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Infectious aetiopathology of peripheral oedema in dogs: a clinico-pathological and molecular study

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Abstract

Peripheral oedema is a common but diagnostically challenging clinical finding in dogs, reflecting underlying disturbances in vascular, oncotic, or lymphatic homeostasis. Infectious agents, particularly vector-borne pathogens and filarial parasites contribute to oedema formation through mechanisms such as vasculitis, hypoproteinaemia, and lymphatic obstruction. This study investigated the aetiopathology of peripheral oedema associated with selected infectious agents in dogs. Thirty dogs presented with non-traumatic peripheral oedema were evaluated using clinical examination, haematology, serum biochemistry, oedema fluid analysis, ultrasonography, electrocardiography, echocardiography and molecular diagnostics. Conventional polymerase chain reaction was employed to detect Ehrlichia canis, Anaplasma phagocytophilum, Babesia gibsoni, Hepatozoon canis, Brugia malayi, and Borrelia burgdorferi. Positive amplicons were sequenced for confirmation. Wet film and blood smear examination detected sheathed microfilariae and Babesia gibsoni. Molecular analysis confirmed Brugia malayi and Babesia gibsoni as the predominant infectious agents associated with peripheral oedema in the study, while other pathogens were not detected. Oedema fluid was characterised as modified transudate. Significant haematological and biochemical alterations, including leucocytosis with neutrophilia, hyperglobulinaemia and increased AST levels, were observed. Although treatment resulted in clinical improvement and clearance of parasitaemia, recurrence of oedema occurred in several cases. This study demonstrates that Brugia malayi, Babesia gibsoni and cardiac diseases play a significant role in the aetiopathology of peripheral oedema in dogs. Aetiology-specific treatment alone often gave temporary improvement, emphasising the need for vasculitis-directed therapy

Keywords: *Peripheral oedema, Brugia malayi, Babesia gibsoni, dogs, vector-borne diseases, PCR*

Peripheral oedema is a commonly encountered clinical finding in dogs and refers to the abnormal accumulation of interstitial fluid within soft tissues, particularly involving the limbs, head, neck, or ventral body regions (German and Hall, 2021). The development of peripheral oedema is attributed to disturbances in plasma oncotic pressure, capillary hydrostatic pressure, vascular permeability or lymphatic drainage. Clinically, it may be classified as localised or

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generalised, and as pitting or non-pitting, depending on its distribution and physical characteristics (Walton, 2023).

Peripheral oedema has a multifactorial aetiology. Infectious causes especially vector-borne diseases such as ehrlichiosis, anaplasmosis, babesiosis, hepatozoonosis, borreliosis and filarial infections, contribute to oedema through mechanisms including vasculitis, immune-complex mediated vascular injury, hypoproteinaemia, increased capillary permeability and lymphatic inflammation or obstruction (Whelchel et al., 2023). In addition, cardiac diseases represent a major non-infectious cause of peripheral oedema, while hepatic and renal dysfunction may act as primary or contributory factors by altering fluid balance and plasma protein concentrations (Ware, 2007).

Despite its frequent occurrence, effective therapeutic management of peripheral oedema remains challenging, particularly when infectious agents are involved. Accurate identification of the underlying infectious aetiology, along with systematic evaluation of clinico-pathological, haematological, and biochemical alterations, is essential for appropriate diagnosis, targeted therapy and improved prognosis. Hence, the present study was undertaken to investigate the aetiopathology of peripheral oedema due to selected infectious agents in dogs.

Materials and methods

Study population

The study included 30 dogs presenting with non-traumatic peripheral oedema to the University Veterinary Hospital, Kokkalai and Teaching Veterinary Clinical Complex, Mannuthy from a time period of November 2024 to November 2025.

Clinical and laboratory evaluation

Detailed anamnesis and clinical examination were recorded. Peripheral blood samples were collected for wet film examination, peripheral blood smear evaluation using Field stain, automated haematology, and serum biochemical analysis including blood urea nitrogen, creatinine, liver enzymes, total protein, albumin and globulin. Oedema fluid was aspirated aseptically wherever feasible and subjected to cytological examination and protein estimation.

Imaging studies

Abdominal ultrasonography was performed to evaluate visceral organs and effusions. Electrocardiography and echocardiography were conducted in dogs suspected of cardiac disease.

