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Cardiorespiratory changes in babesiosis infected sheep: A case report

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Abstract
A 1.5-year-old Bannur sheep, weighing 22 kg, presented to Veterinary College Hospital, Bengaluru, with clinical signs of high fever, anorexia, respiratory distress, blood-mixed urination, and a history of tick infestation. Clinical observations revealed dullness, pale mucous membrane, tick infestation, and hemoglobinuria. Physical examination indicated elevated temperature, heart rate, pulse rate and absence of rumeno-reticular motility. Auscultation revealed muffled heart sounds and respiratory crackles. Blood smear confirmed intraerythrocytic *Babesia* piroplasm. Thoracic radiography revealed pulmonary edema, congestion, and pleural effusions. Echocardiography disclosed mild to moderate pericardial effusion with an increased LA/Ao ratio. Hematology showed reduced RBCs, hemoglobin, platelets, and PCV, while WBC count increased. Serum biochemistry indicated elevated CK-MB, creatinine, BUN, ALT, AST, and total bilirubin with decreased total protein and albumin. Combined findings of thoracic radiography elevated CK-MB and echocardiography suggest significant cardiac and pulmonary involvement in this babesiosis case in Bannur sheep.

Keywords: Bannur sheep, *Babesia*, tick infestation, pulmonary edema and pericardial effusion

Introduction
Babesiosis, a disease affecting both domestic and wild animals in tropical and sub-tropical regions, is caused by protozoa from the genus *Babesia* within the family Babesidae, belonging to the Piroplasmid class (Alessandra and Santo, 2012) [1]. Babesiosis is caused by *B. ovis, B. motasi,* and *B. crassa*. Among these *Babesia ovis*, a small *Babesia* species (with a size of < 2.5 µm) is the most prevalent and pathogenic, responsible for causing sheep babesiosis (Esmaeilnejad *et al.*, 2015) [4]. The life cycle of *Babesia* spp. involves a dual-host system, with sexual reproduction taking place in ticks and asexual reproduction occurring within vertebrate erythrocytes (Chauvin *et al.*, 2009) [2]. The Babesia parasite multiplies inside red blood cells, causing damage during its exit, leading to widespread intravascular hemolysis. This can progress to low oxygen levels (hypoxemia), with changes in the red blood cell membrane reducing flexibility and increasing adhesion to vessel walls. This adherence may obstruct blood flow in the pulmonary vasculature, potentially causing non-cardiogenic pulmonary edema and, ultimately, acute respiratory distress syndrome (ARDS) (Krause *et al.*, 2007) [8]. Babesia infection can damage the heart, causing inflammation, myocardial infarction, hemorrhage, and necrosis, leading to circulatory disorders and contributing to the cause of death in infected animals (Yang *et al.*, 2022) [17]. In babesiosis, various symptoms were observed, including anorexia, interrupted rumination, paleness of the mucous membranes, fever (pyrexia), bilateral nasal discharge, rapid heart rate (tachycardia), accelerated breathing (tachypnoea), and the presence of hemoglobinuria (Venkatesakumar *et al.*, 2018) [15]. The current communication discusses about the involvement of cardiorespiratory changes in babesiosis infected sheep.

Case history
A 1.5-year-old Bannur sheep weighing 22 kg was presented to the Department of Veterinary Medicine, Veterinary College, Bengaluru, exhibiting symptoms such as high fever, anorexia, weakness, respiratory distress, blood-mixed urination, and tick infestation. The onset of these

symptoms occurred two days before the sheep's arrival at the hospital, as reported in the anamnesis. The owner mentioned having 20 additional sheep, none of them displayed similar issues, although ticks were present in the flock.

