

**ACUTE *BABESIA FELIS* INFECTION IN A HIMALAYAN CAT: MOLECULAR DIAGNOSIS, CLINICOPATHOLOGICAL FEATURES AND THERAPEUTIC MANAGEMENT**

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**ABSTRACT**

*The genus Babesia contains haemoprotozoan parasites that cause feline babesiosis, an ailment spread by ticks. Several species of Babesia have been found in cats, the most common of which being Babesia felis. A two-year-old intact male Himalayan cat was presented with the signs of anorexia, lethargy, dehydration, moderate jaundice and decreased urination. The cat was in lateral recumbency and the clinical examination showed palpable lymphadenopathy, icteric mucous membranes, pyrexia and severe dehydration. Anaemia, thrombocytopenia, elevated liver parameters and hypoproteinaemia were all found by hematobiochemical analysis. The causative agent was identified as B. felis by reverse transcription quantitative polymerase chain reaction. The cat was treated with primaquine phosphate, doxycycline and supportive therapy. The cat recovered uneventfully with normal hematobiochemical parameters. This report details the diagnostic approach and successful therapeutic management of B. felis infection in a cat.*

**Key words:** *Babesia felis*, primaquine, anaemia

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**INTRODUCTION**

Hemoprotozoan diseases in cats are caused by protozoan parasites such as

*Hepatozoon felis*, *Cytauxzoon felis*, *Babesia spp.* and hemotropic *Mycoplasma* species, posing an emerging global health concern due to their increasing prevalence, diverse clinical manifestations and potential for severe or fatal outcomes and are primarily transmitted through arthropod vectors like ticks and fleas (Malangmei *et al.*, 2021; Wikander and Reif, 2023). Feline babesiosis, primarily caused by *Babesia felis*, is a tick-borne protozoal disease

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increasingly recognized in veterinary medicine, especially in areas like South Africa and parts of Asia where competent vector ticks are prevalent (Bosman *et al.*, 2010; Penzhorn and Kjemtrup, 2019). Infection typically leads to hemolytic anemia, thrombocytopenia, jaundice and hepatic dysfunction, which may be fatal if untreated (Jacobson, 2018). Historically, diagnosis relied on microscopy of blood smears, however, molecular techniques such as polymerase chain reaction (PCR) afford sensitive and specific detection (Criado-Fornelio, 2003). This report details the clinical course, diagnostics, treatment and outcome of a PCR confirmed case of *B. felis* in a Himalayan cat.

### **CASE HISTORY AND OBSERVATIONS**

A two year and three-month-old male, indoor cat of Himalayan breed, weighing 4.5 kg, was presented with a history of anorexia, lethargy and yellowing of sclera and skin for two days. The cat had been regularly vaccinated and dewormed, but no ectoparasite prevention was done. On physical examination, the cat was found in lateral recumbency and exhibited depressed mentation. The rectal temperature was 104.8°F, indicating fever. Mucous membranes, sclera and skin were icteric (Fig.1). Clinical assessment revealed dehydration of six to eight percent. Capillary refill time was prolonged to three seconds and peripheral lymphadenopathy was noted. Cardiovascular evaluation revealed a heart rate of 174 beats per minute and a bounding pulse. The respiratory rate was elevated at

35 breaths per minute.

Hematological analysis (Table 1) on presentation demonstrated regenerative anemia and thrombocytopenia. The biochemical profile showed severe hyperbilirubinemia and marked elevation of alanine aminotransferase (ALT) levels, indicating significant hepatocellular injury. Additionally, hypoproteinemia and hypoalbuminemia were evident. Creatinine was borderline low, while blood urea nitrogen remained within normal limits.

Molecular testing of whole blood, targeting 18S rRNA, was done using a reverse transcription quantitative polymerase chain reaction (RT-qPCR), which confirmed active infection with *B. felis*, indicated by a cycle threshold (CT) value of 28.4. PCR tests for *Mycoplasma haemofelis*, feline parvovirus and feline immunodeficiency (FIV) virus were negative, excluding these common differential diagnoses.

