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Diagnosis and therapeutic management of leptospirosis in Shih Tzu – A case report

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Abstract

This report describes a severe form of acute leptospiral infection in a 7-month-old Shih Tzu presented with progressive weight loss, inappetence, vomiting, pale conjunctiva, respiratory distress, and biochemical evidence of acute hepatorenal dysfunction. Abdominal ultrasonography revealed bilateral but asymmetrical renal alterations and advanced tubular injury in the kidney. Hematology revealed anemia and leukocytosis. Serum biochemistry showed elevated LFT and KFT parameters. RT-PCR confirmed *Leptospira* infection. Treatment included amoxicillin–clavulanate (12 mg/kg IV BID), followed by doxycycline (5 mg/kg BID), intensive fluid therapy, gastroprotectants (pantoprazole at 1 mg/kg IV, SID followed by sucralfate 1 g orally every BID), antiemetics (maropitant citrate at 1 mg/kg SID), darbepoetin Alfa (0.6 µg/kg subcutaneously once weekly), iron supplementation iron sucrose (1 ml/10 kg weekly IV), hepatoprotective agents (ursodeoxycholic acid with silymarin suspension 1 tsp orally BID), Renal protective agents Virbac Pronefra (1 ml/kg BID) and nutritional support. A rare complication—oral ulcers progressing to tongue gangrene—developed during recovery. Despite severe multisystemic involvement, the dog responded favorably to prolonged and aggressive medical management. This case emphasizes the importance of early clinical suspicion, timely confirmatory diagnosis, ultrasonographic assessment of renal injury, and comprehensive supportive care.

Keywords: Leptospirosis, Shih Tzu, Acute kidney injury, Hepatorenal syndrome, ultrasonography; RT-PCR

Introduction

Leptospirosis is a contagious, globally significant bacterial zoonotic diseases caused by pathogenic, helically shaped spirochetes of the genus *Leptospira*. These thin, motile organisms infect a wide range of domestic and wild animals, as well as humans and persist in nature through chronically infected reservoir hosts that shed the organism in urine (Schuller *et al.*, 2015) [18]. Small rodents serve as the primary reservoirs, although dogs, cats, and humans may also harbor pathogenic leptospires. Important serovars include Icterohaemorrhagiae, Canicola, Pomona, Bratislava, Grippotyphosa, and Autumnalis. Leptospirosis is a multi-systemic disease primarily affecting the kidneys and liver, with possible involvement of the lungs, spleen, eyes, vascular endothelium, and muscle. Infection occurs through direct contact with urine from infected animals or through exposure to contaminated water or soil. Clinical presentations vary widely, ranging from subclinical infections to severe, life-threatening disease. Common signs in dogs include fever, anorexia, vomiting, lethargy, diarrhea, jaundice, dehydration, dyspnea, uveitis, and abortion, usually associated with acute renal failure, hepatic dysfunction, or hemolysis. Less common clinical manifestations include pulmonary hemorrhage and pleural effusion. Diagnosis is based on exposure history, clinical signs, and laboratory confirmation. Confirmatory tests include include PCR, bacterial culture, dark-field microscopy, and the microscopic agglutination test (MAT), which remains the gold standard for serologic diagnosis (Cumberland *et al.*, 1999) [5]. Severely affected dogs may present with dehydration or shock (Greene *et al.*, 2012) [9]. Antimicrobial therapy rapidly reduces fever and leptospiremia, thereby prevents fatal complications. Penicillin derivatives and doxycycline remain the recommended treatments for suspected canine leptospirosis (Sykes *et al.*, 2011) [20].

Case history and Clinical Findings

A 7-month-old female Shih Tzu dog was presented to the small animal clinic with a history of progressive weight loss, inappetence, and vomiting from past two days. Clinical examination revealed a cachectic body condition, pale mucous membranes, respiratory distress, and dullness. Hematology evaluation indicated anemia, while differential leukocyte and platelet counts were within normal range. Serum biochemistry showed marked elevations in liver and kidney function parameters and triglycerides (Table 1), suggestive of multi-organ involvement. Abdominal ultrasonography was performed using a computed sonographic BI Pro Aeroscan unit with a 7.0 MHz micro-convex transducer. The kidneys were evaluated in longitudinal, sagittal, and transverse planes. Ultrasonographic findings revealed hepatomegaly, abdominal effusion, tissue edema, and bilateral renal pathology characterized by diffuse cortical hyperechogenicity and loss of cortico-medullary distinction. The right kidney appeared mildly enlarged with preserved contours, consistent with acute interstitial nephritis, whereas the left kidney showed more advanced cortical and medullary echogenic alterations, prominent medullary echogenic foci, and small tubular dilatations, indicative of acute tubular injury. The left kidney was comparatively smaller than the right (Fig.1). For confirmatory diagnosis, blood samples were submitted for RT-PCR, which confirmed the presence of *Leptospira* spp. (Fig. 2).

