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Diagnosis and therapeutic management of *Babesia gibsoni*-induced paraplegia in dogs

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Abstract

Two dogs presented to VCC, PGIVAS, Akola, with an inability to walk as the primary complaint, accompanied by fever, inappetence for one week and a history of 2-3 episodes of vomiting. Clinical examination revealed mild to high rectal temperatures (102.5-104 °F), swollen lymph nodes, congested mucous membranes, prolonged capillary refill time, tachycardia, polypnea, paraplegia, and mild tick infestation. Hematobiochemical analysis showed thrombocytopenia, neutrophilic leukocytosis, elevated BUN, borderline hypoproteinemia, and increased SGOT, while other parameters were within normal limits. Diagnosis was established based on blood smear, immunochromatographic test (ICT), and confirmed by PCR. Both dogs were treated with artesunate and azithromycin, along with supportive therapy including neurotropic vitamins, methylprednisolone, proton pump inhibitors, and polyherbal liver and platelet stimulants. Both dogs demonstrated uneventful recovery by day 10th and 30th post-treatment, with normalization of clinical and hematobiochemical parameters, and parasitological testing confirmed complete clearance of the infection by day 30th post-treatment.

Keywords: Paraplegia, artesunate, thrombocytopenia, neurotropic vitamin, methylprednisolone etc.

1. Introduction

Canine *Babesia* species are intraerythrocytic protozoan parasites classified under the order Piroplasmida of the phylum Apicomplexa (Schnittger *et al.*, 2012; Koster *et al.*, 2015)^[16, 13]. They are morphologically categorized into large forms (*Babesia canis*), and small forms (*Babesia gibsoni*). Transmission occurs primarily through ixodid ticks, with *Haemaphysalis* spp. recognized as the principal vectors of *B. gibsoni* in dogs (Irwin, 2009; Jongejan *et al.*, 2018)^[9, 11]. In India, *B. canis* and *B. gibsoni* are the predominant causative agents of canine babesiosis (Jain *et al.*, 2017; Baneth, 2018)^[10, 1].

The clinical manifestations of canine babesiosis vary from mild to severe systemic illness. Common signs include anorexia, pyrexia, vomiting, pallor of mucous membranes, tachycardia, and polypnea. However, atypical and severe complications such as ascites and neurological deficits, including paraplegia, have also been reported, indicating the potential for multisystem involvement (Gonde *et al.*, 2016)^[5]. Neurological manifestations, although uncommon, are clinically significant and may arise due to hypoxia, vascular compromise, or immune-mediated mechanisms affecting the central nervous system.

Accurate diagnosis of babesiosis involves a combination of clinical evaluation and laboratory techniques, including microscopic examination of blood smears, complete blood count analysis, serological testing such as the SNAP 4Dx test, and molecular methods like polymerase chain reaction (PCR) (Davikto *et al.*, 2015)^[6]. Additionally, immunochromatographic tests utilizing recombinant antigens have been developed for rapid and specific detection of *B. gibsoni* antibodies (Verdida *et al.*, 2005)^[20].

The conventional treatment of canine babesiosis involves antiprotozoal agents such as imidocarb dipropionate and diminazene aceturate; however, these drugs often exhibit limited efficacy against *Babesia gibsoni*, highlighting the need for alternative therapies (Teodorowski *et al.*, 2022)^[18].

Artesunate, a potent semisynthetic derivative of artemisinin, has shown promise due to its antiparasitic activity, which includes the generation of reactive free radicals, inhibition of heme polymerization, and disruption of parasite membrane transport, ultimately leading to parasite death. Studies have reported that oral administration of artesunate at 12.5 mg/kg once daily for 10 days is effective in treating *B. gibsoni* infection in dogs (Goo *et al.*, 2010; Karasová *et al.*, 2022; and Bhasme *et al.*, 2024)^[7, 18, 2].

2. Materials and Methods

Two cases, from different time periods, were examined, and both dogs exhibited similar clinical histories, including lethargy, acute paraplegia, fever, inappetence, and 2–3 episodes of vomiting, upon presentation at VCC, PGIVAS, Akola. Clinical examination revealed congested CMM, tick infestation and swollen lymph nodes, Ashok as shown in Figures 1 and 2.



Fig 1: Congested CMM



Fig 2: Tick infestation

Clinico-physiological parameters were systematically recorded on day 0 (pre-treatment) and subsequently on the 10th and 30th days post-treatment. These included rectal temperature (°F), respiratory rate (breaths per minute), heart rate (beats per minute), capillary mucous membrane (CMM) status, capillary refill time (CRT, seconds), and body weight (kg). Blood samples were collected for diagnosis, and haemato-biochemical investigations were performed on day 0 (pre-

