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Feline Secondary Immune Mediated Haemolytic Anaemia: A Clinical and Diagnostic Overview

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Abstract

Immune-mediated haemolytic anaemia in cats is a severe and potentially life-threatening haematological disorder characterized by immune-mediated destruction of erythrocytes. In feline patients, the condition is more commonly secondary in origin and may develop in association with a variety of underlying aetiologies, including infectious, neoplastic, inflammatory, drug-induced, and idiopathic causes. Affected cats may present with a broad spectrum of clinical manifestations, with the most frequently observed signs including pallor to icteric mucous membranes, pyrexia, lethargy, tachycardia and systemic inflammatory response syndrome (SIRS)-associated alterations. Diagnosis requires a comprehensive multimodal approach incorporating both conventional and advanced diagnostic techniques. Therapeutic management primarily involves immunosuppressive therapy, particularly corticosteroids, often in combination with adjunct immunosuppressive agents in refractory or severe cases. Long-term follow-up and periodic clinical and laboratory monitoring are essential to assess therapeutic response, detect relapses, and optimize overall prognosis.

Keywords: RBC, IMHA, Immunoglobulins, Agglutination, Anaemia, Jaundice, Corticosteroid

Introduction

Immune-mediated haemolytic anaemia (IMHA) is a clinically important haemato-immunological disorder affecting a wide range of animal species, particularly dogs and cats. Among feline patients, the disease is considered comparatively underdiagnosed, possibly owing to its variable clinical presentation, multifactorial aetiology and complex pathogenesis. The disorder is characterized by immune-mediated destruction of circulating erythrocytes following the attachment of immunoglobulins (IgG, IgM, or both) and complement components to the erythrocyte membrane, ultimately resulting in haemolysis and premature erythrocyte clearance. Pathophysiologically, IMHA is recognized as a classical Type II hypersensitivity reaction and is broadly classified into primary (idiopathic) and secondary forms. The primary form, which may occur as idiopathic IMHA, pure red cell aplasia (PRCA), or non-regenerative immune-mediated

haemolytic anaemia, is reported more frequently in dogs than in cats. Conversely, secondary IMHA is more prevalent in feline patients and is commonly associated with a variety of underlying conditions, including infectious (bacterial, viral, haemotropic, or parasitic), toxic, inflammatory, neoplastic, and paraneoplastic disorders. Certain pharmacological agents have also been implicated as potential triggers. Additionally, pregnancy-associated IMHA has been reported in cats, with resolution observed following ovariohysterectomy (Kopke *et al*., 2019).

Although idiopathic IMHA is considered relatively uncommon in cats compared to dogs, recent advances in diagnostic methodologies and improved clinical recognition have contributed to an increasing number of reported primary cases. The acute and often severe immune-mediated destruction of erythrocytes results in characteristic clinical manifestations, including pyrexia, icterus, and varying degrees of anaemia, which may present as acute or chronic in nature. The severity and spectrum of clinical signs are influenced by both the extent of erythrocyte destruction and the underlying aetiology. Common clinical findings in affected cats include pallor to icteric mucous membranes, tachycardia, tachypnoea, lethargy, anorexia, weakness, dehydration, and, in severe cases, systemic inflammatory response syndrome (SIRS)-associated abnormalities. Although relapse may occur, recurrence rates in feline IMHA are generally considered lower than those reported in canine patients.

Table1: Secondary immune-mediated haemolytic anaemia (IMHA) in cats is commonly associated with infectious agents, neoplastic disorders, drugs, immune stimulation, and transfusion reactions. The listed aetiologies represent reported triggers implicated in immune-mediated erythrocyte destruction in feline patients.

