CASE REPORT
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ELECTROLYTE IMBALANCES, COAGULOPATHY, AND MULTIORGAN DYSFUNCTION IN A DOG WITH CONCURRENT INFECTION BY *BABESIA GIBSONI* AND *LEPTOSPIRA* SPP

[1]Abhijith S.P., [2]Abdul Kalam A, [3]Krishnaswamy Ajjampur, and [4] Anirudh Ajjampur

[1,2]Consultant Veterinarian, RMV Multispecialty Veterinary Clinic, Bengaluru
[3]Director, RMV Multispecialty Veterinary Clinic, Bengaluru
[4]Chief Veterinary Surgeon, RMV Multispecialty Veterinary Clinic, Bengaluru

*Corresponding author: abhijithsp560@gmail.com

ABSTRACT

Concurrent canine leptospirosis and babesiosis, though rare, can synergistically exacerbate systemic illness, leading to multi-organ dysfunction and posing a significant diagnostic and therapeutic challenge.

A two-year-old male Doberman was presented with an acute onset of fever, lethargy, anorexia, progressive weakness, and jaundice. Clinical examination revealed marked pallor, icterus, tachycardia, and dehydration. Hematobiochemical evaluation demonstrated anemia, thrombocytopenia, azotaemia, elevated hepatic enzymes, increased serum creatinine, hyperbilirubinemia, and significant electrolyte imbalances. Molecular confirmation using a multi-antigen-targeted polymerase chain reaction (PCR) assay on Day 1 identified a concurrent co-infection with *Leptospira* spp. and *Babesia gibsoni*. Despite prompt diagnosis and initiation of appropriate therapeutic interventions, the animal showed rapid clinical deterioration and eventually succumbed to the illness. This clinical case report highlights the severe secondary complications arising from the synergistic effects of leptospiral and babesial co-infection, leading to multisystemic involvement and irreversible organ damage.

Keywords: mods, dic, sirs, leptospirosis, babesiosis

INTRODUCTION

Canine leptospirosis and babesiosis are clinically significant infectious diseases of global concern, associated with high morbidity and mortality in dogs. Both conditions target multiple organ systems; particularly the hepatic, renal, and hematopoietic-and can result in severe, life-threatening complications if not diagnosed and treated promptly.

Leptospirosis is a widespread zoonosis caused by pathogenic spirochetes of the genus *Leptospira*. Dogs serve as both incidental and maintenance hosts depending on the infecting serovar (Goldstein, 2010). Transmission typically occurs via exposure to water, soil, or urine contaminated by reservoir hosts such as rodents (Levett, 2001). The disease manifests in a wide clinical spectrum-from mild, subclinical infection to fulminant multiorgan failure. The principal target organs are the kidneys and liver, resulting in interstitial nephritis and hepatocellular necrosis (Adler & de la Peña Moctezuma, 2010). Common clinical findings include fever, vomiting, icterus, dehydration, and variable degrees of azotaemia and hepatic enzyme elevation (Birnbaum *et al.,* 1998; Goldstein, 2010). Diagnosis is based on serology and molecular assays. The microscopic agglutination test (MAT) remains the gold standard but may be negative early in infection; polymerase chain reaction (PCR) allows earlier detection before seroconversion (Miotto *et al.,* 2018). Treatment involves parenteral penicillin or ampicillin to clear leptospiraemia, followed by doxycycline to eliminate renal carriage, alongside aggressive supportive therapy (Goldstein, 2010; Klaasen *et al.,* 2014).

Canine babesiosis is a tick-borne hemoprotozoan disease caused by intraerythrocytic *Babesia* spp., primarily *B. canis* and *B. gibsoni* (Irwin, 2009; Solano-Gallego & Baneth, 2011). *B. gibsoni* infections are increasingly reported across Asia and can also be transmitted through dog fights, or transplacentally (Jefferies *et al.,* 2007). The pathogenesis involves hemolysis due to direct parasitism and immune-mediated mechanisms, leading to anemia, thrombocytopenia, and systemic inflammation (Jacobson, 2006). Diagnosis relies on microscopic detection of piroplasms or molecular confirmation by PCR, which enables species identification (Birkenheuer *et al.,* 2003; Iqbal *et al.,* 2020).

