Short Communication

CANINE EHRLICHIOSIS INDUCED MULTI-ORGAN DYSFUNCTION SYNDROME IN A LABRADOR RETRIEVER: A CASE REPORT

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ABSTRACT

Canine monocytic ehrlichiosis is a tick-borne infectious disease of dogs caused by Ehrlichia canis, transmitted primarily through Rhipicephalus sanguineus. The disease typically progresses through acute, subclinical, and chronic stages, each characterized by distinct hematological and clinical alterations. A three-years-old male Labrador Retriever was presented to the Small Animal Medicine Unit, Veterinary College Hospital, Bengaluru, with a history of progressive anorexia, vomiting, and lethargy for the past seven days.Clinical examination revealed pallor of mucous membranes, multiple lymphadenopathies, mild dehydration, and tick infestation along with enlarged hepatic silhouette on abdominal palpation. Hematological analysis revealed normocytic normochromic non-regenerative anemia and thrombocytopenia, while serum biochemistry indicated markedly elevated alanine aminotransferase (ALT), aspartate aminotransferase (AST), and total bilirubin levels. Molecular confirmation by multiplex PCR detected genome of Ehrlichia canis, establishing a diagnosis of Canine Monocytic Ehrlichiosis. The dog was treated with inj. doxycycline @ 10 mg/kg IV q24h for three days followed by oral route along with supportive treatment. Initial improvement was observed; however, three weeks later, the dog developed azotemia and persistent elevation of BUN and creatinine, consistent with chronic kidney disease (CKD) secondary to multi-organ dysfunction syndrome (MODS).Therefore, early diagnosis combined with effective tick control remains essential, as advanced cases often carry a guarded prognosis despite appropriate antibiotic therapy.

Keywords: *Ehrlichia canis*, labrador retriever, hepatic injury, renal dysfunction, multi-organ dysfunction syndrome

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INTRODUCTION

Canine monocytic ehrlichiosis (CME) is an important and globally distributed tick-borne infectious disease of dogs caused by the obligate intracellular, Gram-negative bacterium Ehrlichia canis transmitted primarily by the brown dog tick, Rhipicephalus sanguineus. (Harrus *et al*., 1997; Dantas-Torres *et al*., 2022; Mylonakis *et al*., 2001; Hegarty and Breitschwerdt, 1994). In India, E. canis infection has been reported from various regions with varied prevalence (Harikrishnan *et al.,* 2009; Dhankar *et al.,* 2011; Lakshmanan *et al*., 2011).

The clinical spectrum of CME ranges from acute, subclinical, to chronic phases (Harrus *et al.,* 1997; Dantas-Torres *et al.,* 2022; Mylonakis *et al.,* 2001). Acute CME typically manifests as fever, anorexia, lethargy, lymphadenopathy, epistaxis, petechiae, and variable hematologic abnormalities, while chronic infection may lead to persistent immune-mediated disorders, bleeding tendencies, and secondary infections (Rikihisa, 1991; Harrus and Waner, 2012; Waner *et al.,* 1997). Subclinical infection is common, often presenting with mild or non-specific signs but harbouring potential risks for progression to severe disease under immunosuppression or co-infection (Harrus and Waner, 2012; Waner *et al.,* 1997).

The diagnosis of CME is based on direct detection of morulae in blood or tissue cytology, serological detection of antibodies (indirect immunofluorescence, ELISA), and molecular identification by polymerase chain reaction (PCR) (Mylonakis *et al*., 2001; Neer *et al*., 2002; Dantas-Torres *et al*., 2022; Greig *et al*., 1996).

Management of CME usually involves doxycycline as the drug of choice, sometimes in combination therapies, but chronic forms may be refractory and require prolonged or repeated treatments (Neer *et al.*, 2002; Waner *et al.*, 1997).

CME continues to pose a notable veterinary challenge in endemic areas due to its multisystemic nature, variable clinical presentations, and capacity to inflict significant deleterious effects on the health of companion and working animals worldwide (Irwin, 2010; Dantas-Torres *et al.*, 2022; Dumler *et al.*, 2001). Ongoing research in vector biology, molecular diagnostics, and pathogen genomics is crucial to controlling and understanding this complex and evolving disease.

CASE HISTORY AND OBSERVATIONS

A three-year-old male Labrador Retriever, weighing approximately 28 kg, was presented to the Small Animal Medicine Unit, Veterinary College Hospital, Hebbal, Bengaluru, with a history of anorexia, vomiting, lethargy, and icterus for the past seven days. Also, the pet had a history of irregular deworming and absence of tick control for the past several months. On clinical examination, the dog appeared dull, depressed, and mildly dehydrated (5%), with a rectal temperature of 103.2 °F. The mucous membranes were

pale and icteric, and multiple adult ticks were noticed along the neck, ears, and interdigital regions. Abdomen palpation revealed mild hepatomegaly with a smooth border. Multiple peripheral lymph nodes were enlarged, and cardiac and pulmonary auscultation were unremarkable.