Aetiology

Agent	Aetiology	Description	Reference
Bacteria	*Haemoplasma* *Mycoplasma spp.* (includes *Mycoplasma haemofelis*)	Pleomorphic epierythrocytic bacteria that lack a cell wall, causing **Feline Infectious Anaemia.**	Ferraz *et al*., 2024
Protozoa	Babesia *spp.* (like *Babies gibsoni*)	Intra-erythrocytic, haemoprotozoan (piroplasms)	Almendros A *et al* ,2023
Virus	Feline Leukemia Virus (FeLV)	Enveloped RNA virus belonging to the genus Gamma retrovirus of the family Retroviridae	Westman ME *et al*., 2025.
Virus	Feline Immunodeficiency Virus (FIV)	Enveloped RNA virus of the genus Lentivirus, subfamily Orthoretrovirinae, family Retroviridae.	L S de Mello, *et al*., 2025

Virus	Feline Infectious Peritonitis	A mutated Feline Corona Virus (FeCoV) RNA virus, a rare and uncommon cause of IMHA.	Černá P.*et al.*, 2025
Drugs	Propyl thiouracil & Methimazole	An anti-thyroid medications for cats with hyperthyroidism; can induce IMHA as an adverse drug reaction.	Peterson ME *et al.*, 1984
Drugs	Famotidine	Histamine (H2) Receptor Antagonist	Paes *et al.*, 2010
Drugs	Trimethoprim-sulfa	Potentiated sulphonamide antimicrobial	-
Immuno therapy	Vaccines	Occasionally reported, but there is no clear evidence of an association with IMHA	-
Idiosyncrasy	Incompatible blood transfusion and xenotransfusion (same species – allotransfusion; different species – xenotransfusion)	Immune cells attack incompatible donor erythrocytes, causing lysis even after the first transfusion	-
Neoplasia	Lymphoma	One of the most common neoplastic causes of IMHA; may arise from lymphoid malignancies of solid organs (e.g., lymph nodes, liver, spleen).	Couto C., 2001 and Akiyoshi M *et al.*, 2020 (reported in a cat with gastrointestinal lymphoma)
Neoplasia	Myeloproliferative neoplasms	Rare proliferative bone marrow neoplasia often associated with feline leukaemia virus infection	-

Neoplasia	Multiple myeloma	A malignant tumour of plasma cells derived from differentiated B - cells.	-
Others	Zootoxins	Agents such as snake venom; in reported cases, the exact trigger for IMHA was unclear but may be related to specific venom components.	Ong H *et al*., 2015

Footnote:
IMHA = Immune-mediated haemolytic anaemia; RBC = Red blood cell; FeLV = Feline leukaemia virus; FIV = Feline immunodeficiency virus; FeCoV = Feline coronavirus. Secondary IMHA develops due to underlying infectious, neoplastic, drug-induced, toxic, or immune-mediated conditions that trigger erythrocyte destruction.

Epidemiology

As secondary causes are frequently associated with persistent infections, chronic inflammatory conditions, or other underlying pathological foci, the epidemiology of Immune-mediated haemolytic anaemia in cats varies considerably among different feline populations and geographical regions. Several predisposing factors, including previous disease history, concurrent pharmacological therapy, vaccination status, exposure to toxic substances, animal bite wounds, vector distribution, and environmental or physiological stressors, may influence both the occurrence and severity of the disease. Feline IMHA is most commonly reported in middle-aged cats; however, animals of any age may be affected. In comparison with dogs, the overall prevalence of IMHA in cats is considerably lower and constitutes only a small proportion of anaemia cases encountered in routine clinical practice. To date, no consistent breed, sex, or geographic predisposition has been conclusively established, although variations in prevalence may occur depending on regional infectious disease burden and environmental risk factors.

