

Clinico-pathological and Ultrasonographical Studies in *Babesia gibsoni* Infection in Dogs

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Abstract

The present study was carried out to investigate the prevalence, clinical signs, haematological, biochemical and ultrasonographic alterations in dogs naturally infected with *Babesia gibsoni* at the Medicine unit, Veterinary Clinical Complex, College of Veterinary and Animal Science, Bikaner, Rajasthan from July 2025 to December 2025. Out of 1628 dogs examined in the Medicine OPD, 23 were confirmed positive for *B. gibsoni* by PCR (18S rRNA gene), yielding an overall hospital prevalence of 1.41%. The highest occurrence was recorded in German Shepherds (30.4%), male dogs (56.5%) and the >1–5-year age group (34.8%). Maximum positivity was observed during July (39.1%). The predominant clinical signs included pyrexia (73.9%), anorexia (69.6%) and tick infestation (65.2%). Ultrasonography revealed splenomegaly (52.3%), hepatomegaly (39.1%) and renal cortical changes (17.4%). Haematological examination disclosed highly significant ($p < 0.01$) reductions in haemoglobin (8.56 ± 0.79 g/dl), total erythrocyte count ($4.45 \pm 0.28 \times 10^6/\mu\text{l}$), PCV ($28.09 \pm 1.98\%$) and platelets ($82.52 \pm 11.64 \times 10^3/\mu\text{l}$). Biochemical analysis revealed significant elevations in AST, ALT and total bilirubin, along with significant decreases in total protein and albumin.

Keywords: *Babesia gibsoni*, Prevalence, PCR, Haemato-biochemical, Ultrasonography, Dogs

Introduction

Babesiosis attributable to *B. gibsoni* follows a comparatively chronic and less fulminant course than disease caused by *B. canis*, although severe multi-organ involvement is well documented (Irwin, 2010). Despite increasing reports from other Indian states, epidemiological and clinico-pathological data specific to the Bikaner region of Rajasthan remain scarce (Bhati, 2025). The present study was done to determine the hospital-based prevalence of *B. gibsoni* and to characterise the associated clinical, haemato-biochemical and ultrasonographic alterations in dogs with Babesiosis.

Materials and Methods

The investigation was conducted at the Medicine unit, Veterinary Clinical Complex, College of Veterinary and Animal Science, Bikaner from July 2025 to December 2025. A total of 1628 dogs of varied breeds, ages and both sexes presented to the Medicine OPD underwent thorough physical and clinical examination. Animals exhibiting pyrexia, history or presence of tick infestation, anaemia, icterus or other signs suggestive of haemoprotozoan infection were subjected to detailed clinical, haemato-biochemical and

ultrasonographic examination. Signet-ring, pyriform and comma-shaped organisms appearing singly or in pairs within erythrocytes were recorded as positive findings. The Ubio Quickvet immunochromatographic rapid test kit (Ag-Ab based) was employed for all 110 suspected cases as per Eljadar (2010). Genomic DNA was extracted from 200 μl of EDTA-anticoagulated blood using a standard commercial DNA extraction kit. PCR amplification targeting a 670 bp fragment of the 18S rRNA gene of *B. gibsoni* was performed with species-specific primers and the protocol described by Mahalingaiah *et al.* (2017) with minor modifications to annealing temperature. Amplicons were visualised under UV light after electrophoresis on 1.5% agarose gel stained with ethidium bromide.

Results and Discussion

Of 1628 dogs examined in the Medicine OPD during the study period, 110 were identified as suspects for *B. gibsoni* infection. PCR confirmed 23 positive cases, yielding an overall hospital-based prevalence of 1.41% (23/1628). Among the 110 suspected cases, microscopy detected 9 (8.18%), the rapid test kit detected 7 (6.36%) positives, while PCR identified 23 (20.9%) cases, confirming its superiority as a diagnostic modality. The PCR prevalence of 1.41% closely agreed with Gonmei *et al.* (2020), who recorded 1.25% in Mizoram, and with Kushwaha *et al.* (2018), who reported 1.82% hospital-

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based PCR positivity. Variation in prevalence across different studies may be attributed to differences in agro-climatic conditions, diagnostic techniques employed, tick vector density and host immunity as reported by Baneth (2018). In contrast, significantly higher prevalences of 34.39% and 47.05% have been reported from Tamil Nadu by Sahithi *et al.* (2024) and Soundarya *et al.* (2023), respectively, likely reflecting differences in tick pressure and climate.