Molecular diagnosis

Genomic DNA was extracted from whole blood samples using origin DNA genomic extraction kit and subjected to conventional PCR for detection of *Ehrlichia canis, Anaplasma phagocytophilum, Babesia gibsoni, Hepatozoon canis, Brugia malayi and Borrelia burgdorferi* (Table 1). Positive amplicons were sequenced and analysed using NCBI BLAST, and representative sequences were submitted to GenBank.

Treatment

Dogs that tested positive for sheathed microfilaria were treated with Levamisole at a dosage of 10 mg/kg body weight for one week. Dogs diagnosed with *Babesia gibsoni* received a combination of Doxycycline at 10 mg/kg body weight, Clindamycin at 11 mg/kg, Metronidazole at 20 mg/kg for 14 days orally or parenterally depending on their feed intake status respectively (Almendros et al., 2020). For cardiac diseases, treatment was done with ACE inhibitor enalapril at 0.5 mg/kg bodyweight BID, supported with diuretics like frusemide or spironolactone or their combination at 2 mg/kg bodyweight BID orally. Pimobendan was given at 0.25 mg/kg bodyweight BID was added in dilated cardiomyopathy. For hepatic aetiology, hepatoprotectant drugs like S-adenosylmethionine at 20 mg/kg once daily and for renal aetiology, fluid therapy with ringer lactate solution and normal saline were given. Supportive treatment was provided as and when required. Response to the treatment was assessed by resolution of clinical signs and absence of organism in the blood smear at the end of treatment.

Statistical analysis

Haemato-biochemical Data were analysed using SPSS version 24.0 and R programming. One-sample t-test was employed to assess significance.

Table 1. PCR protocol references

Sl. No	Organism	PCR protocol reference	Amplicon size (bp)
1.	*Ehrlichia canis*	Chua et al. (2020)	1250
2.	*Anaplasma phagocytophilum*	Sukanya (2017)	293
3.	*Babesia gibsoni*	Athira (2024)	671
4.	*Hepatozoon canis*	Otranto et al. (2020)	666
5.	*Brugia malayi*	Majitha, (2023)	153
6.	*Borrelia burgdorferi*	Altug et al. (2022)	254

Results and discussion

Clinical signs observed in dogs with peripheral oedema included anorexia, pyrexia, lethargy, unilateral or bilateral limb oedema, ascites, scrotal oedema, lymphadenopathy, dermatitis, and congested mucous membranes (Fig- 1). Wet film examination revealed presence of microfilariae in sixteen out of 30 dogs, while no trypanosomes were detected Peripheral blood smear examination demonstrated *Brugia malayi* microfilariae in seven cases and *Babesia gibsoni* in two cases under oil immersion objective. (Fig 2,3). Wet film and blood smear examination served as rapid and cost-effective screening tools, particularly valuable in endemic regions. Direct visualisation of motile microfilariae on wet film enabled immediate presumptive diagnosis, supporting the continued relevance of this technique in field conditions (Ambily et al., 2011). However, the lower sensitivity of microscopy in cases with low parasitaemia or chronic infection was evident, necessitating confirmatory molecular diagnostics (Rani et al., 2010).

PCR confirmed the presence of *Brugia malayi* in 13 dogs and *Babesia gibsoni* in four dogs (Fig 4,5). None of the samples were positive for *Ehrlichia canis, Anaplasma phagocytophilum, Hepatozoon canis,* or *Borrelia burgdorferi*. There were no mixed infections.

Sequencing of *Brugia malayi* and *Babesia gibsoni* isolates yielded GenBank accession numbers PX891067 and PX894895 respectively. BLAST analysis of isolates revealed 98.67 per cent identity with existing *Brugia malayi* sequences from Kadakkarappally, Alappuzha (Accession number- JN601136) and 100 per cent identity with existing *Babesia gibsoni* sequences from Lanzhou, China (Accession number- OP445697) in Genbank respectively. Polymerase chain reaction (PCR) proved superior in detecting occult infections, supporting previous studies that emphasised its high sensitivity and specificity for canine filarial and other vector borne diseases. (Rishniw et al., 2006; Birkenheuer *et al.*, 2003).

Cytological examination of oedema fluid revealed neutrophils, monocytes, and lymphocytes with protein concentrations ranging from 2.5–7.5 g/dL, consistent with modified transudate. Ultrasonography revealed splenomegaly in four animals, hepatic parenchymal alterations in two cases (Fig 7), renal architectural changes in two animals (Fig 6), and Doppler evidence of vascular engorgement and vasculitis in six animals (Fig 9). Echocardiography identified dilated cardiomyopathy in four dogs (Fig 8) and mitral valve regurgitation in one dog. Statistical analysis of haematological parameters revealed significant increase in total leukocyte count and neutrophil count, while serum biochemical analysis showed significant increase in globulin and AST level (Table-2).