Pathophysiology

Due to the diverse etiological factors described above, immune responses initially directed against infectious or foreign antigens may become aberrantly targeted toward self-erythrocyte surface antigens, particularly membrane glycoproteins. This autoimmune dysregulation results in the binding of immunoglobulins predominantly IgM, followed by IgG, or both, and less commonly IgA to the surface of red blood cells (RBCs). In certain cases, autoantibodies may also target erythroid precursor cells within the bone marrow. The attachment of immunoglobulins to erythrocyte membranes, which subsequently function as autoantigens, promotes erythrocyte destruction through mechanisms including extravascular haemolysis, intravascular haemolysis, and intravascular erythrocyte agglutination (Balch and Mackin, 2007). Intravascular haemolysis, although less frequently observed, is primarily mediated by complement activation and results in direct erythrocyte lysis. This form of haemolysis is often acute, severe, and may be life-threatening

(Goggs and Behling-Kelly, 2019). Binding of immunoglobulins to erythrocyte surfaces activates the classical complement pathway, thereby initiating complement-mediated erythrocyte destruction. Complement activation may also contribute to extravascular haemolysis through the deposition of complement components on circulating RBCs, a process referred to as complement-mediated lysis (Swann and Skelly, 2016). This mechanism is associated with membrane injury, osmotic imbalance, cellular swelling, and eventual erythrocyte rupture.

Extravascular haemolysis occurs predominantly within the spleen and liver, where macrophages of the mononuclear phagocyte system recognize antibody- and complement-coated erythrocytes via Fc and complement receptors. Subsequent phagocytosis and destruction of these sensitized erythrocytes result in premature erythrocyte removal from circulation (Swann and Skelly, 2016). In some cases, erythrocytes may undergo agglutination within the vasculature; however, in contrast to canine IMHA, thromboembolic complications are considered uncommon in feline secondary immune-mediated haemolytic anaemia (IMHA).

Biochemical Alterations

The biochemical alterations associated with immune-mediated haemolytic anaemia (IMHA) vary according to the underlying aetiology and the predominant mechanism of haemolysis. In cases of intravascular haemolysis, extensive erythrocyte destruction within the vasculature results in the release of free haemoglobin into the circulation, leading to haemoglobinaemia and subsequent haemoglobinuria. The rapid and excessive release of haemoglobin may exceed the binding capacity of physiological scavenging systems, particularly haptoglobin, as well as the clearance capacity of hepatic reticuloendothelial cells. Consequently, accumulation of haem pigments and increased bilirubin production contribute to the development of hyperbilirubinaemia and clinically evident icterus. In contrast, extravascular haemolysis is predominantly mediated by macrophage phagocytosis of antibody-coated erythrocytes within the spleen and liver. During this process, haem catabolism generates unconjugated bilirubin, which binds to albumin and circulates within the bloodstream. Provided hepatic uptake and conjugation mechanisms remain functionally adequate, the increased bilirubin load can be effectively compensated for. However, when bilirubin production exceeds hepatic processing capacity, or when concurrent hepatic dysfunction is present, bilirubinaemia, bilirubinuria, and overt icterus may develop.

In addition to these haemolytic changes, inflammatory mediators and cytokines are believed to contribute to disease pathogenesis and systemic complications. Increased cytokine production has been associated with immune activation and concurrent infectious or inflammatory conditions in affected animals. Nevertheless, in companion animals, particularly cats, the precise role of cytokines in the development and progression of systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction syndrome (MODS) remains insufficiently characterized and warrants further investigation.

Clinical Manifestations

Affected animals commonly present with generalized weakness, lethargy, progressive weight loss, and varying degrees of reduced appetite, ranging from inappetence to complete anorexia. Clinical examination may reveal intermittent or persistent pyrexia, peripheral lymphadenopathy, and hepatosplenomegaly, reflecting activation of the mononuclear phagocyte system and increased erythrocyte sequestration. Delayed capillary refill time, dehydration, and evidence of compromised