Therapeutic management

Treatment was initiated with amoxicillin-clavulanate at 12 mg/kg IV BID for 15 days, followed by doxycycline at 5.0

mg/kg body weight IV BID for five days, as recommended by Greene and Decaro (2012) [9]. Gastroprotectants included pantoprazole at 1 mg/kg IV once daily for 15 days, followed by sucralfate 1 g orally every 12 hours for seven days. Vomiting was initially managed with ondansetron (0.5 mg/kg), but due to inadequate response, maropitant citrate at 1 mg/kg SID for 10 days was administered. To address anemia and support erythropoiesis, darbepoetin alfa (0.6 µg/kg was administered subcutaneously once weekly for four weeks, then once every two weeks as per Devadevi *et al.*, 2021) [6] and iron sucrose (1 ml/10 kg weekly for four doses) followed by oral Sharkoferol Pet syrup were administered. An immunosuppressive dose of prednisolone was given at 0.5 mg/kg orally once daily for five days, followed by 0.25 mg/kg for an additional three days. Fluid therapy for rehydration and anemia included Ringer's lactate, DNS, and Haemaccel at 10 ml/kg body weight BID for two weeks and SID for one week. Supportive care included B-complex (Inj. Tribivet, 0.6 ml IV daily for 15 days) followed by oral Zip-Vit, Renal protective agent Pronefra syrup (1 ml/kg BID) and hepato protective agent ursodeoxycholic acid with silymarin suspension (1 tsp orally twice daily). After 15 days, mirtazapine (<7 kg dose, SID) was used as an appetite stimulant for 10 days.

Complications: On the 14th day of treatment, the dog developed oral ulcers that progressed to tongue gangrene (Fig. 4). The necrotic portion was carefully removed, and daily oral cleansing with diluted povidone-iodine was performed and orogel ointment was applied. Additional complications included progressive emaciation, uveitis, corneal ulcer and mild bowing of the forelimbs.

