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IMMUNE-MEDIATED HAEMOLYTIC ANAEMIA SECONDARY TO FELINE HAEMOTROPIC MYCOPLASMOSIS

[1]Abhijith S.P., [2]Apoorva H.J., [3]Sanjay H.V., [4]Kshama M.A. and [5]Lathamani V.S.

[1,2,3]M. V. Sc Scholar, Department of Veterinary Medicine, Veterinary College, Bengaluru
[4]Professor & Head, Department of Veterinary Medicine, Veterinary College, Bengaluru
[5]Assistant Professor, Department of Veterinary Medicine, Veterinary College, Bengaluru

*Corresponding author: abhijithsp560@gmail.com

ABSTRACT

Feline haemotropic mycoplasmosis (FHM) results from infection with *Mycoplasma haemofelis*, a small epicellular bacterial organism that adheres to the surface of feline erythrocytes. Formerly known as *Hemobartonella felis*, this pathogen induces haemolysis either through direct damage to red blood cells or by triggering immune-mediated destruction as the altered erythrocytes are recognised as foreign by the host's immune system. Progressive destruction of erythrocytes may lead to varying degrees of anaemia, depending on the severity and duration of infection. A four-year-old neutered domestic short-haired cat was presented to Small Animal Clinic, Teaching Veterinary Hospital, Veterinary College, Bengaluru with a history of anorexia, lethargy, abnormal breathing and yellow mucous membrane. Following clinical evaluation, haematobiochemical profiling, and PCR analysis, a diagnosis of Feline Haemotropic Mycoplasmosis was established. The concurrent development of Immune-Mediated haemolytic anaemia was confirmed via a positive Coombs test. The case was successfully treated and recovered. This clinical case report describes about the case and therapeutic management.

Keywords: Mycoplasma, immune mediated haemolytic anaemia, Coomb's test

INTRODUCTION

Feline haemotropic mycoplasmosis (FHM), formerly known as feline infectious anaemia (FIA), is caused by haemotropic mycoplasmas that parasitise feline erythrocytes (Tasker, 2010). Haemotropic mycoplasmas are small (0.3–0.8 µm), pleomorphic, unculturable, epierythrocytic bacteria that can cause severe haemolytic anaemia in cats (Sykes, 2010). The primary pathogen, *Mycoplasma haemofelis*, is a

unique, pleomorphic, cell wall-deficient bacterium that attaches to the surface of red blood cells (Messick, 2003). Upon attachment, it alters the membrane structure and exposes hidden antigens, thereby triggering an immune-mediated response leading to haemolysis (Greene *et al.,* 2012). Clinically, affected cats present with pallor, lethargy, anorexia, weakness and occasionally icterus (Westfall *et al.,* 2001). The severity of anaemia correlates with both the degree of parasitaemia and the host's immune response (Tasker, 2010). Upon attachment to erythrocytes, *Mycoplasma haemofelis* proliferates on the red blood cell (RBC) surface, altering membrane antigenicity and thereby triggering an immune-mediated response in which the host's immune system identifies the parasitised erythrocytes as foreign and targets them for destruction (Hartmann *et al.,* 2003). This immune-mediated haemolysis is further substantiated by Coomb's-positive results in many affected cats, indicating the presence of erythrocyte-bound antibodies (Hartmann *et al.,* 2003). The clearance of these antibody-coated erythrocytes is primarily facilitated by splenic macrophages through extravascular haemolysis (Tasker, 2010; Messick, 2003). The rapid and excessive destruction of RBCs often surpasses the regenerative capacity of the bone marrow, leading to either regenerative or non-regenerative anaemia depending on the disease stage (Foley *et al.*, 2001). Clinically, this progressive anaemia manifests as pallor, lethargy, weakness, and, in some cases, icterus, with severity correlating directly to both the parasitaemia level and the intensity of the immune-mediated response (Tasker, 2010).

MATERIALS AND METHODS

A four-year-old, male neutered cat was presented to the Small Animal Clinic, Veterinary College Hospital, Bengaluru, with a history of lethargy, inappetence, progressive weight loss over two weeks and yellowish colouration of the gums. On physical examination, the cat was depressed and mildly dehydrated. The mucous membranes appeared pale with an icteric tinge (Fig.1.1-1.4). Mild tachycardia (heart rate: 210 bpm) and tachypnoea (respiratory rate: 40 breaths per minute) were noted. Rectal temperature was 102.3ºF (slightly elevated). Abdominal palpation revealed no significant abnormalities, but splenomegaly was suspected.

Laboratory tests were conducted including complete blood count and serum biochemical analysis. Blood smear examination was conducted for direct demonstration of the organism. Molecular confirmation was done using PCR.

Fig.1.1-1.4: Icteric skin, gums, mucus membranes and paw pads (left to right)

RESULTS AND DISCUSSION

Laboratory findings included reduced haematocrit (PCV: 18%), reduced haemoglobin (5.9g-dL), RBC (2.9x10^6/µL), reticulocytosis (1.8%) and polychromasia. Leucocytosis (23000/µL) with neutrophilia and mild monocytosis (2100/µL) was present. Serum biochemistry showed hyperbilirubinemia (TBIL 2.4 mg/dL). Blood smear examination demonstrated small, epicellular organisms attached to the surface of red blood cells suggestive of *Mycoplasma haemofelis*. The PCR assay on whole blood confirmed the presence of *Mycoplasma haemofelis* DNA. No evidence of concurrent FeLV or FIV infection (Fig.1.5). A positive Coomb's test confirmed the coexistence of immune-mediated haemolytic anaemia as a complication of the infection.