peripheral perfusion may also be observed. In some cases, immune-mediated haemolytic anaemia (IMHA) may occur concurrently with immune-mediated thrombocytopenia, a condition referred to as Evan's syndrome. Ongoing erythrocyte destruction results in clinically apparent anaemia, most commonly manifested by pale mucous membranes. In cases of marked haemolysis, excessive bilirubin production leads to icterus, with yellow discoloration of the mucous membranes and connective tissues, a clinical appearance sometimes colloquially described as the "yellow cat." Haemoglobinuria or bilirubinuria may also occur, resulting in abnormal urine discoloration due to renal excretion of haem pigments. When pigment load exceeds renal clearance capacity, pigment-associated nephropathy and impaired renal function may develop. Cats are generally considered relatively tolerant of chronic anaemia; however, prolonged reductions in oxygen-carrying capacity may induce compensatory cardiovascular adaptations. These responses commonly include tachycardia, tachypnoea, and, in some cases, functional cardiac murmurs detectable during auscultation. Chronic or severe anaemia may additionally predispose affected animals to myocardial stress and secondary cardiomyopathic changes. In advanced cases, inadequate tissue oxygenation may result in syncopal episodes, weakness, collapse, or signs consistent with cerebral hypoxia secondary to severe anaemia.

Diagnosis

Diagnosis can be established through integration of clinical presentation, haematological abnormalities, serum biochemical alterations, and confirmatory immunologic testing. Because the clinical manifestations of IMHA are often variable and nonspecific, accurate diagnosis requires comprehensive laboratory evaluation to minimise the risk of misdiagnosis.

a. Haematological Findings

Haematological evaluation typically demonstrates severe, and often life-threatening, anaemia characterised by markedly reduced haematocrit values and altered erythrocyte indices. Concurrent thrombocytopenia warrants investigation for Evans syndrome, defined as the coexistence of IMHA and immune-mediated thrombocytopenia. Examination of peripheral blood smears may reveal *Mycoplasma* spp. organisms attached to the erythrocyte surface (epierythrocytic organisms), suggesting secondary IMHA associated with haemotropic mycoplasmosis. The identification of spherocytes, which result from partial phagocytosis of erythrocytes by splenic macrophages, provides evidence of ongoing immune-mediated haemolysis and is considered a characteristic morphological feature of IMHA.

b. Serum Biochemical Alterations

Serum biochemical analysis commonly reveals hyperbilirubinaemia secondary to increased erythrocyte destruction, together with mild to moderate elevations in hepatic enzyme activities. Albumin concentrations are frequently normal to decreased, whereas globulin concentrations are often increased due to inflammatory and immune-mediated responses. Additional biochemical abnormalities may include elevated blood lactate concentrations, azotaemia, increased serum iron and total iron-binding capacity, and increased concentrations of acute-phase proteins such as feline serum amyloid A. Electrolyte disturbances may also be present, depending on the severity of haemolysis and the extent of concurrent renal involvement.

Confirmatory Tests

A. The Saline Agglutination Test (SAT)

SAT is commonly employed as a rapid screening method for the detection of erythrocyte autoagglutination in cats suspected of immune-mediated haemolytic anaemia (IMHA). In this procedure, one drop of anticoagulated (EDTA) blood is mixed with three to four drops of isotonic saline on a microscope slide. Persistence of erythrocyte clumping after saline dilution, which disperses rouleaux formation, is indicative of true autoagglutination. On microscopic examination, the presence of grape-like aggregates consisting of four to five or more erythrocytes is considered a positive finding. Nevertheless, the SAT has limited sensitivity, and a negative result does not definitively exclude IMHA.

B. Direct Antiglobulin Test (Coombs' Test)

The Direct Antiglobulin Test (DAT), commonly referred to as the Coombs' test, is regarded as the gold standard for the diagnosis of IMHA. This assay detects immunoglobulins (primarily IgG and IgM) and/or complement components (C_3) bound to the surface of erythrocytes. The test is performed using a 2% suspension of washed erythrocytes, which is incubated with Coombs' reagent containing polyclonal antibodies directed against feline immunoglobulins and complement proteins. The development of microscopic agglutination is interpreted as a positive reaction, confirming immune-mediated erythrocyte destruction. Quantitative variants of the assay, including microtiter plate methods, have been described; however, their routine use is limited by higher costs and restricted availability of specialised reagents.

