RESEARCH ARTICLE

Concurrent Gallbladder Mucocele and Chronic Hepatitis In A Female Siberian Husky: A Case Report

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Abstract

A five-year-old, 23-kg, female Siberian Husky was presented to Clínica Vet D´Pelos, Texcoco, State of Mexico, Mexico, with acute jaundice, persistent vomiting, and loss of appetite. Gallbladder mucocele and chronic hepatitis are important hepatobiliary disorders in dogs. Their concurrent presentation may complicate clinical interpretation and diagnosis. Physical examination revealed jaundiced mucous membranes and general weakness without abdominal pain. Haematological analysis indicated severe leukocytosis with neutrophilia and a left shift, which was consistent with a systemic inflammatory response. Biochemical profiling revealed conjugated hyperbilirubinemia and marked elevation of hepatic enzyme activities, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), gamma-glutamyl transferase (GGT), and alkaline phosphatase (ALP), as well as hypoalbuminemia, hypocholesterolemia, and electrolyte disturbances, indicative of obstructive cholestasis and hepatocellular insufficiency. Urinalysis demonstrated bilirubinuria and abundant bile pigment. Ultrasound imaging revealed diffuse liver disease, with no evidence of biliary obstruction. Immunological testing by microagglutination for the detection of anti-Leptospira antibodies yielded negative results. Histopathology results confirmed the presence of a mature vesicular mucocele with cystic hyperplasia of the epithelium, mucin accumulation, and obliteration of the vesicular lumen. Chronic hepatitis with a portal inflammatory infiltrate, biliary proliferation and bile pigment accumulation was observed in the liver. These findings suggested a mixed hepatobiliary disease process, in which the gallbladder mucocele acted as the primary obstructive factor and trigger for secondary liver inflammation.

Key words: Gallbladder disease, Hepatobiliary disorder, Hyperbilirubinemia, Histopathology.

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Introduction

Canine gallbladder mucocele (GBM) is characterized by the progressive accumulation of a dense mass of bile and mucin within the gallbladder. This mass can vary in color from pale yellow to dark green (Aiello and Moses, 2016). This gelatinous material may extend into the cystic, hepatic, and common bile duct, resulting in varying degrees of biliary obstruction, cholestasis and jaundice. Progressive distension of the gallbladder may compromises the vascularization of the gallbladder wall, predisposing it to ischemia, necrosis, cholecystitis, and, in severe cases, gallbladder rupture with biliary peritonitis (Mehler and Mayhew, 2023).

Multiple predisposing factors for the formation of vesicular mucoceles have been identified. Gallbladder mucocele usually occurs in middle-aged to older dogs, with no gender predilection (Mehler and Mayhew, 2023; Poblanno Silva *et al*., 2023). Breeds such as the Shetland Sheepdog, Miniature Schnauzer and Cocker Spaniel are at greater risk, and a genetic mutation (ABCB4, also called MDR3) associated with the development of mucoceles has been described in these breeds (Bexfield *et al*., 2011; Galley *et al*., 2022). Notable comorbidities include endocrinopathies (typical or atypical hyperadrenocorticism), hypothyroidism, diabetes mellitus, and dyslipidaemias (hypertriglyceridaemia or hypercholesterolaemia) due to idiopathic causes, high-fat diets, nephrotic syndrome, or pancreatitis (Dini *et al*., 2024). Deusdado *et al*. (2023) evaluated 30 dogs diagnosed with biliary mucocele, of which two-thirds (66.6%) had endocrinopathies could explain the occurrence of gallbladder mucocele.

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From a pathophysiological perspective, the formation of mucoceles is associated with alterations in the composition of bile mucins. In healthy canines and those afflicted with mucocele, biliary mucus is predominantly constituted of gel-forming mucins, namely MUC5B and MUC5AC (Nagao *et al*., 2023). In canines afflicted with mucocele, the vesicular content has been found to contain MUC5AC at levels 17 times higher than in healthy canines with no significant change in MUC5B (Uusaed and Aldridge, 2022). This imbalance has been shown to increase the viscosity of bile, causing the mucin-rich gel to adhere firmly to the epithelial surface of the gallbladder. Prolonged bile stasis, in combination with fluid absorption (dehydration of the contents) by the gallbladder wall, has been demonstrated to promote the formation of a thick viscoelastic matrix (Gookin *et al*., 2024). This material becomes intimately integrated into the folds of the gallbladder mucosa, creating a substrate that hinders or prevents normal emptying of the gallbladder. The resultant is the progressive accumulation of mucous bile and biliary sludge that progresses to mucocele and, when dehydrated, transforms into a semi-solid, rubbery mass that obstructs the lumen (Itoh *et al*., 2022). On ultrasonographic examination, the characteristic pattern of a mature vesicular mucocele (kiwi-like appearance) is highly recognizable, and early diagnosis followed by surgical intervention (cholecystectomy) improves the prognosis for dogs with this condition (Parkanzky *et al*., 2019). Conversely, prolonged biliary obstruction can lead to chronic liver damage and critical acute events such as severe jaundice, gallbladder rupture, or acute liver failure (Deusdado *et al*., 2023). The present study describe a five- years-old, 23-kg, female Husky dog with mature vesicular mucocele, obstructive cholestasis, and acute icterus. The clinicopathological, ultrasonographic, immunological, and histopathological findings are described.