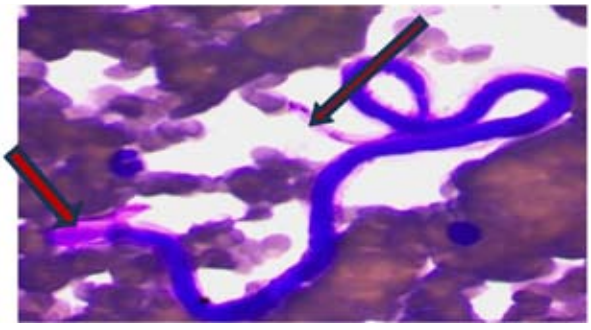

Fig 2. Field staining (100x) of sheathed microfilaria showed pink staining sheath with two discrete overlapping terminal nuclei at tail end

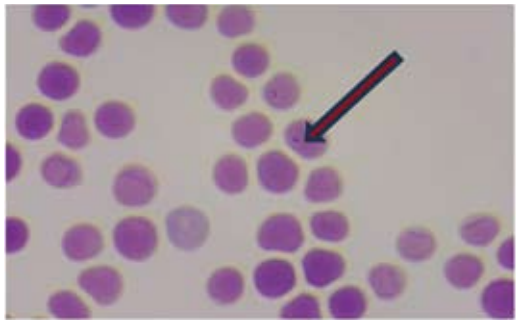

Fig 3. Signet ring shaped ***Babesia gibsoni*** within RBCs of infected dog 100x objective using field stain

Fig 4. Species specific PCR for *Brugia malayi* ; Legends L: 100 bp ladder, 1-5: Positive Samples, P: Positive control N-Negative control

Fig 5. Species specific PCR for *Babesia gibsoni* : Legends L: 100 bp ladder, 1-4: Positive Sample, P: Positive control, N: Negative control

Table 2. Statistical analysis of Haemato- biochemical parameters of 30 dogs with peripheral oedema(n=30)

Variable	Maximum	Minimum	Mean±SE	Normal range	t- value (P value) for comparison with	
					Lower limit	Upper limit
WBC (10^3 /µl)	41.06	8.69	20.53±1.56	06 - 17	9.33**	2.27*
NEU (103 /µl)	36.64	5.63	16.81±1.46	3.62- 11.32	9.03**	3.71**
AST(IU/L)	84.16	18.39	35.87±2.71	13-15	8.42**	7.69**
GLO(g/dl)	8.12	2.07	5.34±0.24	2.7-4.4	10.99**	3.92**

** significant at 1 percent level * significant at 5 percent level

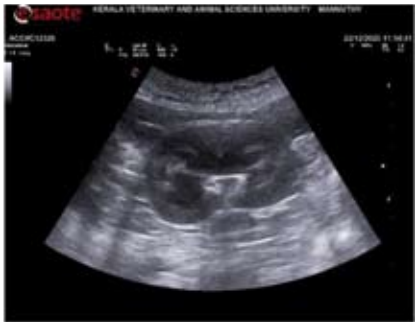

Fig 6. Renal architecture with poor cortico-medullary differentiation in a dog with chronic kidney disease

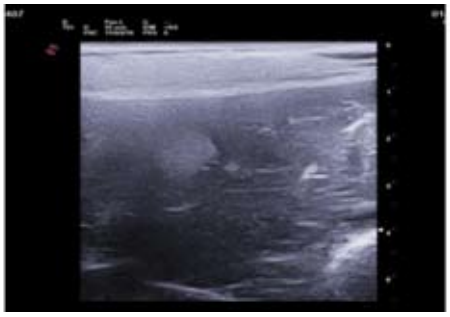

Fig 7. Mixed echogenicity of liver observed in a dog with hepatitis

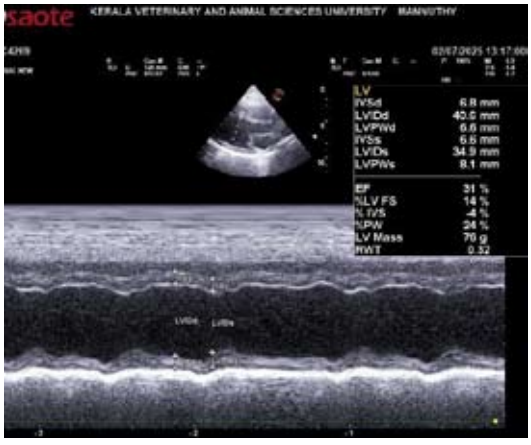

Fig 8. M mode echocardiogram (Right parasternal view) of a dog with peripheral oedema due to DCM