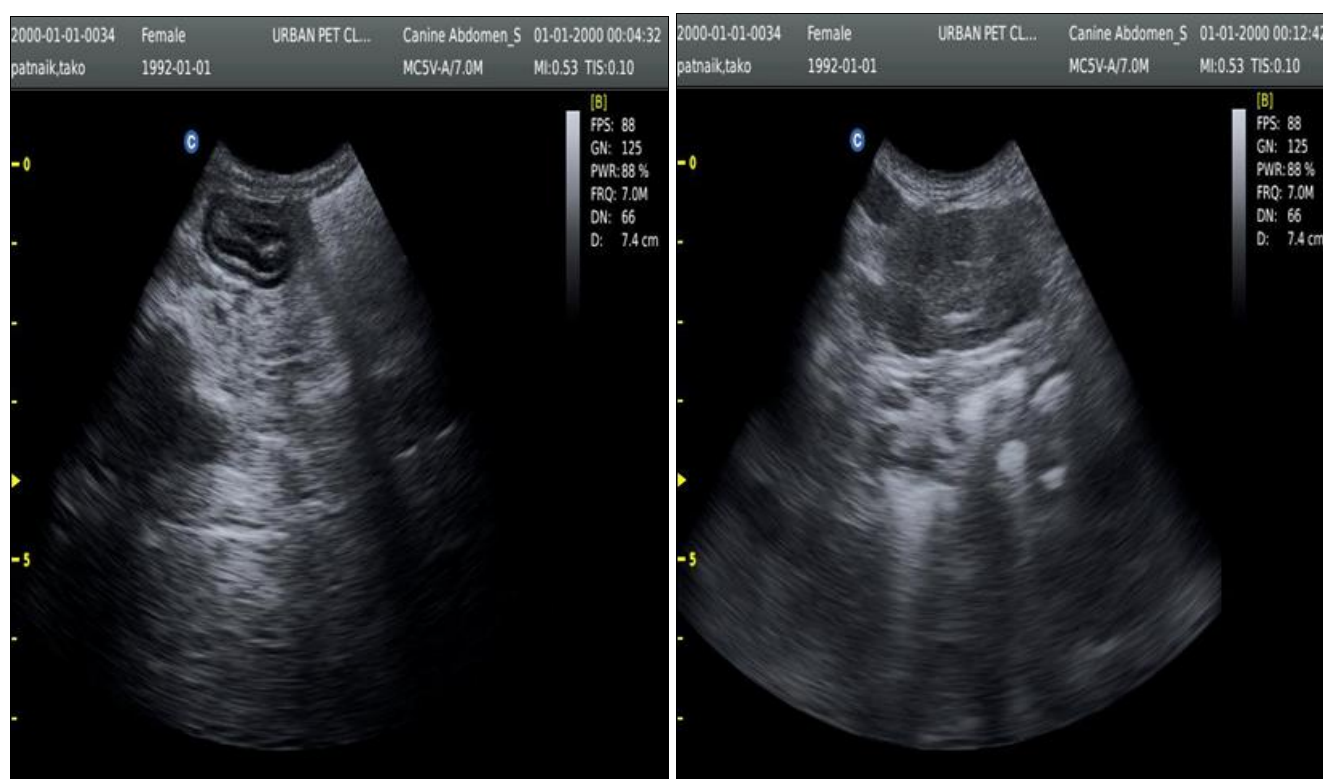


Fig 1: Nephrosonographic changes in dog with leptospirosis

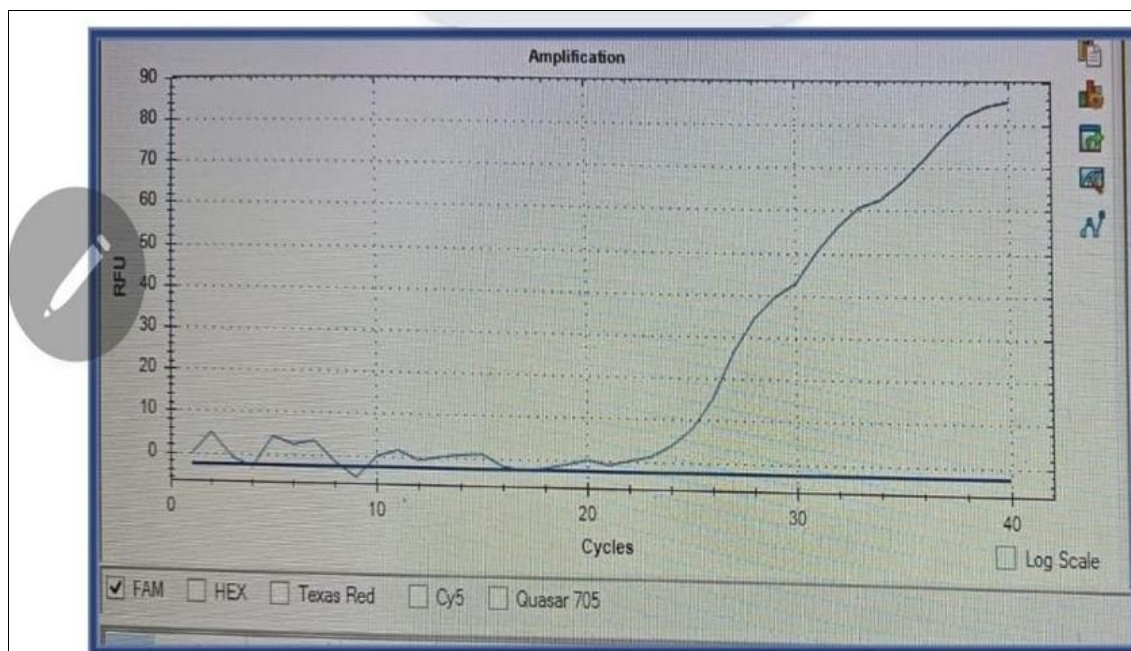


Fig 2: Real –time PCR assay revealed positive amplification for leptospira spp.,with Ct value approximately 27-30, confirming the presence of leptospiral DNA in tested sample

