The only abnormality detected was a mild reduction in aPTT.

An exploratory laparotomy was performed to obtain hepatic biopsy samples. Two tissue samples were fixed in formalin for histopathological analysis, and an additional sample was preserved in sterile saline for bacterial culture, antimicrobial susceptibility testing, fungal culture, and hepatic copper quantification. Postoperative therapy included dipyrone (25 mg/kg every 8 hours for 5 days), tramadol hydrochloride (2.5 mg/kg every 8 hours for 3 days), amoxicillin–clavulanate (20 mg/kg every 12 hours for 5 days), and prednisolone (1 mg/kg every 24 hours for 5 days). Following surgery, the patient showed progressive clinical improvement, including weight gain, improved appetite, and normalization of mucous membrane color. No postoperative complications or clinical relapses were observed.

Histopathological evaluation of the liver biopsy revealed moderate diffuse hepatocellular degeneration associated with moderate subacute hepatitis, bile duct hyperplasia, and cholestasis. Microscopically, hepatocytes showed a cloudy cytoplasmic appearance, occasionally with large non-staining vacuoles. A moderate inflammatory infiltrate composed of neutrophils and lymphocytes was observed, predominantly distributed in the periportal region. Additionally, brownish pigment compatible with bilirubin was detected within hyperplastic bile ducts and within the cytoplasm of hepatocytes (Figure 2).

**Figure 2:** Histopathological findings in liver biopsy of a dog with hepatitis. Photomicrograph of hepatic tissue showing inflammatory infiltrate within the hepatic parenchyma associated with hepatocellular degeneration and areas of hepatocyte necrosis. Mixed inflammatory cells are distributed within the portal areas and hepatic lobules, consistent with inflammatory hepatopathy. Hematoxylin and eosin stain.



Bacterial culture of the hepatic tissue yielded growth of *Staphylococcus pseudintermedius*, a facultative anaerobic bacterium commonly associated with opportunistic infections in dogs. Antimicrobial susceptibility testing demonstrated sensitivity to multiple antibiotics, including amoxicillin–clavulanate, doxycycline, enrofloxacin, marbofloxacin, and cefovecin. Fungal culture of the liver sample showed no growth of pathogenic fungi, ruling out fungal hepatic infection. After bacterial culture and antimicrobial susceptibility results were obtained, antimicrobial therapy was extended to a total duration of 30 days after surgery.

Hepatic copper quantification was performed using Atomic Absorption Spectroscopy (ICP-OES), and results were expressed on a dry weight basis, with a measured concentration of 1057 ppm (reference range: 120–500 ppm). Hepatic copper quantification results became available approximately 45 days after the procedure and revealed a concentration of 1057 ppm (reference range: 120–500 ppm). Based on these findings, a commercial low-copper diet (Royal Canin Hepatic®) was initiated and maintained. In addition, S-adenosylmethionine (SAMe) was prescribed at a dosage of 20 mg/kg once daily for 20

days. During the 10-month follow-up period, the patient remained clinically stable, with no recurrence of clinical signs. Periodic biochemical monitoring demonstrated progressive improvement and stabilization of liver enzyme activities, supporting a favorable long-term outcome.

### 3. Discussion

Hepatic disorders are frequently encountered in canine clinical practice and may arise from a wide range of etiologies, including metabolic, congenital, toxic, infectious, and neoplastic conditions [1]. Given this heterogeneity, accurate identification of the underlying cause is essential to guide appropriate therapeutic strategies and improve clinical outcomes. Clinical manifestations of liver disease in dogs are typically nonspecific and may overlap among different etiologies. Common clinical signs include weight loss, hyporexia, jaundice, ascites, vomiting, diarrhea, hematochezia, melena, polyuria, and polydipsia [7]. None of these signs are pathognomonic, reinforcing the need for a systematic diagnostic approach.

In the present case, the patient exhibited progressive clinical signs over approximately three months, including weight loss, anorexia, diarrhea, mild ascites, and jaundice, without progression to severe complications such as hepatic encephalopathy. These findings are consistent with previous reports, in which decreased appetite (61%), lethargy (56%), icterus (34%), and ascites (32%) are among the most frequently observed clinical signs in dogs with hepatopathy [13].

Biochemical evaluation revealed marked elevations in liver enzymes, particularly alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), indicating hepatocellular injury and cholestatic involvement. Notably, a marked increase in ALP activity was observed 10 days after biopsy, which may be attributed to a combination of factors, including postoperative cholestasis, progression of hepatobiliary disease, and potential induction associated with corticosteroid administration. Despite these alterations, blood glucose, urea, and albumin concentrations remained within reference intervals, suggesting preserved hepatic synthetic function at the time of evaluation. Mild anemia and a shortened activated partial thromboplastin time (aPTT) were also observed, which may indicate early coagulation disturbances associated with hepatic dysfunction.