Clinical findings

Clinical findings of the animal were as follows: a temperature of 106.8°F, respiratory rate of 38/min, heart rate of 98/min, pale conjunctival mucous membrane, swollen prescapular lymph nodes and the absence of rumeno-reticular contractions. Auscultation of heart revealed muffled heart sounds and auscultation of lungs showed respiratory crackles. Tests on fecal samples were negative for parasitic eggs, and Giemsa-stained blood smears was found positive for Babesia piroplasm in the erythrocytes.

The hematological, serum biochemical, and echocardiographic parameter values are presented in Tables 1, 2, and 3, respectively. Thoracic radiography indicated pulmonary edema, congestion, and pleural effusions in sheep infected with babesiosis (Fig. 3).

Table 1: Hematological parameters in babesiosis affected sheep

Haematological Parameter	Findings	Reference range (Constable *et al.*, 2017) [3]
Haemoglobin (g/dL)	7.3	9.0 - 15.0
TEC (× 10^6/µL)	7.94	9.0 - 15.0
TLC (×10^3/µL)	14.2	4 - 12
Thrombocytes (× 10^3/µL)	366	800 - 1100
PCV (%)	22.8	27 - 45

Table 2: Serum biochemical parameters in babesiosis affected sheep

Serum biochemistry	Findings	Reference range (Constable *et al.*, 2017) [3]
Total protein (g/dL)	5.8	6.0 – 7.9
Albumin (g/dL)	2.1	2.4 – 3.0
Globulin (g/dL)	1.7	1.7 -2.6
Total bilirubin (mg/dL)	0.8	0.1 – 0.5
Alanine transaminase (IU/L)	38.4	5 - 20
Aspartate aminotransferase (IU/L)	128.3	60 - 280
Gamma glutamate transferase (IU/L)	61.8	20 - 52
Creatinine (mg/dL)	1.7	1.2 – 1.9
Blood urea nitrogen (mg/dL)	22.3	8.0 - 20
Creatinine kinase MB	216.7	-

Table 3: Echocardiographic parameters in babesiosis affected sheep

Echocardiographic parameter	Value	Echocardiographic parameter	Value
RVIDd (cm)	0.78	LVPWd. (cm)	1.27
RVIDs (cm)	0.39	LVPWs (cm)	1.09
LVIDd (cm)	2.66	EDV (ml)	26.02
LVIDs (cm)	1.75	ESV (ml)	9.07
FS (%)	34.09	SV (ml)	16.94
IVSd (cm)	0.97	EF (%)	65.12
IVSs (cm)	0.91	EPSS (cm)	0.64
LA (cm)	2.12	Ao (cm)	1.24
LA/Ao ratio	1.71	Pericardial effusion	Mild