treatment) and on the 10th and 30th days post-treatment. The parameters evaluated included haemoglobin (Hb, g/dl), total erythrocyte count (TEC, $\times 10^6$ /cu.mm), packed cell volume (PCV, %), platelet count ($\times 10^5$ / μ l), total leukocyte count (TLC, $\times 10^3$ /cu.mm), and absolute neutrophil count ($\times 10^3$ /cu.mm). Biochemical parameters included total protein (g/dl), liver enzymes ALT/SGPT (IU/L) and AST/SGOT (IU/L), renal markers included blood urea nitrogen (BUN, mg/dl) and creatinine (mg/dl), and the iron metabolism indicator total iron-binding capacity (TIBC, μ g/dl). Diagnosis of *Babesia gibsoni* infection had been carried out using blood smear examination and the antibody rapid test (ICT), which were performed on day 0 (pre-treatment) and on the 10th and 30th days post-treatment for direct parasite detection and serological screening, while PCR targeting the cox3 gene was conducted on day 0 (pre-treatment) and day 30th (post-treatment) to confirm infection and monitor parasite clearance.

6. Diagnosis

The diagnosis of the infection was carried out using blood smear examination, the rapid antibody test (ICT), and PCR. The results are summarized in Table 1 and illustrated in Figures 3-5.

Table 1: Diagnostic parameters of Dogs I and II

Diagnostic parameter	DOG I	DOG II
Blood smear	Positive	Positive
The <i>B. gibsoni</i> Antibody Rapid Test (ICT)	Positive	Negative
PCR	Positive	Positive

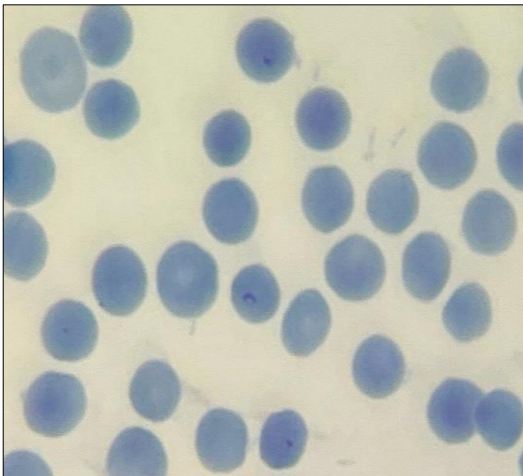


Fig 3: *B. Gibsoni* Trophozoites in RBCs



Fig 4: Positive *B. gibsoni* Antibody Rapid Test.

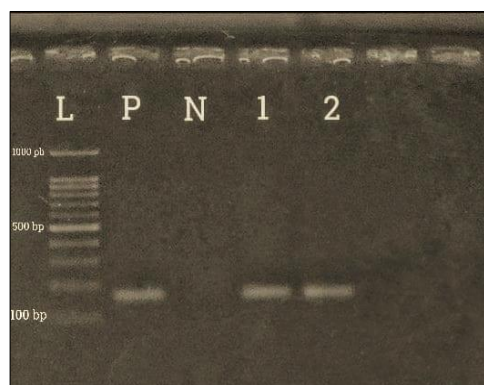


Fig 5: PCR amplification of *B. gibsoni* cytochrome c oxidase subunit III (cox3) gene (164 bp) using Bgx3 primers before treatment. L: 100 bp DNA ladder; P: Positive control; N: Negative control; Lanes 1-2: Positive samples.

6. Treatment

Both dogs were treated with artesunate at a dose of 12.5 mg/kg orally once daily and azithromycin at 10 mg/kg orally once daily for 10 days. In addition to specific therapy, supportive treatment was provided on the day of admission, which included antipyretics (inj. meloxicam @ 0.2 mg/kg, IM) and antiemetics (inj. Ondansetron @ 0.2 mg/kg, IM). Neurotropic vitamin therapy containing mecobalamin (1500 µg/dog, IM)

and inj. Methylprednisolone acetate (120 mg/dog, IM) was administered on days 0, 3, and 7 to support neural repair and to reduce neuropathic pain and inflammation associated with *Babesia gibsoni*-induced paraplegia. Polyherbal liver and platelet stimulants were also administered orally to manage systemic complications and support recovery.

Owners of both the dogs were advised to present the patients for follow-up examinations on the 10th and 30th days post-treatment. Additionally, owners were also instructed to assist their dogs with physical activity like walking with weight-bearing support during the early stages of recovery to ensure safe ambulation and to prevent injury.

7. Results and Discussion

Canine babesiosis commonly presents with clinical signs such as fever, lethargy, anorexia, and enlargement of lymph nodes and the spleen (Boozar and Macintire, 2003)^[3]. Occasionally, it has also been reported to manifest as paraplegia (Gonde, 2016)^[5].

The clinical parameters of both dogs over the treatment period are summarized in table 2. The elevated body temperature in the dogs was caused by infection-induced inflammation and vascular damage (Zygner *et al.*, 2023)^[21], and he also stated that babesiosis can cause hemodynamic instability, hypovolemia, and microvascular obstruction due to red blood cell sequestration which causes prolonged CRT.