Definitive diagnosis of hepatitis requires integration of histopathological evaluation, microbiological culture with antimicrobial susceptibility testing, and hepatic copper quantification. Liver biopsy remains the gold standard diagnostic method and may be obtained via laparotomy, laparoscopy, or ultrasound-guided techniques [7]. In the present case, biopsy was performed via exploratory laparotomy due to the unavailability of laparoscopy and concerns regarding hemorrhagic risk and limited sampling associated with ultrasound-guided techniques. Although laparotomy is more invasive, no intraoperative or postoperative complications were observed.

To minimize the risk of sample contamination during surgical biopsy, strict aseptic technique was employed, and hepatic tissue samples were collected using sterile instruments with minimal manipulation. The sample for microbiological culture was obtained directly from the hepatic parenchyma immediately after abdominal entry, avoiding contact with the skin or surrounding tissues. An additional methodological aspect to consider is the sampling strategy adopted during liver biopsy. In the present case, multiple samples were collected from different hepatic lobes based on macroscopic intraoperative assessment, prioritizing areas with the most evident gross alterations.

This approach likely increased the diagnostic yield, as hepatic lesions may be heterogeneously distributed, particularly in inflammatory and cholestatic conditions. However, targeted sampling of visually affected areas may also introduce a degree of selection bias, potentially overrepresenting more severely affected regions. Despite this limitation, combining samples from different lobes improves overall diagnostic representativeness and is consistent with current recommendations for hepatic biopsy in dogs.

Bacterial culture identified *Staphylococcus pseudintermedius* as the causative agent. Although this organism is commonly considered a commensal of canine skin, it is also recognized as an opportunistic pathogen capable of causing systemic infections. Schlachet et al. [12] reported that *Staphylococcus spp.* account for approximately 12% of bacterial hepatobiliary infections, with a subset demonstrating multidrug resistance. In the present case, the isolate was susceptible to multiple antimicrobial classes, supporting the use of targeted therapy. Furthermore, the concordance between microbiological findings, histopathological evidence of inflammation, and favorable clinical response to antimicrobial therapy supports the clinical relevance of the isolated organism rather than contamination.

The selection of antimicrobial therapy was based on reported resistance patterns, with lower resistance rates observed for aminopenicillins, amoxicillin–clavulanate, and cephalosporins (0–15%) compared with tetracyclines, fluoroquinolones, and trimethoprim–sulfamethoxazole, which demonstrate resistance rates exceeding 20% [12]. The optimal duration of antimicrobial therapy for bacterial hepatitis remains poorly defined, and repeated liver sampling is impractical due to procedural risks. Therefore, clinical improvement remains a key parameter for guiding treatment duration. In this case, antimicrobial therapy was extended to 30 days, resulting in sustained clinical recovery.

An additional relevant finding was the marked hepatic copper accumulation, with a concentration of 1057 ppm, exceeding both reference limits and the threshold recommended by the WSAVA for diagnosis of copper-associated hepatopathy (>1,000 µg/g dry weight). Histopathological evaluation revealed moderate diffuse hepatocellular degeneration, moderate subacute hepatitis, bile duct hyperplasia, and cholestasis. However, the histopathological report did not include a detailed description of the distribution pattern of copper within the hepatic lobule, which represents an important limitation. The assessment of copper distribution (centrilobular versus periportal) is critical to differentiate primary metabolic disorders from secondary accumulation associated with cholestasis.

Despite this limitation, the presence of cholestasis and bile duct hyperplasia supports the hypothesis of secondary copper accumulation due to impaired biliary excretion. Previous studies have demonstrated that cholestatic conditions may lead to progressive copper retention within hepatocytes [18, 19]. Conversely, copper accumulation itself may contribute to hepatocellular injury through oxidative stress, creating a complex interplay between metabolic and inflammatory processes. The absence of specific histochemical staining, such as Rhodanine or Rubeanic acid staining, further limits the ability to visually confirm copper deposition and distribution, although quantitative analysis provided objective evidence of accumulation.

It remains unclear whether bacterial hepatitis or copper accumulation represented the primary insult in this case. Secondary bacterial infection in dogs with copper-associated hepatopathy has been reported, although with relatively low prevalence (0–15%) [11]. In the present case, the delayed availability of copper quantification results led to initiation of a low-copper diet only after significant clinical improvement had already occurred following antimicrobial therapy. This temporal association suggests that bacterial hepatitis was likely the primary condition, with secondary copper accumulation resulting from cholestasis.