Table 1: Hematobiochemical findings in dog with leptospirosis

Parameter	0 th day	7 th day	14 th day	30 th day	45 th day	90 th day	Reference range *
Hemoglobin (g/dl)	11.8	9.5	6.3	6.0	4.7	8.6	14.1-20.1
TLC (10 ³ /cmm)	10.1	22.3	31.9	42.9	15.8	9.7	5.7- 14.2
DLC							42-84
N%	07	05	05	05	05	08	9-47
L%	90	92	92	93	93	87	2-10
E%	03	03	03	02	02	04	1-18
M%	00	00	00	00	00	01	2-12
RBC (millions /cumm)	4.55	4.04	2.73	2.61	2.06	3.5	4.95-7.87
PCV (%)	31.8	28.9	18.7	19.4	15.0	25.2	42-54%
MCV (fL)	69.1	71.7	68.4	74.3	15.0	72.1	64-76
MCH(Pg)	25.9	23.5	23.2	23.0	22.8	24.5	21-26
MCHC(g/dl)				32.0	31.3	33.9	33-36
Platlet count (×10 ³ /μL)	374	83	103	553	685	633	186-545
Serum creatinine (mg/dl)	8.5	5.9	4.44	3.95	1.95	2.8	0.6-1.4
BUN (mg/dl)	124	99.9	73.7	110.2	50.4	88.2	9-26
Total bilirubin (mg/dl)	6.3	0.5	0.6	0.4	0.4	0.7	0-0.2
Direct bilirubin (mg/dl)	2.8	0.1	0.2	0.2	0.2	0.3	0-0.1
Indirect bilirubin (mg/dl)	3.5	0.4	0.4	0.2	0.2	0.4	0.01
SGOT (U/L)	56	52.7	30.2	53.2	54.1	24.6	18-56
SGPT (U/L)	31	50.6	24.3	48.5	37.8	18.9	17-95
ALP (IU/L)	240	312	573	519	480	265	7-115
Total Protein (g/dl)	8	7.2	4.86	5.6	5.0	6	5.5-7.2
Albumin (g/dl)	2.9	2.3	2.5	2.2	2.0	2.5	3.2-4.1
Globulin (g/dl)	5.1	4.9	2.3	3.4	3.0	3.5	1.9-3.7
A/G ratio	0.56	0.46	1.08	0.64	0.66	0.71	0.9-1.9
Serum cholesterol (mg/dl)	1070	840	890	712	520	338	136-392
Serum triglycerides (mg/dl)	2460	2134	2087	1457	724	199	23-102

*Hematology Advia 2120 and chemistry (cobas) College of veterinary medicine, Cornell university 2017



Fig 3: a) Before treatment, b) After treatment

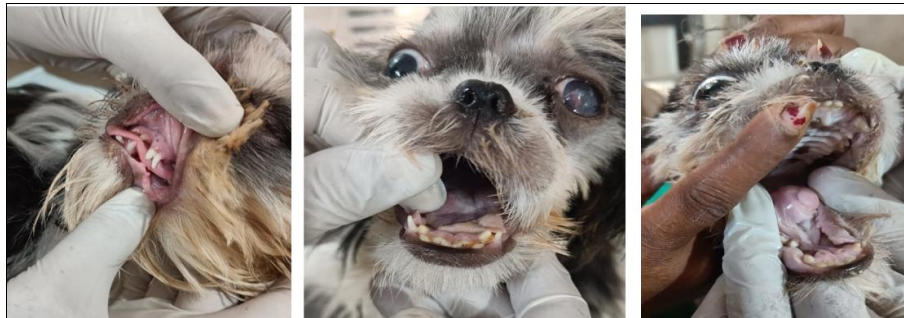


Fig 4: a.) 0th day normal tongue, b) 20th day necrotic tongue with corneal ulcer, c) 3rd month after partial glossectomy