While infections with *Leptospira* spp. or *Babesia* spp. individually are well recognized, concurrent infection is uncommon but poses complex diagnostic and therapeutic challenges with a guarded prognosis (Greene *et al.,* 2006; Sykes *et al.,* 2011). Co-infection with multiple vector-borne or zoonotic pathogens is increasingly recognized in endemic regions; however, concurrent *Leptospira* and *Babesia* infections in dogs are rarely documented (Shaw *et al.,* 2001; Solano-Gallego *et al.,* 2016; Ajith *et al.,* 2016). Overlapping clinical and biochemical abnormalities often mask dual infections unless molecular testing is pursued. Such co-infections can intensify systemic pathology: hemolysis from babesiosis increases bilirubin

load, compounding hepatic injury from leptospirosis, while simultaneous renal and endothelial damage predisposes to acute kidney injury, coagulopathy, and multiple organ dysfunction (Jacobson, 2006; Miotto *et al.,* 2018). Recognizing these synergistic effects is crucial for timely diagnosis and effective management in co-endemic settings.

CASE HISTORY & OBSERVATIONS

A two-year-old intact male Doberman was presented to the clinic with a sudden onset of high fever, marked lethargy, anorexia, and progressive weakness accompanied by visible jaundice. The owner reported that the dog had been dull and inappetent for the past 2–3 days,