Hematological examination revealed normocytic, normochromic, non-regenerative anemia characterized by decreased hemoglobin (8.4 g/dL) and packed cell volume (25%), with normal erythrocyte indices (MCV: 65 fL; MCHC: 33 g/dL), a low reticulocyte count (<60,000/µL)and marked thrombocytopenia (platelets: 90 × 10^3/µL). Serum biochemical evaluation revealed elevated hepatic enzymes (ALT: 168 U/L; AST: 134 U/L), increased total bilirubin (2.4 mg/dL), and hypoalbuminemia (2.0 g/dL), indicating acute hepatocellular injury. Mild azotemia was initially observed (BUN: 32 mg/dL; creatinine: 1.7 mg/dL), which was presumed to be prerenal due to dehydration. Peripheral blood smear examination revealed the presence of intracellular morulae within monocytes confirming *Ehrlichia canis* (Fig.1.1).

Abdominal ultrasonography revealed diffuse hepatic hyperechogenicity and mild gallbladder distension (Fig.1.4), consistent with hepatic parenchymal degeneration. Kidneys appeared slightly hyperechoic, but of normal size and echotexture. Multiplex PCR (Pet Biotech, GKVK, Bengaluru) confirmed the presence of Ehrlichia canis, establishing a definitive diagnosis of Canine Monocytic Ehrlichiosis (CME) with acute hepatic involvement.

TREATMENT AND DISCUSSION

Following the confirmation of *Ehrlichia canis* infection by multiplex PCR, the dog was treated with inj. doxycycline @ 10 mg/kg iv, sid for three days, after which the treatment was transitioned to the oral route.

Comprehensive supportive therapy was instituted to mitigate hepatic dysfunction and prevent further systemic complications. Intravenous fluid therapy with Normal Saline administered to correct dehydration and ensure adequate hepatic and renal perfusion. Hepatoprotective agents, including silymarin (20 mg/kg PO q24h) and ursodeoxycholic acid (10 mg/kg PO q24h), were prescribed to promote hepatocellular recovery and enhance bile flow. N-acetylcysteine (140 mg/kg IV, diluted in normal saline) was administered to attenuate oxidative stress and minimize free radical–induced hepatic injury. Additionally, vitamin B-complex supplementation was provided to support hepatic regeneration and reduce oxidative damage.

During the initial phase of treatment, mild clinical improvement was observed in appetite and decreased serum bilirubin levels. However, by the end of the third week, renal parameters began to worsen, with elevated BUN and creatinine levels. Urinalysis revealed isosthenuria and mild proteinuria, consistent with acute kidney injury (AKI). Fluid therapy was adjusted to half-strength Ringer's lactate and 0.45% saline with 2.5% dextrose, and furosemide @ 2 mg/kg im q12hwas administered briefly to promote

diuresis. Despite supportive measures, renal values continued to deteriorate, progressing to the stage of chronic kidney disease (CKD).

During the course of therapy, the dog initially showed clinical improvement; however, by the third week post-diagnosis, it developed progressive azotemia (BUN: 67 mg/dL; creatinine: 3.8 mg/dL), isosthenuria (USG: 1.010), and persistent lethargy.

In the present case, the occurrence of jaundice can be attributed to severe hepatocellular injury and cholestasis, consistent with the observations of Raguvaran *et al.* (2020), who emphasized that ehrlichiosis-associated jaundice arises from hepatocellular damage, hemolysis, and bile flow impairment, and that early diagnosis combined with supportive and antibiotic therapy markedly improves clinical recovery.

Follow-up ultrasonography revealed reduced renal cortical echogenicity and mild nephromegaly (Fig.1.2, Fig.1.3), confirming the development of Acute Kidney Injury (AKI) secondary to systemic ehrlichiosis. Despite aggressive fluid therapy and renal protectants, the renal function continued to deteriorate over the next few weeks, transitioning into chronic kidney disease (CKD) characterized by sustained azotemia, weight loss, and inappetence. The dog eventually succumbed to ehrlichiosis-induced multi-organ dysfunction, where initial hepatic damage culminated in irreversible renal failure.

The pathogenesis of ehrlichiosis-induced renal damage is attributed to immune complex deposition in the glomeruli, leading to glomerulonephritis, interstitial nephritis, and renal fibrosis (Eddlestone *et al.,* 2007; Harrus *et al.*, 1996). Ehrlichia canis infection triggers a strong humoral immune response, resulting in circulating immune complexes that cause vasculitis, endothelial injury, and chronic inflammatory changes in vital organs (Harrus and Waner, 2012). In this case, initial acute hepatic injury and endothelial dysfunction likely caused a systemic inflammatory response, progressing to multi-organ dysfunction syndrome (MODS) involving both the liver and kidneys. The involvement of renal pathology is believed to be a leading cause of mortality associated with Ehrlichia canis infection in dogs (Chandrasekar *et al.,* 2022).