The present case describes a severe multisystemic form of leptospiral infection in a young Shih Tzu, emphasizing the need for early clinical suspicion, prompt diagnosis and aggressive therapeutic intervention. Young dogs are more susceptible to acute and fulminant leptospirosis due to immature immune responses and are often more severely affected than adults (Greene *et al.* 2012) ^[9]. The dog in this report exhibited classical signs of acute leptospirosis—including anorexia, vomiting, lethargy, dehydration and pale mucous membranes—which align with descriptions provided by Greene *et al.* 2012 ^[9] and Schuller *et al.* (2015) ^[18]. Vomiting in renal failure may be attributed to uremic gastropathy and retention of uremic toxins. Hematological examination revealed a progressive decline in PCV and hemoglobin values, indicative of normocytic normochromic anemia, commonly associated with hemorrhage and blood loss in leptospirosis. Although leukogram and differential counts were normal on day 0, a progressive leukocytosis developed from day 7 onwards, suggesting ongoing inflammation. Platelet count decreased markedly by day 7 and gradually returned to normal by one and three months. These findings correspond with Abdullathief *et al.* (2018) ^[1] and Geetha and Selvaraju (2021) ^[8], who reported leukocytosis and mild anemia in affected dogs. Marked biochemical abnormalities reflected acute hepato-renal involvement, resulting from leptospiral colonization of renal tubules and hepatocellular injury during both the leptospiremic and immune phases. Similar biochemical alterations have been reported by Patel *et al.* (2021) ^[16]. Severe hypertriglyceridemia noted in this case may represent systemic inflammatory response and metabolic dysregulation associated with endotoxemia, consistent with previous reports (Khan *et al.*, 2009; Chandrasekaran *et al.* 2011) ^[14, 4]. Ultrasonography served as a valuable tool for assessing renal involvement and asymmetry of pathological changes. Diffuse cortical hyperechogenicity with loss of corticomedullary

distinction is typical of acute nephritis and tubular injury in leptospirosis. In this case, the left kidney showed more advanced damage and early reduction in size, suggesting asymmetric progression of leptospira-associated nephropathy. Ultrasonography is increasingly recognized as an important adjunct for evaluating disease severity, guiding prognosis and differentiating leptospirosis from other causes of acute kidney injury (Greene *et al.*, 2012; Sonet *et al.*, 2017) ^[9]. Additional findings—such as hepatomegaly, abdominal effusion and tissue edema—support systemic endothelial and vascular injury consistent with leptospiral pathophysiology. RT-PCR provided definitive confirmation and is highly sensitive during the acute phase before detectable antibody titers develop. Its use facilitates early therapeutic decision-making (Geetha and Selvaraju, 2021) ^[8].

Treatment followed the recommendations of Greene and Decaro (2012) ^[9], emphasizing early antimicrobial administration to reduce the leptospiremic phase and prevent renal colonization. Doxycycline, known for eliminating renal shedding, was combined with amoxicillin–clavulanate broad antimicrobial coverage. Extensive supportive care, including fluid therapy, antiemetics, hepatoprotective agents and nutritional support, was essential due to severe hepatic and renal dysfunction (Greene and Decaro, 2012) ^[9]. The development of anemia in this patient necessitated darbepoetin therapy, which has been used increasingly in dogs with chronic or acute kidney injury to stimulate erythropoiesis. Iron supplementation complemented erythropoietic therapy, addressing potential functional iron deficiency this supported by findings of Devadevi *et al.* (2022) ^[7] who stated darbepoietin is a ideal drug of choice against CKD induced anemia in dogs. Transient immunosuppressive therapy with prednisolone may have been beneficial in mitigating immune-mediated components of tissue injury, though its use requires caution, as noted by Pankaj Kumar Patel *et al.*, (2018) ^[15]. A notable and rare

complication observed in this case was the development of oral ulcers progressing to tongue gangrene, consistent with the findings of Jitendra Bargujar *et al.* (2021) [13]. Mucosal necrosis is uncommon in leptospirosis, and possible contributing factors include uremia, severe vascular injury, ischemia due to shock, and secondary bacterial infection. Progressive emaciation and mild limb deformities during recovery may reflect chronic catabolic state and mineral imbalances associated with renal dysfunction, particularly impaired phosphorus excretion and reduced vitamin D activation (Grünberg, 2018) [11]. Despite severe multisystemic illness and significant complications, the dog demonstrated substantial improvement with prolonged and intensive therapy. This case reinforces the value of early diagnostic imaging, timely molecular confirmation and comprehensive supportive management. It also highlights the need for clinicians to recognize atypical complications such as mucosal necrosis, which may influence prognosis and require specialized intervention.

Conflict of Interest

Not available

Financial Support

Not available

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