Case Presentation

A five-year-old, splayed female Siberian Husky named Maki, weighing 23 kg, was presented to Clínica Vet D´Pelos, Texcoco, State of Mexico, Mexico, due to persistent vomiting, anorexia and mucocutaneous jaundice. A thorough clinical examination was conducted, which revealed signs of lethargy, intensely jaundiced mucous membranes, a normal body temperature, and the absence of any discernible abdominal pain. No masses or significant abdominal distension were detected. The remaining vital signs were within normal references ranges.

A diagnosis of hepatobiliary cholestasis was suspected, therefore, blood and urine samples were collected for complete blood count, serum biochemistry, urinalysis, and immunodiagnostic tests (for prevalent infectious diseases such as *Ehrlichia* spp., *Anaplasma* spp., and *Leishmania* spp.). Imaging evaluations were performed, including abdominal ultrasography and dorsoventral chest radiographs, to assess biliary obstructions, structural liver changes, or evidence of metastatic disease.

Blood count analysis

The results of the blood count analysis indicated leukocytosis with neutrophilia, the presence of band neutrophils, monocytosis and a left shift. Slight thrombocytopenia was also observed (Table 1).

Table 1: Blood count of a five-year-old female dog with jaundice and suspected liver damage

Parameters	Result (Reference value)	Observations
Haematocrit, L L^{-1}	0.49 (0.37-0.55)	***
Haemoglobin, g L^{-1}	170 (120-180)	***
Erythrocytes, Cell x 10^{12} L^{-1}	6.8 (5.5-8.5)	***
Mean Corpuscular Volume, ƒL	73 (60-77)	***
Mean Corpuscular Haemoglobin Concentration, g L^{-1}	345 (300-360)	***
Reticulocytes, Cell x 10^9 L^{-1}	*** (<60)	***
Platelets, Cell x 10^9 L^{-1}	185 (200-900)	Low
Total Solids, g L^{-1}	64 (60-75)	***
Leukocytes, Cell x 10^9 L^{-1}	64.5 (6.0-17.0)	High
Segmented neutrophils, Cell x 10^9 L^{-1}	55.5 (3.0-11.5)	High
Band neutrophils, Cell x 10^9 L^{-1}	4.5 (0-0.3)	High
Eosinophils, Cell x 10^9 L^{-1}	0 (0-0.9)	***
Basophils. Cell x 10^9 L^{-1}	0 (Rare)	***
Lymphocytes, Cell x 10^9 L^{-1}	1.9 (1.0-4.8)	***
Monocytes, Cell x 10^9 L^{-1}	2.6 (0.1-1.4)	High
Metamyelocytes, Cell x 10^9 L^{-1}	0 (0)	***
Myelocytes, Cell x 10^9 L^{-1}	0 (0)	***
Cell morphology abnormalities:		
Erythrocytic: Negative	Leukocytic: Negative	

***: Values within the reference range, without significant clinical alteration

Serum biochemistry

Serum biochemistry results revealed mild hyperazotaemia, metabolic acidosis, and electrolyte disturbances including hyponatraemia, hypochloremia, and hypokalaemia. Severe hyperbilirubinaemia was observed, with a predominance of the conjugated fraction. In addition, marked increases in alanine aminotransferase, aspartate aminotransferase,

and alkaline phosphatase activities were detected. Hypoalbuminaemia and hypocholesterolaemia were also recorded (Table 2).

Urine test

Urinalysis revealed intense bilirubinuria and abundant bile pigment (Table 3). Mild bacteriuria, characterized by a moderate number of cocci, and leukocytes were also identified. Fine granular casts were observed.