### **TREATMENT AND DISCUSSION**

Therapeutic intervention began immediately upon diagnosis with single dose oral administration of primaquine phosphate tablet (Malirid 7.5mg, IPCA Laboratories Ltd.) at 0.5 mg/kg body weight. Concurrently, intravenous doxycycline (Paladox 100, Oriheal Lifesciences) at 10 mg/kg once daily was initiated to address possible coinfections and to exert immunomodulatory effects. Supportive therapy was instituted with intravenous fluids of five percent dextrose saline to correct dehydration, antioxidant vitamin C to mitigate oxidative stress, intravenous dextrose 25 percent and

B-complex vitamins to provide metabolic and nutritional support. Prednisolone was administered intramuscularly at 0.5 mg/kg daily to manage inflammation and. On day five, follow-up blood test reports (Table 1) indicated improvement in all hematological parameters and ALT levels and total bilirubin decreased significantly. From day six, the appetite of the cat improved and oral doxycycline syrup was continued at 5 mg/kg orally twice daily for five days. Hepatoprotective therapy with liver supplement syrup (Hepamust, Mankind Pharmaceuticals, India) at two ml twice daily was provided for fifteen days, and iron supplementation using haematinic syrup (Ferrikind, Mankind Pharmaceuticals, India) at 0.5 ml twice daily was administered for 10 days to support liver function and hematologic recovery, respectively. The icterus reduced and normal appetite and activity levels returned by day 10.

The clinical presentation in this case, characterized by severe hemolytic anemia, thrombocytopenia, marked icterus and hepatocellular injury was consistent with acute *B. felis* infection. Rapid, species-specific confirmation through RT-qPCR allowed for the prompt initiation of targeted therapy, a critical factor in improving clinical outcomes in feline babesiosis (Baneth *et al.*, 2004; Almendros *et al.*, 2023). Primaquine phosphate remains the cornerstone of therapy for *Babesia spp.* in cats due to its proven efficacy in parasite clearance (Baneth *et al.*, 2004), while doxycycline is employed both to address potential co-infections

with other tick-borne pathogens and to exert beneficial immunomodulatory effects (Kumar, 2008). Aggressive supportive care, including intravenous fluid administration, hepatoprotective agents and nutritional support with iron and B-complex vitamins, plays an essential role in correcting dehydration and supporting hepatic recovery (Kumar, 2008). The administration of prednisolone at anti-inflammatory doses assists in the management of immune-mediated hemolysis and associated inflammatory responses (Pusoonthornthum *et al.*, 2012). The absence of co-infections such as *M. haemofelis* or FIV enabled a focused therapeutic plan. The marked clinical and hematologic improvements observed following this multimodal therapeutic protocol underscore the importance of integrating targeted anti-babesial therapy, immunomodulation and intensive supportive measures for the successful management of feline babesiosis.

## CONCLUSION

*Babesia felis* infection should be a differential diagnosis in cats presenting with acute hemolytic anemia and jaundice, particularly in endemic areas. Early molecular identification using RT-qPCR coupled with comprehensive medical management enables favourable outcomes. This case exemplifies the integration of advanced diagnostics and therapeutic strategies leading to full clinical recovery in feline babesiosis.

**Table 1: Hematological and biochemical analysis on day 0 and day 5.**

Parameter	0th day	5th day	Reference
TLC	12.10	10.17	5.00 - 18.90 K/ $\mu$ l
HGB	6.9	8.2	8.0 - 15.0 g/dl
HCT	22.0	31.4	24.0 - 45.0 %
MCHC	31.4	32.6	30.0 - 36.9 g/dl
PLT	111	182	175 - 500 K/ $\mu$ l
Creatinine	0.41	0.9	0.8-1.8 mg/dl
BUN	25.9	34.9	17.6 - 32.8 mg/dl
ALT	361	102	22 - 84 IU/l
Total bilirubin	1.3	0.5	0.1 - 0.4 g/dl
Total protein	4.8	5.5	5.7 - 7.8 g/dl
Albumin	1.5	2.7	2.3 - 3.5 g/dl
Blood glucose	62	104	90-120 mg/dl



**Fig.1. Icteric mucus membrane and skin**

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