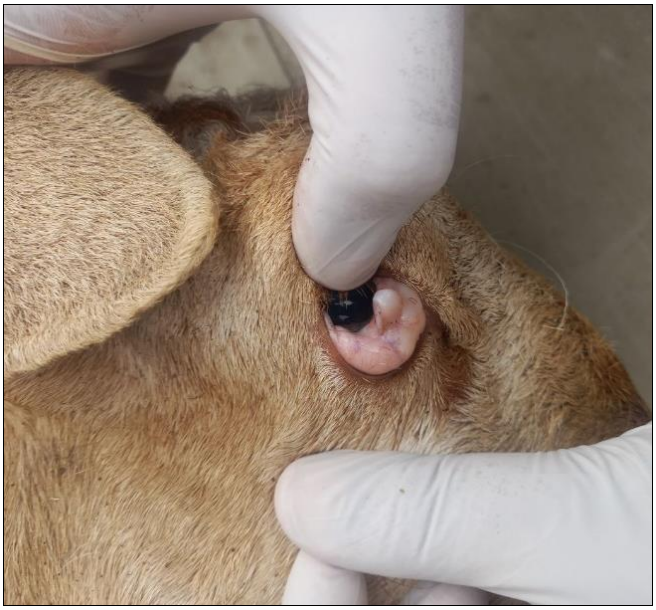

Fig 1: Pale conjunctival mucous membrane

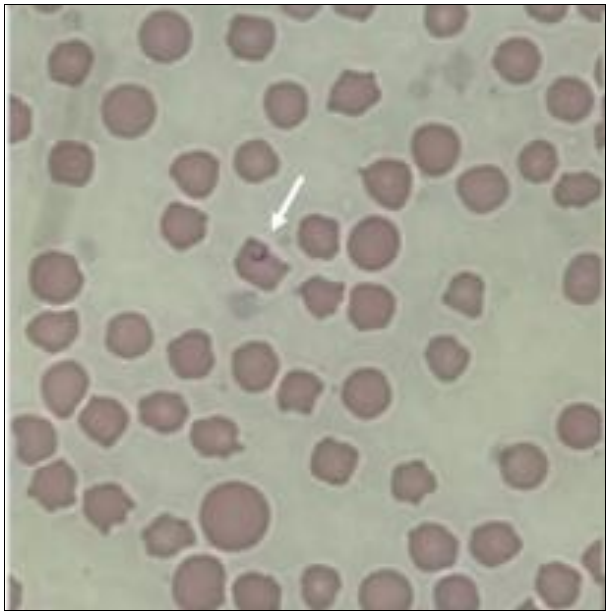

Fig 2: Piroplasm of *Babesia* Spp. Giemsa 100X

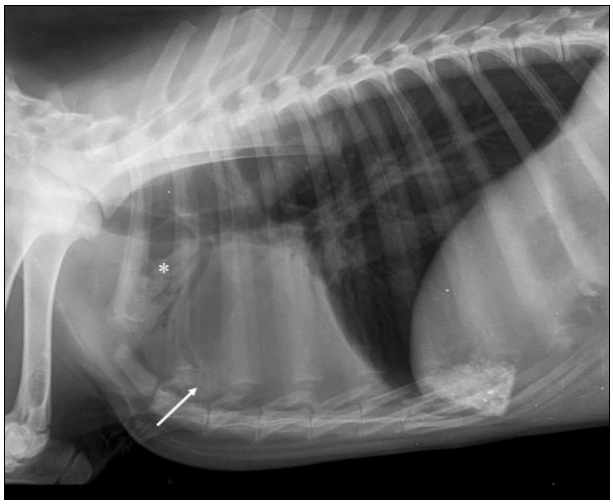

Fig 3: Lateral thoracic radiograph showing congestion in the lungs (*) and pleural effusions (arrow)

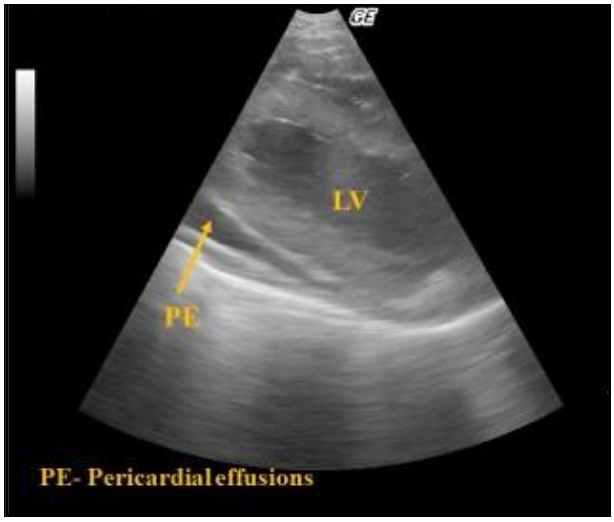

Fig 4: Anechoic region in right parasternal long axis left ventricular outflow view indicative of mild to moderate pericardial effusions

Result and discussion

Sheep infected with babesiosis displayed notable clinical signs, including anorexia/inappetence, weakness, fever, pale conjunctival mucous membrane, tachycardia, respiratory distress, absence of rumeno-reticular motility, haemoglobinuria, and a history of tick infestation. These observations align with findings reported by Venkatesakumar *et al.* (2018) [15] and Stuen (2020) [12]. Elevated cytokines (TNF-α, IL-1, and IL-6) produced by mononuclear cells in response to the systemic inflammatory response induced by piroplasm contribute to diverse clinical symptoms (Hasanpour *et al.*, 2008) [5]. The presence of *Babesia* piroplasm within red blood cells may link to severe hemolysis, leading to hemoglobinuria (Ul-Haq *et al.*, 2017) [14]. Ruminal hypomotility in infected animals, which was attributed to increased body temperature or fever (Hassan *et al.*, 2015).