Table 2: Biochemical profile of a five-year-old female dog with jaundice and suspected liver damage

Parameters	Result (Reference value)	Observations
Glucose, mmol L^{-1}	3.66 (3.38-6.88)	***
Urea, mmol L^{-1}	23 (2.1-7.9)	High
Creatinine, µmol L^{-1}	129 (60-126)	High
Cholesterol, mmol L^{-1}	3.5 (2.85-7.76)	***
Triglycerides, mmol L^{-1}	0.8 (0.6 – 1.2)	***
Total bilirubin, µmol L^{-1}	268 (< 5.2)	High
Conjugated bilirubin, µmol L^{-1}	208 (< 3.2)	High
Unconjugated bilirubin, µmol L^{-1}	60 (< 2.0)	High
ALT, UI L^{-1}	485 (< 70)	High
AST, UI L^{-1}	286 (< 55)	High
Alkaline phosphatase, UI L^{-1}	632 (< 189)	High
CK, UI L^{-1}	104 (< 213)	***
Total proteins, g L^{-1}	56 (56-75)	***
Albumin, g L^{-1}	24 (29-40)	Low
Globulins, g L^{-1}	32 (24-39)	***
Ionised calcium, mmol L^{-1}	1.12 (1.11-1.37)	***
Inorganic phosphorus, mmol L^{-1}	2.96 (0.75-1.70)	High
Sodium, mmol L^{-1}	133 (141-153)	Low
Chloride, mmol L^{-1}	93 (108-118)	Low
Potassium, mmol L^{-1}	3.23 (3.82-5.34)	Low
Bicarbonate, mmol L^{-1}	16 (17-25)	Low
DIF, mmol L^{-1}	40 (28-40)	***
Anion gap, mmol L^{-1}	27 (12-24)	High
Osmolality, mOsm K^{-1}	293 (280-305)	***
Amylase, UI L^{-1}	540 (<1100)	***
Albumin/Globulins = 0.75 Creatinine/Urea = 5.61 BC / BNC = 3.47 Cai / P = 0.38		

Observations: Icteric serum 3+. ***: Values within the reference range, without significant clinical alteration, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase, CK: Creatine kinase, DIF: Strong ionic difference, Osmolality: Total concentration of solutes per kg of solvent, BC/BNC: Conjugated Bilirubin/Unconjugated Bilirubin Ratio, Cai/P: Ionic Calcium/Phosphorus Ratio.

Table 3: Urinalysis of a five-year-old female dog with jaundice and suspected liver damage

Parameters	Parameters	Result	Reference range / interpretation
Microscopic examination	Erythrocytes (cells/40×)	0	< 5/field
	Leukocytes (cells/40×)	0-3	< 5/field
	Transitional epithelial cells (cells/40×)	0-2	Occasional cells may be observed
	Squamous epithelial cells (cells/40×)	0-3	Occasional cells may be observed
	Casts (number/40×)	0-1 (fine granular)	A few hyaline or granular casts may be normal
	Crystals (number/40×)	0	None to occasional crystals may be observed
	Bacteria (cells/40×)	Moderate number of cocci	Negative
	Others, ***	Abundant bilirubin pigment	Negative
Chemical examination	pH	6.5	Typically acidic in dogs
	Leukocytes	Traces	Dipstick leukocyte test not valid in animals
	Nitrites	Negative	Dipstick nitrite test not valid in animals
	Proteins	Negative	Negative
	Glucose	Normal	Negative
	Ketone bodies	Negative	Negative
	Urobilinogen	Normal	Small amount may be normal
	Bilirubin	3+(70 µmol/L)	Negative to trace; small amounts may be normal only in concentrated canine urine
Physical examination	Erythrocytes	Negative	Negative
	Appearance	Cloudy 1+	N/A
	Colour	Deep yellow	Yellow to amber
	Density	1.014	1.016–1.060
	Odour	Typical	Characteristic / typical

Urine sample was collected by puncture. All urinary sediment elements were evaluated at 40× microscopic magnification. Reference values and interpretive criteria were based on the MSD/Merck Veterinary Manual (Whitbread, 2025). Leukocyte esterase and nitrite dipstick tests are not considered valid in animals.

Imaging and immunodiagnostic tests

Chest X-rays revealed no obvious cardiopulmonary abnormalities in this case (Figure 1). Liver ultrasonography

indicated diffuse abnormalities in hepatic echogenicity consistent with liver disease (Figure 2). No evidence of biliary obstruction or liver masses was observed. Immunological testing testing by microagglutination for the detection of anti-Leptospira antibodies yielded negative results.