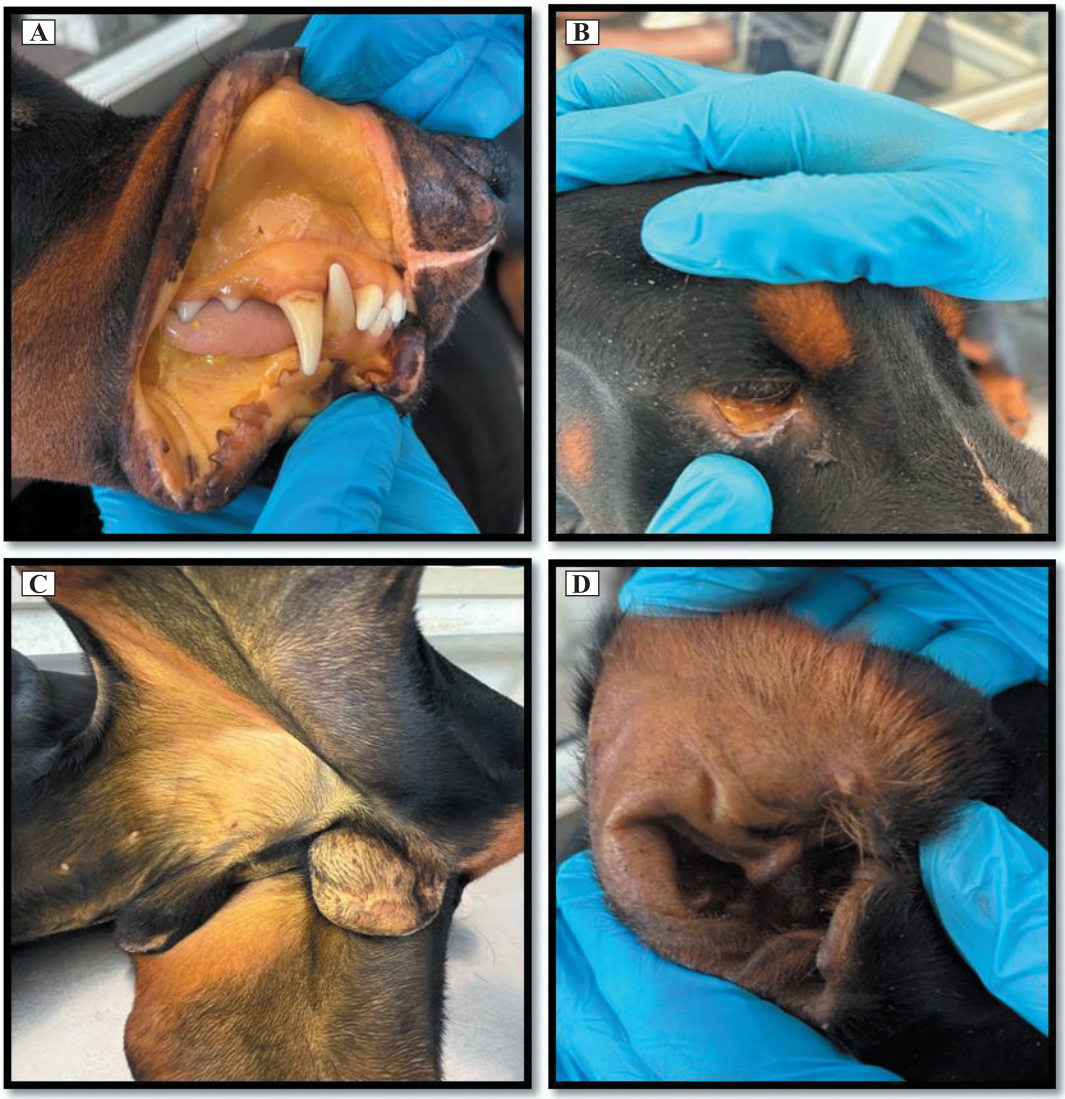

Fig. 1. Generalized Icterus

Table 1. Hematobiochemical Parameters on Day 0,1 & 3

Parameter	Day 0	Day 1	Day 3	Reference Range
Hemoglobin (g/dL)	7.8	6.5	5.2	12 – 18
Packed Cell Volume (%)	23	19	15	37 – 55
Total RBC ($\times 10^6/\mu L$)	3.1	2.6	2.1	5.5 – 8.5
Mean Corpuscular Volume (fL)	74	73	72	60 – 77
Mean Corpuscular Hemoglobin (pg)	25	26	27	19 – 23
Mean Corpuscular Hgb. Conc. (g/dL)	33	34	35	32 – 36
Total WBC ($\times 10^3/\mu L$)	19.5	26	42	6 – 17
Neutrophils (%)	84	90	93	60 – 77
Lymphocytes (%)	9	6	4	12 – 30
Monocytes (%)	6	3	2	3 – 10
Eosinophils (%)	1	1	1	2 – 10
Platelets ($\times 10^3/\mu L$)	95	68	54	200 – 500
Blood Urea Nitrogen (mg/dL)	178	190	210	10 – 28
Creatinine (mg/dL)	5	7.6	12.6	0.5 – 1.5
Total Bilirubin (mg/dL)	5.6	7.9	8.3	0.1 – 0.6
Direct Bilirubin (mg/dL)	4.9	6.9	7.1	0 – 0.2
ALT (IU/L)	460	520	610	10 – 100
AST (IU/L)	380	420	480	10 – 50
ALP (IU/L)	560	610	680	20 – 150
Total Protein (g/dL)	5.2	4.8	4.4	5.5 – 7.5
Albumin (g/dL)	2.1	1.8	1.5	2.5 – 4.0

with rapid deterioration in activity and hydration status. On clinical examination, the patient exhibited noticeable pallor of the mucous membranes, generalized icterus (Fig.1;A,B,C,D), tachycardia, and moderate dehydration. Palpation revealed mild abdominal discomfort without appreciable organomegaly. Hematological evaluation demonstrated severe anemia and thrombocytopenia, while serum biochemistry indicated azotaemia, significantly elevated liver enzymes, hyperbilirubinemia, elevated creatinine, and electrolyte imbalances suggestive of renal and hepatic involvement. Molecular diagnostic testing, performed using a multi-antigen PCR assay (outsourced to an external laboratory), confirmed concurrent infection with *Leptospira* spp. and *Babesia gibsoni*.

The hematological profile revealed progressively worsening anemia, with hemoglobin declining from 7.8 g/dL on Day 0 to 6.5 g/dL on Day 1 and 5.2 g/dL by Day 3, accompanied by falling packed cell volume and RBC count. The leukogram showed progressive leukocytosis with marked neutrophilia, rising from 19.5 $\times 10^3$/

µL (84%) on Day 0 to 42 ×10³/µL (93%) by Day 3, reflecting a systemic inflammatory response (SIRS). Thrombocytopenia was pronounced and progressive, with platelet counts dropping from 95 ×10³/µL on Day 0 to 54 ×10³/µL by Day 3. This suggests consumptive coagulopathy and evolving disseminated intravascular coagulation (DIC), a known complication of both babesiosis (via hemolysis-mediated coagulation activation) and leptospirosis (via vasculitis and endothelial injury).

Overall, the CBC trends reflect rapidly progressive multi-organ dysfunction with SIRS and DIC, highlighting the poor prognosis associated with concurrent *Babesia gibsoni* and leptospiral infections in dogs.

In leptospirosis, AKI arises due to direct leptospiral nephritis, tubular necrosis, and ischemic injury, while babesiosis contributes through hemolysis-induced tubular damage and hypoxic renal injury (Greene *et al.,* 2012; Jacobson, 2006).

Hepatic markers also worsened over the same period. Total bilirubin increased from 12.4 to 16.8 mg/dL and direct bilirubin from 5.6 to 8.3 mg/dL, consistent with cholestatic jaundice and hepatocellular injury. Concurrent elevations in ALT (460 → 610 IU/L), AST (380 → 480 IU/L), and ALP (560 → 680 IU/L) reflect hepatocellular necrosis and cholestasis, which may result from systemic hypoxia due to babesial hemolysis and direct leptospiral hepatotoxicity (Jacobson, 2006; Greene *et al.,* 2012).

Protein alterations were notable: total protein decreased from 5.2 to 4.4 g/dL, primarily due to albumin loss (2.1 → 1.5 g/dL), whereas globulin remained relatively stable (3.1 → 2.9 g/dL).