Age-wise, the highest positivity was recorded in the >1–5 year group (34.8%), followed by the >5–10 year group (30.4%), while dogs up to 1 year and those above 10 years each contributed 17.4%. This distribution is comparable to Aarthi (2017), who noted peak infections in the 1–5 year cohort (71.68%), and to Chaurasia *et al.* (2022), who documented highest prevalence in dogs aged 1–3 years (41.18%), reflecting greater outdoor activity and tick exposure in younger, more active dogs. Breed-wise, German Shepherds predominated (30.4%), followed by Labradors (26.1%). This aligns with findings of Chaurasia *et al.* (2022), who attributed genetic susceptibility and carrier status in German Shepherds to sustained seropositivity. Gender-wise, males comprised 56.5% of positive cases compared to 43.5% in females, which is consistent with Mahalingaiah *et al.* (2017), and Soundarya *et al.* (2023), who attributed higher male susceptibility to roaming behaviour and greater tick vector exposure.

Seasonally, the rainy months of July–September accounted for 73.8% of all cases, with July alone contributing 39.1%. This pattern is concordant with Bhati (2025) and Chaurasia *et al.* (2022), attributing peak incidence to increased activity of *Rhipicephalus sanguineus* under warm, humid conditions.

Pyrexia (>102.5°F) was the most frequent systemic finding, recorded in 73.9% (17/23) of cases, with a mean body temperature of $103.7 \pm 0.4^\circ\text{F}$. Anorexia was documented in 69.6% (16/23), followed by tick infestation or tick history in 65.2% (15/23) and lethargy in 47.8% (11/23). Pale mucous membranes indicative of anaemia was present in 34.8% of cases. These findings are in close agreement with Hajare (2023) and Bhati (2025), who reported similar predominance of fever, inappetence and tick infestation. Gastrointestinal manifestations such as vomiting (26.1%), melena (30.4%) and diarrhoea (4.3%) were recorded. Gonde *et al.* (2014) similarly documented vomiting in 27.02% and melena in 2.70% of naturally occurring babesiosis cases. Petechial haemorrhages and limb oedema

were each observed in 17.4% of dogs, consistent with thrombocytopenic manifestations. Jaundice, corneal opacity and emaciation were less frequently encountered (8.7% each), while haemoglobinuria, epistaxis, seizures and ascites were noted in solitary cases (4.3%). Chan *et al.* (2025) also recorded jaundice in only 7% of cases and attributed haemoglobinuria to the rapid intravascular haemolysis more characteristic of *B. rossi* than *B. gibsoni*. Atypical signs such as seizures and corneal opacity, observed previously by Shinde *et al.* (2021) in 3.22% and 6.45% of cases respectively, were attributed to cerebral babesiosis arising from sequestration of parasitised erythrocytes in CNS microvasculature.

Splenomegaly was the most common finding, present in 52.3% (12/23) of cases, observed as diffuse hypoechoic parenchymal pattern with enlargement, consistent with observations of Yogeshpriya *et al.* (2018). Moth-eaten echotexture indicative of multifocal splenic necrosis was detected in 8.7% of animals, suggesting vascular compromise with venous outflow obstruction (Raut, 2014). Hepatomegaly was identified in 39.1% (9/23) of dogs, with hyperechoic parenchyma noted in 4 animals correlating with elevated ALT and ALP values, consistent with the findings of Fraga *et al.* (2011). Gallbladder distension was recorded in 21.7% (5/23) of cases and gallbladder sludge or wall oedema in 13%, with Sarma *et al.* (2014) suggesting that distension beyond normal range is a reliable indicator of anorexia in companion animals. Renal ultrasonographic abnormalities included loss of corticomedullary distinction (17.4%), increased cortical echogenicity (13%) and renal pelvis dilation (pyelectasia) in 8.7% of dogs. These findings are consistent with increased renal echogenicity and altered corticomedullary ratio characteristic of *B. gibsoni*-associated renal injury, as previously described by Eduardo *et al.* (2011) and Yogeshpriya *et al.* (2019). Ascitic free fluid was identified in one dog (4.3%), consistent with observations of Kaur (2019) who reported ascites in 14.3% of affected animals.