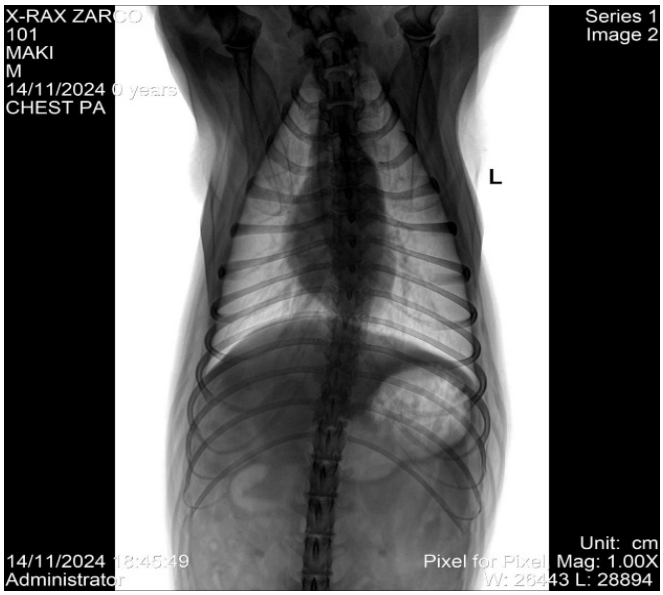


Fig. 1: Dorsoventral thoracic radiograph of a five-year-old female Siberian Husky dog. No cardiopulmonary abnormalities or radiographic signs of thoracic metastasis were observed. The cardiac silhouette and bronchopulmonary pattern were consistent with the breed and age of the dog. Mild hepatomegaly was observed, with caudal displacement of the diaphragm, consistent with increased liver size associated with hepatobiliary disease.

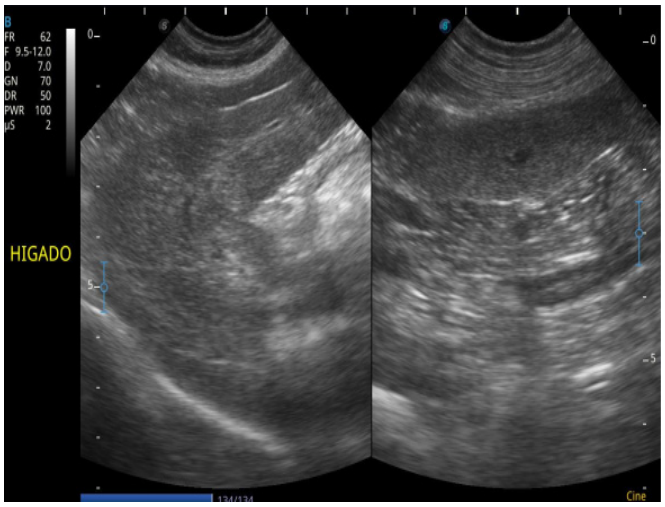


Fig. 2: Abdominal ultrasonographic image of the liver in a five-year-old female Siberian Husky dog. Increased hepatic echogenicity was observed, consistent with diffuse hepatic alteration.

Histopathology

For histopathological analysis, the gallbladder sample measured between 1.4 and 2.6 × 1.4 × 5.0 cm, with fluctuating consistency, a smooth external surface, and an opaque white color. Upon sectioning, the mucosa was markedly distended by dark green gelatinous material that completely blocked the vesicular lumen. The hepatic lobe measured 9.2 × 6.0 × 2.0 cm and exhibited a firm consistency, with a smooth external surface of a greenish-brown hue, alternating between slight elevations and depressions. Upon sectioning, the internal surface exhibited a consistent colorimetric and morphological pattern.

Microscopic examination of the gallbladder revealed hyperplasia of the mucosal epithelium, with the formation of prominent papillary structures. Marked dilation of the subepithelial glands was observed, which contained acellular, homogeneous, and slightly eosinophilic material. This same material was found in the vesicular lumen. In the liver, inflammatory infiltrates were observed in multiple portal spaces. The infiltrate was composed of lymphocytes, plasma cells, and reactive fibroblasts. In addition, neovascularization and biliary proliferation were observed. Several bile ducts were occluded by eosinophilic proteinaceous material similar to that identified in the gallbladder. In the hepatic parenchyma, numerous hepatocytes and bile canaliculi contain abundant bile pigments.