C. Flow Cytometry

Flow cytometry represents an advanced diagnostic modality and, in certain laboratory settings, may provide a more practical alternative to the Coombs' test. This technique enables quantitative assessment of erythrocyte-bound IgG and IgM and can also be utilised for monitoring therapeutic response. Antibody binding is evaluated through measurement of mean fluorescence intensity, which is visualised on dot plots or histograms generated by the flow cytometer. Limlenglert *et al.* (2011) reported that flow cytometry demonstrated a sensitivity of 100% and a specificity of 50% when compared with the direct agglutination test. The positive predictive value and negative predictive value were 41.66% and 100%, respectively, indicating that while false-positive results may occur, a negative result is highly reliable for excluding disease.

Differential diagnosis

Secondary immune-mediated haemolytic anaemia (IMHA) frequently overlaps with numerous other causes of anaemia and icterus, thereby necessitating a comprehensive and systematic differential diagnostic approach. Infectious diseases represent important differential diagnoses and should be carefully excluded in cats presenting with haemolytic anaemia. These include parasitic and infectious conditions such as ancylostomiasis, haemoplasmosis, babesiosis, cytauxzoonosis, and feline retroviral infections. Polymerase chain reaction (PCR) assays and serological testing are valuable diagnostic tools for confirming or excluding these infectious aetiologies. A thorough therapeutic and medical history may provide important diagnostic information, particularly in cats receiving treatment for concurrent disorders. Drug-associated haemolysis should be considered in patients exposed to medications such as methimazole used in the management of hyperthyroidism, as

well as in cats with a recent history of blood transfusion. In addition, exposure to toxic agents must be carefully investigated through detailed environmental and clinical history-taking, with particular attention to substances such as acetaminophen that are known to induce oxidative erythrocyte injury. The identification of Heinz bodies on peripheral blood smears may support a diagnosis of toxin-induced haemolytic damage.

Metabolic and systemic disorders, including hepatic lipidosis, chronic kidney disease (CKD), and sepsis, should also be considered, as these conditions may either mimic the clinical manifestations of IMHA or occur concurrently, thereby complicating diagnosis and management. Furthermore, disorders associated with bone marrow suppression should be excluded, particularly in cases characterised by non-regenerative anaemia, as such findings may indicate underlying marrow pathology rather than peripheral erythrocyte destruction. Advanced diagnostic investigations, including thoracic and abdominal imaging as well as bone marrow aspiration or biopsy, may be warranted to identify neoplastic or paraneoplastic conditions when routine diagnostic evaluation fails to establish a cause. Despite extensive investigation, the underlying aetiology of IMHA often remains poorly defined. In such circumstances, a diagnosis of primary IMHA should only be considered after all identifiable secondary causes have been thoroughly excluded.

Treatment

Principles of Therapy

1. Supplementation of blood, blood products, and haematinics to maintain circulating erythrocyte mass and haematocrit.
2. Prevention of further complement-mediated erythrocyte destruction.
3. Treatment of the underlying aetiology, if identified.
4. Clinical stabilization of the patient.
5. Provision of supportive therapy to enhance recovery and minimize complications.

Blood Transfusion

Emergency blood transfusion is indicated in cats presenting with severe anaemia. Unlike in canines, feline blood groups include A, B, and AB, and compatibility testing is essential even for the first transfusion.

- Donor requirements: Healthy, vaccinated cats weighing >5 kg, free from ectoparasites and haemoparasites.
- Anticoagulant: Acid citrate phosphate.
- Constant (K) value: 66 (approximately 50-60 mL/kg body weight).
- Blood typing: Determination of A, B, or AB blood group is mandatory prior to transfusion**.**
- Prior to transfusion, if an allogenic feline donor is available, both major and minor cross-matching and the tri-drop agglutination test should be performed to check for clumping or agglutination.