Table 2: Clinical parameters of Dogs I and II before and after treatment

Parameters	Reference range	Pre-treatment		Post-treatment			
		Day 0		10 th day		30 th day	
		Dog I	Dog II	Dog I	Dog II	Dog I	Dog II
Rectal Temp. (°F)	101.0 -102.5	103.5	102.9	101.9	101.8	101.3	101.4
Respiratory Rate (/min)	18- 34	20	30	24	26	26	28
Heart Rate (beats/min.)	70-120	130	90	112	108	108	88
CMM	-	Cong.	Cong.	Pink	Pink	pink	Pink
CRT (seconds)	1-2	2.5	1.5	2	1	1.5	1
Body Weight (kg)	-	50	32	50.6	32	53	33.5

Hematobiochemical parameters of the dogs are presented in table 2. Thrombocytopenia, the most common abnormality in babesiosis (Chan *et al.*, 2025)^[4], is caused by splenic sequestration and immune-mediated platelet destruction. Leukocytosis in naturally infected babesiosis cases is often associated with neutrophilia and secondary bacterial infections (Gurjar *et al.*, 2023 and Sivajothi *et al.*, 2014)^[8, 17]. Neutrophilia serves as a primary defense mechanism against piroplasm infection (Mittal *et al.*, 2019)^[15]. The increase in

AST without a marked ALT rise suggests an extra-hepatic origin (Thomas, 2000; Koseoglu *et al.*, 2011)^[19, 12]. The finding of normal serum creatinine with elevated BUN indicated pre-renal azotemia; an increased urea-to-creatinine ratio with normal creatinine is typically of non-renal origin (Lobetti *et al.*, 2012)^[14].

The clinical and hematobiochemical improvements observed indicate successful recovery following treatment.

Table 3: Hematobiochemical parameters of Dogs I and II before and after treatment

Parameters	Reference range	Pre-treatment		Post-treatment			
		Day 0		10 th day		30 th day	
		Dog I	Dog II	Dog I	Dog II	Dog I	Dog II
Hb (g/dL)	12-16	10.4	15.7	11.2	14.2	13.9	15.4
TEC ($\times 10^6$ /cu.mm)	5.5 - 8.5	5.35	7.76	5.47	6.58	6.77	6.9
PCV (%)	37-55	32.42	44.49	35.4	44.95	45.98	43.8
Platelet (10^5 /µl)	2.00 - 5.00	1.56	1.39	2.49	2.39	2.39	2.98
TLC ($\times 10^3$ /cu.mm)	6.0 -16.0	19.78	20.99	15.08	8.35	13.75	10.5
AbNC ($\times 10^3$ /cu.mm)	3-12	17.60	18.68	12	5.53	11.24	7
Total Protein (g/dl)	5.4 - 7.5	5.2	4.8	5.8	5.4	6.2	5.6
ALT/SGPT (IU/L)	10 - 100	58	45	46	27	35	30
AST/SGOT (IU/L)	23 -55	115	85	48	56	32	28
BUN (mg/dl)	7 - 27	46	68	29	36	32	28
Creatinine (mg/dl)	0.5 - 1.5	1.2	1.3	0.9	1.06	0.7	0.86
TIBC (µg/dl)	280 - 400	313	258	342	280	298	261

Diagnostic evaluations over the treatment period are summarized in Table 3. On day 0, both dogs tested positive by blood smear, the *B. gibsoni* antibody rapid test (ICT), and PCR, confirming active infection. By day 10 post-treatment, blood smears were negative in both dogs, while ICT was negative in

Dog I and remained negative in Dog II. By day 30, all diagnostic tests including blood smear, ICT, and PCR were negative in both dogs, indicating effective parasite clearance as shown in Table 4.

Table 4: Diagnostic evaluation of Dogs I and II before and after treatment

Diagnostic parameter	Pre-treatment		Post-treatment			
	Day 0		10 th day		30 th day	
	Dog I	Dog II	Dog I	Dog II	Dog I	Dog II
Blood smear	+ve	+ve	-ve	ve	- ve	- ve
The <i>B. gibsoni</i> Antibody Rapid Test (ICT)	+ve	- ve	-ve	- ve	- ve	- ve
PCR	+ve	+ve	-	-	- ve	- ve

Overall, the combined clinical, hematobiochemical, and parasitological findings demonstrate that the treatment regimen successfully cleared the infection and facilitated complete recovery in both dogs, including resolution of paraplegia shown in figures 6 and 7. These observations are consistent with previous studies on effective management of *B. gibsoni* infection (Bhasme *et al.*, 2024) [2].



Fig 6: Paraplegic dog before treatment



Fig 7: Recovered dog after treatment

8. Conclusion

Accurate diagnosis guided timely and targeted treatment of *Babesia gibsoni*-induced paraplegia, and combined therapy with artesunate, azithromycin, and supportive treatments including neurotropic vitamins, methylprednisolone, and polyherbal liver and platelet stimulants effectively cleared the infection and promoted complete clinical, hematological, biochemical, and neurological recovery

9. Conflict of Interest

The authors declare that they have no conflicts of interest.

10. Acknowledgment

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