Haematobiochemical values are summarized in Table 2. The mean \pm SE values of haemoglobin (8.56 ± 0.79 g/dl), TEC ($4.45 \pm 0.28 \times 10^6/\mu\text{l}$) and PCV ($28.09 \pm 1.98\%$) in infected dogs were highly significantly ($p < 0.01$) reduced compared to healthy controls (15.16 ± 0.61 g/dl; $6.98 \pm 0.35 \times 10^6/\mu\text{l}$; $44.56 \pm 1.94\%$), indicative of severe regenerative anaemia. Similar findings were documented by Yogeshpriya *et al.* (2018), Bhati (2025) and Chan *et al.* (2025). Platelet count was highly

significantly ($p < 0.01$) decreased in infected animals ($82.52 \pm 11.64 \times 10^3/\mu\text{l}$) compared to controls ($346.13 \pm 31.92 \times 10^3/\mu\text{l}$), indicative of severe thrombocytopenia, in agreement with Chan *et al.* (2025).

AST (82.89 ± 11.77 IU/L) and ALT (105.37 ± 13.16 IU/L) were highly significantly ($p < 0.01$) elevated compared to healthy controls (30.39 ± 3.16 and 36.72 ± 6.86 IU/L, respectively). Total protein (5.52 ± 0.12 g/

dl) and albumin (2.40 ± 0.21 g/dl) were significantly ($p < 0.05$) reduced in infected dogs, comparable to healthy values of 6.11 ± 0.22 and 2.99 ± 0.16 g/dl respectively, indicating hypoproteinaemia and hypoalbuminaemia. Blood urea nitrogen (35.63 ± 16.98 mg/dl) and creatinine (2.22 ± 0.75 mg/dl) were non-significantly increased relative to healthy controls (15.79 ± 1.38 and 1.02 ± 0.09 mg/dl).

Table 1: Haemato-biochemical values of *Babesia gibsoni* infected dogs (Mean \pm SE)

Parameters	Healthy Dogs (n=8)	<i>B. gibsoni</i> Infected (n=23)
Hb (g/dl)	15.16 ± 0.61	$8.56^{**} \pm 0.79$
TEC ($\times 10^6/\mu\text{l}$)	6.98 ± 0.35	$4.45^{**} \pm 0.28$
PCV (%)	44.56 ± 1.94	$28.09^{**} \pm 1.98$
Platelets ($\times 10^3/\mu\text{l}$)	346.13 ± 31.92	$82.52^{**} \pm 11.64$
WBC ($\times 10^3/\mu\text{l}$)	10.39 ± 0.92	18.01 ± 4.56
MCHC (g/dl)	33.45 ± 0.53	$29.99^* \pm 1.53$
Neutrophils (%)	71.90 ± 2.25	76.27 ± 2.32
Lymphocytes (%)	21.88 ± 2.04	19.15 ± 2.09
AST (IU/L)	30.39 ± 3.16	$82.89^{**} \pm 11.77$
ALT (IU/L)	36.72 ± 6.86	$105.37^{**} \pm 13.16$
ALP (IU/L)	124.69 ± 16.33	173.20 ± 39.87
Total Protein (g/dl)	6.11 ± 0.22	$5.52^* \pm 0.12$
Albumin (g/dl)	2.99 ± 0.16	$2.40^* \pm 0.21$
Globulin (g/dl)	3.0 ± 0.23	3.12 ± 0.21
Total Bilirubin (mg/dl)	0.32 ± 0.06	$1.19^{**} \pm 0.17$
BUN (mg/dl)	15.79 ± 1.38	35.63 ± 16.98
Creatinine (mg/dl)	1.02 ± 0.09	2.22 ± 0.75

* $p < 0.05$ – significant; ** $p < 0.01$ – highly significant; no superscript – non-significant

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