The delay in pursuing definitive diagnostic investigation prior to referral represents an important limitation in the clinical management of this case. The patient received empirical treatment for approximately three months, resulting in temporary stabilization but delaying etiological diagnosis. This highlights the importance of early liver biopsy in dogs presenting with progressive hepatobiliary disease and persistent clinical signs, as early diagnosis may improve therapeutic outcomes. The primary source of *S. pseudintermedius* infection was not definitively identified. Although no history of dermatological or otic disease was reported, hematogenous dissemination from a subclinical source cannot be excluded. Opportunistic infections caused by this organism may occur in the presence of underlying systemic or immunological alterations.

Therapeutic management consisted of targeted antimicrobial therapy, dietary copper restriction, and hepatoprotective supplementation [5]. Although hepatic copper levels exceeded the threshold at which chelation therapy is typically recommended, D-penicillamine was not administered due to its limited availability and challenges associated with long-term use. Zinc supplementation was also not implemented, despite its potential benefits in reducing intestinal copper absorption. Given the favorable clinical response following antimicrobial therapy, bacterial hepatitis was considered the primary driver of disease, and dietary management was prioritized. Prednisolone was administered post-operatively for a short duration at an anti-inflammatory dose. Although the use of corticosteroids in the presence of bacterial infection remains controversial, the limited duration of therapy combined with concurrent antimicrobial treatment likely minimized the risk of immunosuppression. No clinical deterioration attributable to corticosteroid use was observed.

Prognosis in canine hepatopathies varies widely depending on the underlying etiology, disease severity, and response to treatment. Bacterial hepatitis generally carries a guard to good prognosis, depending on antimicrobial susceptibility and timeliness of intervention. In contrast, copper-associated hepatopathy demonstrates variable outcomes, with reported survival times ranging from 22.5 to 561 days [7]. Negative prognostic indicators include hyperbilirubinemia, coagulation abnormalities, hypoalbuminemia, ascites, and hepatic fibrosis, whereas elevations in liver enzymes alone are not associated with poorer outcomes.

During the 10-month follow-up period, the patient remained clinically stable, with no recurrence of clinical signs and progressive improvement in laboratory parameters, supporting a favorable long-term outcome. Overall, this case highlights the importance of comprehensive diagnostic evaluation in canine hepatopathies and underscores the potential coexistence of infectious and metabolic factors in liver disease.

#### 4. Conclusion

This report describes a case of bacterial hepatitis caused by *Staphylococcus pseudintermedius* associated with hepatic copper accumulation in a dog, highlighting the complexity of diagnosing multifactorial hepatopathies. The integration of clinical findings, imaging, histopathology, microbiological culture, and hepatic copper quantification was essential for establishing the diagnosis and guiding targeted therapy. The findings support the hypothesis that bacterial hepatitis was the primary condition, with secondary copper accumulation likely associated with cholestasis. However, the absence of detailed copper distribution analysis and specific histochemical staining represents a limitation in definitively distinguishing primary from secondary copper-associated hepatopathy.

This case underscores the importance of early and comprehensive diagnostic investigations, including liver biopsy and microbiological analysis, particularly in patients with progressive or unresponsive hepatobiliary diseases. In addition, it highlights the value of aseptic sampling techniques and targets antimicrobial therapy in improving clinical outcomes. Recognition of the potential coexistence of infectious and metabolic factors is essential for accurate diagnosis and appropriate therapeutic decision-making in canine hepatopathies.

**Funding:** None.

**Research Ethics Committee Approval:** None.

**Acknowledgments:** The authors thank the veterinary diagnostic laboratory for performing the microbiological and histopathological analyses.