In hematology, Babesia-infected sheep exhibited decreased levels in hemoglobin, total erythrocyte count (TEC), platelets, and packed cell volume (PCV) compared to reference values. Similar findings were reported by Ul-Haq *et al.* (2017) [14], Stuen (2020) [12], Haq *et al.* (2021) [7], and Villanueva-Saz *et al.* (2022) [16] regarding hemoglobin, TEC, thrombocytes, and PCV. The observed anemia can be attributed to red blood cell destruction containing piroplasms, complemented by factors like autoimmune hemolysis and suboptimal bone marrow response (Constable *et al.*, 2017) [3]. Leukocytosis was also observed, potentially due to extended tissue damage by parasites and the maturation of neutrophils and lymphocytes (Sivajothi *et al.*, 2022) [11].

In serum biochemistry, elevated levels of creatinine kinase MB were observed, consistent with findings reported by Orunc-Kilinc *et al.* (2015) [10] in sheep babesiosis. This elevation is attributed to myocardial muscle cell necrosis resulting from anemic anoxia due to severe hemolytic anemia (Constable *et al.*, 2017) [3]. Babesia-infected sheep showed a decrease in total protein, albumin, and globulin, in line with similar findings reported by Stuen (2020) [12] and Villanueva-Saz *et al.* (2022) [16]. The reduction in serum proteins could be attributed to disturbances in liver function, urinary loss of proteins associated with renal failure (proteinuria), and anorexia (Sivajothi *et al.*, 2022) [11]. Elevation in ALT and GGT could result from hepatic damage by the parasites in the tissue and muscle trauma caused by prolonged recumbency in haemoprotozoa infection (Hasanpour *et al.*, 2008) [5]. Increased bilirubin levels may stem from both intravascular and extravascular destruction of parasitized erythrocytes, particularly through erythrophagocytosis in the spleen, lymph nodes, and other organs of the reticuloendothelial system, and also from hepatic dysfunction (Venkatesakumar *et al.*, 2018) [15]. The elevation in creatinine and BUN be due to kidney dysfunction, increased muscle catabolism in babesiosis (Constable *et al.*, 2017 and Villanueva-Saz *et al.*, 2022) [3, 16].

In thoracic radiography of infected sheep, pulmonary edema and pleural effusions are attributed to a breakdown in the blood-air barrier, possibly due to disruption in the endothelial or pneumocyte layers. The triggering mechanism involves inflammation-induced injury, notably with monocyte macrophage accumulation in the lungs and the buildup of inflammatory fluid leading to the development of pulmonary edema and congestion (Martin *et al.*, 2023) [9].

Echocardiography showed an elevated LA/Ao ratio, while all other parameters remained within the normal range. Similar findings in haemoparasitic diseases were reported by Tejaswini (2017) [13]. This elevation may be attributed to the impact of babesiosis on the left atrium and myocardial dysfunction. Pericardial effusion could result from myocarditis in response to babesia organisms or an inflammatory response.

Conclusion

In conclusion, the presented case highlights the varied and complex effects of babesiosis on a Bannur sheep, affecting not only hematological and biochemical parameters but also cardiac and pulmonary functions. The observed clinical signs, coupled with abnormalities in echocardiography, thoracic radiography and serum biochemistry, provide a comprehensive understanding of the disease's pathophysiology.

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