The elevated liver enzymes (ALT, AST, and bilirubin) and hypoalbuminemia observed were consistent with hepatocellular injury described in previous reports (Mylonakis *et al.*, 2004). Subsequent renal involvement with progressive azotemia supports the theory that ehrlichiosis can induce secondary renal failure, either through immune-mediated mechanisms or direct vascular compromise (Buhles *et al*., 1974; Eddlestone *et al.*, 2007).

Despite intensive therapy, including renal diet modification, fluid therapy, and antioxidant supplementation, the dog's condition gradually deteriorated, and death occurred due to the pet succumbed due to irreversible multi-organ failure.

CONCLUSION

This case highlights the sequential progression of Ehrlichia canis infection from acute hepatic injury to renal involvement, ultimately culminating in chronic kidney disease (CKD). The findings underscore that Canine monocytic ehrlichiosis (CME) is not only confined to hematological abnormalities but also represents a multisystemic disorder capable of inducing irreversible organ dysfunction through immune-mediated and inflammatory pathways. The transition from acute kidney injury (AKI) to CKD observed in this Labrador Retriever exemplifies the long-term renal consequences of systemicehrlichial infection. Early molecular diagnosis using PCR, along with comprehensive hematobiochemical evaluation, is crucial for timely intervention. Although doxycycline remains the cornerstone of therapy, adjunctive hepatoprotective, nephroprotective, and antioxidant support play a pivotal role in limiting disease progression and preventing multiple organ damage. In endemic regions such as Bengaluru, clinicians should maintain vigilance for ehrlichiosis in dogs presenting with concurrent hepatic and renal abnormalities to enable early management and prevent progression to multi-organ failure.

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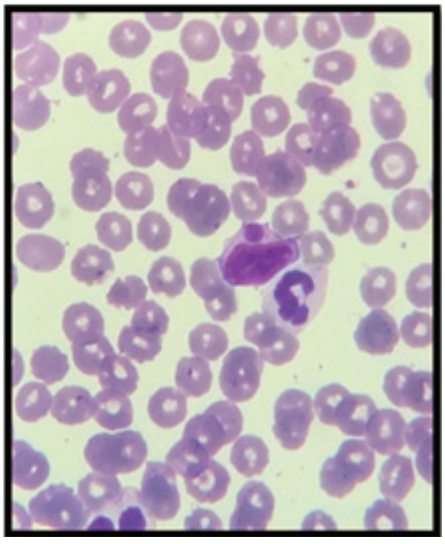

Fig.1.1. Peripheral blood smear revealing the presence of *Ehrlichia canis* morulae within monocytes (Giemsa stained, 100X magnification)

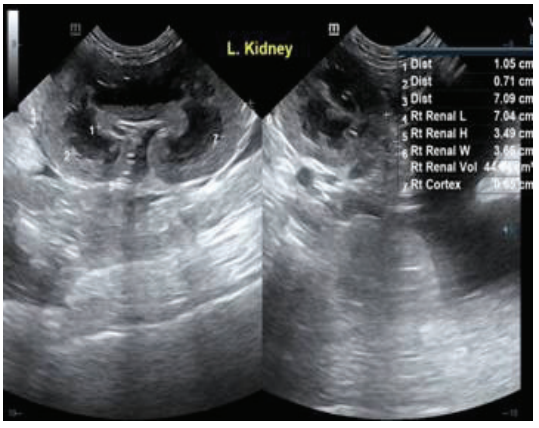

Fig.1.2. Ultrasound image showing reduced renal cortical echogenicity and mild nephromegaly (left kidney)

Fig.1.3. Ultrasound image showing reduced renal cortical echogenicity and mild nephromegaly (right kidney)

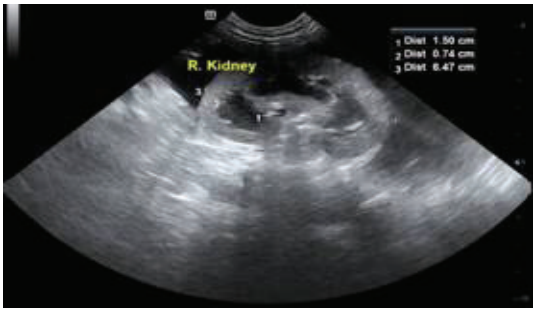

Fig.1.4. Diffuse hepatic hyperechogenicity

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