**Conflicts of Interest:** All other authors declare no conflicts of interest.

## References

1. Stockman J. Nutritional management of hepatobiliary diseases in dogs and cats. *Vet Clin North Am Small Anim Pract.* 2025;55(4):579–593. doi:10.1016/j.cvsm.2025.03.007.
2. Maté de Haro L, Vila A, di Bella A, Mallol C, Anselmi C, Barreiro-Vazquez JD, et al. Computed tomographic findings in dogs with hepatic bacterial parenchymal infection and abscessation. *Animals (Basel).* 2024;14(23). doi:10.3390/ani14233399.
3. Phumthanakorn N, Potivanakul S, Kitjarak S, Lopnapun T, Moonkaew N, Changtrakul T, et al. Characteristics of gallbladder microbiome in healthy dogs and cats, dogs with gallbladder mucocele, and cats with suspected cholangitis/cholangiohepatitis. *Can J Vet Res.* 2024;88:77–86.
4. Mutton J, Yeomans S, White J. Copper hepatopathies in Australian dogs. *Aust Vet J.* 2024;102(8):385–391. doi:10.1111/avj.13338.
5. den Boer ER, Fieten H, Aicher KM. Copper-associated chronic hepatitis in dogs. *Vet Clin North Am Small Anim Pract.* 2025;55(4):e25–e54. doi:10.1016/j.cvsm.2025.08.010.
6. Larose PC, Brisson BA, Foster RA, Monteith G. Comparing 3 mm and 5 mm laparoscopic liver biopsy samples in dogs. *Vet Surg.* 2024;53(4):742–753. doi:10.1111/vsu.14006.
7. Webster CRL, Center SA, Cullen JM, Penninck DG, Richter KP, Twedt DC, et al. ACVIM consensus statement on the diagnosis and treatment of chronic hepatitis in dogs. *J Vet Intern Med.* 2019;33(3):1173–1200. doi:10.1111/jvim.15467.
8. Pavlick M, Webster CRL, Penninck DP. Bleeding risk assessment for percutaneous ultrasound guided hepatic biopsy in dogs and cats. In: *ACVIM Forum; 2017 Jun 8–10; National Harbor (MD).*
9. Cavasin JP. Hepatic histopathology: what a clinician should know. *Vet Clin North Am Small Anim Pract.* 2025;55(4):e1–e11. doi:10.1016/j.cvsm.2025.07.006.
10. Twedt D, Cullen J, Rothuizen J, Desmet V, Bunch S, Van Winkle T, et al. *WSAVA liver standardization group.* Amsterdam: Elsevier; 2006.
11. Wagner KA, Hartmann FA, Trepanier LA. Bacterial culture results from liver, gallbladder, or bile in 248 dogs and cats evaluated for hepatobiliary disease: 1998–2003. *J Vet Intern Med.* 2007;21(3):417–424. doi:10.1111/j.1939-1676.2007.tb02984.x.
12. Schlachet AT, Boulouis HJ, Beurlet-Lafarge S, Canonne MA. Antimicrobial susceptibility patterns of bacteria associated with hepatobiliary disease in dogs and cats (2010–2019). *J Vet Intern Med.* 2025;39(2). doi:10.1111/jvim.70007.
13. Lidbury JA. Complications of liver disease. *Vet Clin North Am Small Anim Pract.* 2025;55(4):559–577. doi:10.1016/j.cvsm.2025.03.008.
14. Lee GY, Yang SJ. Comparative assessment of genotypic and phenotypic correlates of *Staphylococcus pseudintermedius* strains isolated from dogs with otitis externa and healthy dogs. *Comp Immunol Microbiol Infect Dis.* 2020;70. doi:10.1016/j.cimid.2019.101376.
15. Parasana DK, Kalyani IH, Kachchhi AV, Koringa PG, Makwana PM, Patel DR, et al. Profiling of antimicrobial resistance genes from *Staphylococcus pseudintermedius* isolated from dogs with pyoderma using whole genome sequencing. *Comp Immunol Microbiol Infect Dis.* 2025;116. doi:10.1016/j.cimid.2024.102288.
16. Soimala T, Lübke-Becker A, Hanke D, Eichhorn I, Fessler AT, Schwarz S, et al. Molecular and phenotypic characterization of methicillin-resistant *Staphylococcus pseudintermedius* from ocular surfaces of dogs and cats suffering from ophthalmological diseases. *Vet Microbiol.* 2020;244. doi:10.1016/j.vetmic.2020.108687.
17. van Duijkeren E, Kamphuis M, van der Mije IC, Laarhoven LM, Duim B, Wagenaar JA, et al. Transmission of methicillin-resistant *Staphylococcus pseudintermedius* between infected dogs and cats and contact pets, humans and the environment in households and veterinary clinics. *Vet Microbiol.* 2011;150(3–4):338–343. doi:10.1016/j.vetmic.2011.02.012.
18. Lawrence YA, Ruauz CG, Nemanic S, Milovancev M. Characterization, treatment, and outcome of bacterial cholecystitis and bactibilia in dogs. *J Am Vet Med Assoc.* 2015;246(9):982–989.
19. Tamborini A, Jahns H, McAllister H, Kent A, Harris B, Procoli F, et al. Bacterial cholangitis, cholecystitis, or both in dogs. *J Vet Intern Med.* 2016;30(4):1046–1055. doi:10.1111/jvim.13974.