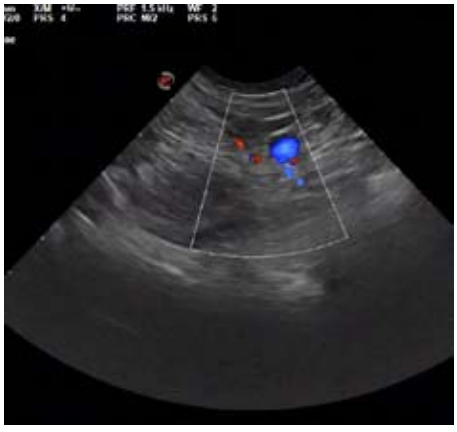

Fig 9. Transverse view of limb oedema; Engorgement of blood vessels on Doppler ultrasonography in a vasculitis affected dog

The present study demonstrated that peripheral oedema in dogs is a multifactorial condition arising from both infectious and non-infectious aetiologies, ultimately converging on common pathophysiological mechanisms involving inflammation, vascular permeability and lymphatic dysfunction. The predominance of bilateral and dependent limb oedema observed in this study supports earlier observations that such distribution patterns are typically associated with systemic disease rather than localised pathology (Whelchel et al., 2023).

Among infectious causes, *Brugia malayi* was the most prevalent agent associated with peripheral oedema in the present study. This finding agrees with reports from filariasis-endemic regions of India, where canine filarial infections are commonly linked to lymphatic obstruction, chronic lymphangitis and inflammatory vascular changes (Ravindran et al., 2014; Rani et al., 2010). The occurrence of splenomegaly and hyperglobulinaemia in *B. malayi* positive dogs reflects prolonged antigenic stimulation and reticuloendothelial activation, as previously documented in chronic filarial infections (Nelson & Couto, 2019).

Babesia gibsoni infection was another significant infectious contributor. PCR-confirmed cases showed anaemia and thrombocytopenia, consistent with haemolysis and immune-mediated platelet destruction reported in chronic babesiosis (Birkenheuer et al., 2003; Qurollo et al., 2019). These haematological alterations, together with systemic inflammation, likely predispose affected dogs to endothelial injury and increased capillary permeability, thereby facilitating peripheral oedema formation.

Cardiac diseases constituted an important non-infectious cause, with dilated cardiomyopathy being the

predominant diagnosis. Echocardiographic evidence of ventricular dilatation and atrial enlargement explains dependent oedema through venous congestion and impaired lymphatic drainage (Ware, 2007; Atkins et al., 2009). Electrocardiographic findings such as atrial fibrillation and supraventricular tachycardia further indicated advanced myocardial disease and atrial remodelling (Tilley & Smith, 2015). Loss of renal architexture in chronic kidney disease caused protein loss and sodium retention, leading to oedema formation. (Polzin, 2011). The hypoalbuminemia due to hepatic dysfunction, reduced plasma oncotic pressure, predisposing dogs to peripheral oedema. Laboratory evaluation of liver enzymes and serum proteins was considered essential in such cases. (Hall and German, 2005)

Overall, the findings confirm that peripheral oedema in dogs represents a common clinical manifestation of diverse underlying disorders, reinforcing the need for an integrated diagnostic approach combining clinical evaluation, haemato-biochemical analysis, imaging and molecular diagnostics. In chronic or recurrent cases, especially those associated with persistent vasculitis or lymphatic dysfunction, oedema may recur despite organism clearance, indicating that long-term management rather than permanent cure may be required.

Conclusion

The study concludes that *Brugia malayi* and *Babesia gibsoni*, cardiac diseases play a significant role in the aetiopathology of peripheral oedema in dogs in the study population. Peripheral oedema is a multifactorial clinical manifestation rather than a standalone disease entity. Accurate identification of infectious aetiologies using molecular diagnostics, combined with comprehensive clinico-pathological evaluation, is essential for effective management and improved clinical outcomes. The development of a therapeutic protocol focused on managing vasculitis-associated sequelae is crucial for effective treatment and prevention of recurrent peripheral oedema.

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