Xenotransfusion may be considered only when no allogenic donor is available. However, compatibility testing remains critical, as xenotransfusion may exacerbate pre-existing IMHA. The use of xenotransfusion in cats is limited, and mean survival post-discharge is generally poor. Nonetheless, compatible blood can extend survival time in critical cases.

Immunosuppressive Therapy

Immunosuppressive therapy is the cornerstone of IMHA management. Corticosteroids and adjunct immune suppressants suppress immune activity, prevent further erythrocyte lysis, and reduce antibody production and macrophage activation. Although relapse is common, immunosuppression significantly reduces recurrence rates.

Continuous monitoring is necessary to assess efficacy and detect adverse effects**.**

12.3.1 Corticosteroids

- Prednisolone: 2 mg/kg q12h (Rosama et al., 2012).
- Duration: Up to 2–3 months; taper the dose gradually based on haematological improvement.
- Dexamethasone: 0.25–0.4 mg/kg q24h (recommended for hospitalized patients).
- Methylprednisolone acetate: Indicated when oral therapy is not tolerated.

Adjunct Immune suppressants

- Cyclosporine: 5 mg/kg q12h.
- Chlorambucil: 0.1–0.2 mg/kg q24h (or 2 mg/m² q48h).
- Mycophenolate mofetil: 10 mg/kg q12h.

Other immunosuppressive agents such as leflunomide and azathioprine are generally avoided in cats due to increased toxicity (though they are commonly used in dogs). Combination therapy with the above drugs has been reported, but its use remains limited.

Monitoring: Corticosteroids should be tapered according to haematological response. Continuous monitoring for relapse and steroid-induced hepatotoxicity is essential throughout therapy.

Antimicrobial Therapy

In cases of concurrent feline haemoplasmosis, administer: Doxycycline: 10 mg/kg q24h for 28 days, if clinical improvement is not observed or infection persists, follow with Marbofloxacin: 2 mg/kg q24h.

Supportive Therapy

- Iron sucrose should be avoided, as serum iron concentration is already elevated due to ongoing haemolysis.
- Blood products: Packed RBCs at 12–15 mL/kg via slow IV infusion.
- Anti-babesia drugs if immune trigger is due to *Babesia Sp*.
- Fluid therapy: Administer cautiously to avoid haemodilution.
- Oxygen therapy and antioxidants (e.g., acetylcysteine and vitamin E) may improve tissue oxygenation.
- Hepatoprotective agents: S-adenosylmethionine (SAM) and silymarin are indicated for hepatic dysfunction.
- Appetite stimulant: Mirtazapine at 1.88 mg/cat q48h may be used to encourage feeding.

Prognosis

The prognosis is generally guarded and largely depends on the underlying cause and the cat's response to therapy. Cases associated with lymphoma, severe systemic inflammatory response syndrome (SIRS), or multiple organ dysfunction typically carry a poor prognosis.

Prevention and Control

1. Avoid immune-triggering stress in susceptible or previously affected animals.
2. Exercise caution with drugs known to induce immune reactions; maintain close monitoring during therapy.
3. Conduct routine health evaluations, including haemoparasites screening and ectoparasite (flea and tick) control.
4. Administer only cat-approved formulations; avoid human medications.
5. Perform blood compatibility testing before every transfusion.
6. Maintain optimal shelter hygiene and environmental management to reduce infectious triggers.
7. Educate pet owners regarding the risks of over-the-counter medication use in cats.

Conclusion

Secondary immune-mediated haemolytic anaemia (IMHA) in cats is a clinically important

and often secondary condition associated with infectious, neoplastic, drug-related and immune-mediated triggers. The disease requires prompt recognition through combined clinical and laboratory evaluation, with the Coombs' test serving as a key confirmatory tool. Effective management depends on early initiation of corticosteroid-based immunosuppressive therapy, supportive care and identification of underlying causes. Long-term monitoring is essential due to the risk of relapse and timely intervention significantly improves survival and clinical outcomes in affected cats.

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