Treatment and oucome

Initial medical treatment included omeprazole (20 mg, every 24 h), administered as a gastroprotective agent to reduce gastric acid secretion and minimize gastrointestinal irritation associated with persistent vomiting and systemic illness. Ursodeoxycholic acid (Ursofalk®; 250 mg, every 12 h) was prescribed to promote bile flow and support hepatobiliary function in the context of cholestasis. Furosemide (Furasen®; 50 mg, every 12 h) was administered as a diuretic as part of supportive management. Panclasa® (80/80 mg, every 24 h) was also included in the treatment regimen as supportive therapy. Despite medical management and the diagnostic work-up performed, the clinical condition of the dog progressively deteriorated, and the patient died.

Ethical approval

Ethical approval was not required for this case report. Informed consent was obtained from the owner for the clinical evaluation, diagnostic procedures, sample collection, and use of anonymized clinical and pathological data for scientific publication. Due to financial constraints, euthanasia had initially been considered; however, the owner agreed to allow the diagnostic work-up and sample collection at no cost. All procedures and patient handling were performed according to accepted veterinary clinical practice described by Birchard and Sherding (2006) and in accordance with the applicable Mexican regulations for the care and use of animals (NOM-062-ZOO-1999).

Discussion

The clinicopathological findings in the present case report the concurrent presence of gallbladder mucocele (GBM), cholestasis, and severe hepatobiliary injury. Histopathological examination revealed portal inflammatory infiltrates, biliary proliferation, and bile pigment accumulation in the liver, together with the characteristic lesions of a mature gallbladder mucocele. These findings are partially consistent with those reported by Jablonski *et al*. (2024), who described biliary hyperplasia, fibrosis, and portal inflammation as common histological alterations in dogs with advance of concurrent hepatobiliary disease rather than as proof of a direct causal sequence between GBM and chronic hepatitis.

Clinically, the presentation with mucocutaneous jaundice and nonspecific gastrointestinal signs, including vomiting and anorexia, is consistent with biliary mucocele, although these signs are not exclusive to this condition (McKenna *et al*., 2021). Similary Dini *et al*. (2024) reported that many dogs with mucoceles may show vague clinical signs until advanced stages. In agreement with Itoh *et al*. (2022), jaundice is a common finding in advanced disease. In contrast, the dog described here did not show marked abdominal pain, suggests that clinical presentation may vary among cases (Kesimer *et al*., 2015). The marked jaundice and inflammatory leukogram observed in this dog were consistent with severe hepatobiliary involvement (McKenna *et al*., 2021).

The marked increase in alanine aminotransferase (ALT) and aspartate aminotransferase (AST), together with the high activity of alkaline phosphatase (ALP) and conjugated hyperbilirubinaemia, supported the presence of severe cholestasis and hepatocellular injury (Kesimer *et al*., 2015). In the absence of ultrasonographic appearance of GBM may vary according to the stage of bile gelation, ranging from echogenic biliary sludge in earlier stages to the classic organized kiwi-like pattern in advanced cases (Uusaed and Aldridge, 2022). In the present case, histopathological examination was essential not only to confirm the gallbladder lesion but also to characterize the extent of hepatic involvement.

Taken together, these findings emphasize the diagnostic value of integrating clinical examination, laboratory testing, and histopathological evaluation in dogs with complex hepatobiliary disease (Winter and Moses, 2023). In this case, the combination of severe jaundice, conjugated hyperbilirubinemia, inflammatory leukogram, ultrasonographic hepatic abnormalities, and histopathological evidence of GBM with associated hepatic lesions supported the diagnosis of a severe concurrent hepatobiliary disorder. Although early diagnosis is considered important for clinical decision-making in dogs with GBM, the outcome in the present case was unfavorable, since the dog deteriorated despite medical management and died. This case therefore highlights the potential severity of advanced hepatobiliary disease and the importance of timely and comprehensive diagnostic assessment.

Conclusion

The present case highlighted the importance of considering gallbladder mucocele as a differential diagnosis in dogs with severe jaundice and hepatobiliary dysfunction. A comprehensive diagnostic approach integrating imaging studies, hematological and biochemical analyses, immunological testing, and histopathological evaluation was essential to characterize the concurrent hepatobiliary lesions observed in this dog. Further studies are needed to better clarify the clinicopathological relationship between gallbladder mucocele and hepatic lesions in dogs with complex hepatobiliary disease.

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Conflict of Interest

The authors declare that there is no economic, personal or academic conflict of interest that could have inappropriately influenced the conduct of this research or the preparation of the manuscript. The authors declared that the manuscript is original, has not been published elsewhere, and confirmed the latest version before publication.

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