Peripheral blood smear revealed moderate anisocytosis, polychromasia, spherocytosis and epicellular basophilic inclusions on erythrocytes suggestive of *Mycoplasma haemofelis* organisms. Coomb's test (direct antiglobulin test)

Table 1: PCR report of the case (Multi-Panel PCR was done to rule out co-infections)

MOLECULAR DIAGNOSIS

Performed using gradient polymerase chain reaction on Applied Biosystem Thermocycler

Date	Sl no	Test for	Result
28-06-2025	1.	*Mycoplasma*	**POSITIVE**
28-06-2025	2.	*Feline Babesiosis*	NEGATIVE
28-06-2025	3.	*FeLV*	NEGATIVE
28-06-2025	4.	*FIP*	NEGATIVE

was positive, supporting the presence of immune-mediated erythrocyte destruction.

Hyperbilirubinemia was attributed to intravascular and extravascular haemolysis. Mild ALP elevation possibly due to cholestasis from increased bilirubin. Renal and hepatic parameters were otherwise unremarkable.

Mycoplasma haemofelis attaches to the surface of feline erythrocytes, triggering immune-mediated destruction that leads to massive haemolysis and excessive bilirubin production. This overwhelms the liver's capacity to conjugate bilirubin, resulting in its accumulation due to haemolysis rather than hepatic dysfunction or biliary obstruction, and is therefore classified as haemolytic (prehepatic) jaundice.

Doxycycline (10 mg/kg IV OD 5 days) given as first-line antimicrobial targeting *Mycoplasma* and prednisolone (2 mg/kg IM OD, immunosuppressive dose) to control immune-mediated haemolysis. Supportive care with IV fluids and close monitoring was done for five days. Within five days of initiating therapy, the cat demonstrated improved appetite, activity and stabilisation of haematocrit. By day 14, PCV improved to 28%, and icterus resolved. Prednisolone was gradually tapered over eight weeks after haematologic normalisation.

Feline haemotropic mycoplasmosis is caused by *Mycoplasma haemofelis*, a small, epicellular, erythrocyte-parasitising bacterium (Tasker, 2010; Sykes, 2014). Haemotropic mycoplasmas are pleomorphic bacteria that infect feline erythrocytes by attaching to the cell surface and transmission occurs through multiple routes: flea bites, blood transfusions, vertical transmission, and direct inter-cat contact (Senthil *et al.,* 2014). The organism attaches to red blood cells, leading to both direct damage and immune-mediated destruction (Willi *et al.,* 2007; Tasker, 2010). In this case, the regenerative anaemia (as evidenced by reticulocytosis and polychromasia), spherocytosis, hyperbilirubinemia, and a positive Coomb's test confirmed an ongoing haemolytic process with a strong immune-mediated component (Sykes, 2014; Hartmann *et al.,* 2015). The leucocytosis with neutrophilia and monocytosis reflected a systemic inflammatory response to infection (Sykes, 2014). While FHM can cause haemolysis directly via oxidative and mechanical injury to RBCs, a significant proportion of cases also involve secondary IMHA, where the host immune system produces antibodies against parasitised and even non-parasitised RBCs (Tasker, 2010; Hartmann *et al.,* 2015). This complicates the disease course and often worsens anaemia (Tasker, 2010).

FHM can result in severe regenerative anaemia often complicated by IMHA (Sykes, 2014; Tasker, 2010). Accurate diagnosis using a combination of blood smear, PCR and Coombs' test is critical (Willi *et al.,* 2007; Tasker, 2010). Early antimicrobial and immunosuppressive therapy can achieve favourable outcomes (Hartmann *et al.,* 2015; Sykes, 2014).

Strandberg *et al.* (2023) reported the first case of haemophagocytic syndrome in a cat infected with *Mycoplasma haemofelis*, diagnosed via PCR despite no organisms visible cytologically. The cat exhibited bicytopenia and prominent hemophagocytic macrophages in both spleen and bone marrow, confirmed histologically in the absence of neoplasia. Lymphocytic erythrophagocytosis highlighted an immune-mediated cytopenia secondary to *M. haemofelis*, indicating that hemophagocytic syndrome should be considered a potential, severe complication of feline Haemotropic mycoplasmosis.

In the case described by Sukumaran and Okene (2019), microscopic examination revealed rod-shaped, epicellular mycoplasma organisms on feline erythrocytes, confirming haemotropic mycoplasmosis, while concurrent *Escherichia coli* infection in the urinary tract established a dual diagnosis of FHM with bacterial cystitis.

To summarise, a five-year-old male cat was presented with lethargy, pale and icteric mucous membranes, tachycardia, and splenomegaly. Haematology revealed severe regenerative anaemia, leucocytosis, hyperbilirubinemia and spherocytosis. Blood smear and PCR confirmed *Mycoplasma haemofelis* infection, with a positive Coombs' test indicating secondary immune-mediated haemolytic anaemia (IMHA). The cat was treated with doxycycline and immunosuppressive prednisolone along with supportive care. Rapid clinical improvement was noted within days, with gradual normalisation of haematologic values. Complete recovery was achieved after tapering immunosuppressive therapy.

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