Collectively, these trends indicate progressive multi-organ involvement—renal, hepatic, and hematopoietic—with worsening clinical severity. The combination of AKI, hepatic dysfunction, and hypoproteinemia significantly increases

Table 2. Electrolyte Profile & Clotting Parameters on Day 0

Parameter	Result	Reference Range
Sodium (mmol/L)	128	145 – 154
Potassium (mmol/L)	6.8	3.5 – 5.8
Chloride (mmol/L)	92	105 – 115
Calcium (mg/dL)	6.9	9 – 11.5
Phosphorus (mg/dL)	19.5	2.5 – 6
Prothrombin Time (PT, sec)	28	8 – 14
Activated Partial Thromboplastin Time (aPTT, sec)	68	12 – 20
Fibrinogen (mg/dL)	110	150 – 400

the risk of SIRS, MODS, and poor clinical outcome in co-infected dogs.

The coagulation panel revealed a markedly prolonged prothrombin time (PT: 28 sec; RR: 8–14) and activated partial thromboplastin time (aPTT: 68 sec; RR: 12–20), along with hypofibrinogenemia (110 mg/dL; RR: 150–400). These abnormalities indicate a severe coagulopathy consistent with disseminated intravascular coagulation. In canine babesiosis, intravascular hemolysis and systemic inflammation trigger widespread activation of the coagulation cascade, leading to consumption of clotting factors and platelets (Jacobson & Clark, 1994). Similarly, leptospirosis is well documented to cause vasculitis, endothelial damage, and hepatic dysfunction, all of which impair coagulation and potentiate hemorrhagic diathesis (Greene *et al.,* 2012). The concurrent infection likely acted synergistically, with babesial hemolysis and leptospiral vasculitis amplifying systemic inflammatory response syndrome (SIRS), ultimately exhausting coagulation factors and fibrinogen reserves. Such derangements predispose to spontaneous bleeding, petechiation, and multi-organ dysfunction, correlating with the grave prognosis in this case.

TREATMENT & DISCUSSIONS

A combination therapy consisting of Doxycycline at 5 mg/kg IV twice daily and Ampicillin at 30 mg/kg IV twice daily was administered. Hepatic support was provided using intravenous N-acetylcysteine at 140 mg/kg once daily to replenish glutathione stores and mitigate oxidative stress. Fluid therapy with Ringer's Lactate was promptly initiated to correct dehydration and maintain renal perfusion. Analgesic management included Buprenorphine at 0.02 mg/kg IV twice daily, while Pantoprazole at 1 mg/kg IV once daily was given to reduce gastric acidity.

Despite these therapeutic interventions and timely diagnosis, the pet unfortunately succumbed to the illness, underscoring the vital importance of early detection and aggressive management. This case further demonstrates the heightened severity of co-infections, which can accelerate disease progression and precipitate systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), and disseminated intravascular coagulation (DIC).

Hyperkalemia in this case likely results from acute kidney injury (AKI), reduced renal excretion of potassium, and intravascular hemolysis caused by babesiosis, which releases intracellular potassium into circulation (Jacobson, 2006). The hypocalcemia with concurrent hyperphosphatemia is consistent with

advanced renal failure, where reduced glomerular filtration impairs phosphate clearance, leading to secondary calcium–phosphorus imbalance (Bartges & Polzin, 2011).

SIRS is a frequent complication of severe leptospirosis and babesiosis. In leptospirosis, vascular endothelial injury, cytokine storm, and hypovolemia precipitate multi-organ dysfunction, while in babesiosis, hemolysis, nitric oxide overproduction, and immune-mediated injury drive systemic inflammation (Adler and de la Peña Moctezuma, 2010; Jacobson, 2006). When combined, these mechanisms act synergistically, overwhelming compensatory mechanisms and leading to MODS and DIC. Hypoalbuminemia may result from hepatic synthetic failure, proteinuria due to renal involvement, and systemic inflammatory protein redistribution, all of which are described in severe leptospiral and babesial infections (Greene *et al.,* 2012; Bartges & Polzin, 2011).

Clinical recognition of MODS is based on dysfunction across two or more organ systems, such as renal failure, hepatic insufficiency, hematologic derangements, and cardiovascular instability. Once MODS develops, prognosis becomes guarded to poor, despite aggressive therapy (Goldstein, 2010; Jacobson, 2006).

SUMMARY

This report contributes to existing literature by detailing a rare case of *Leptospira* and *Babesia gibsoni* co-infection in a young Doberman, which culminated in MODS and DIC despite aggressive treatment. It emphasizes the synergistic pathogenesis of these pathogens, the diagnostic role of PCR assays, and the challenges in clinical management of dual infections.

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