International Journal of Veterinary Sciences and Animal Husbandry 2025; 10(11): 201-206

ISSN: 2456-2912
NAAS Rating (2025): 4.61
VET 2025; 10(11): 201-206

www.veterinarypaper.com
Received: 18-09-2025
Accepted: 19-10-2025

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Immune-mediated hemolytic anemia in dogs and cats: Comparative pathophysiology, diagnosis and management

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DOI: https://www.doi.org/10.22271/veterinary.2025.v10.i11d.2724

Abstract
Immune-mediated hemolytic anemia (IMHA) is a severe and potentially fatal hematologic disorder in which the immune system targets and destroys the body's own red blood cells (RBCs), resulting in regenerative or nonregenerative anemia. The disease can occur as primary (idiopathic) or secondary, triggered by infections, neoplasia, drugs, or vaccines. This review aimed to summarize the current understanding of IMHA's etiology, pathogenesis, clinical presentation, diagnostic criteria, management, and prognosis in both dogs and cats. Information was compiled from recent studies, consensus statements, and case reports, including retrospective clinical analyses and molecular investigations. IMHA is primarily mediated by autoantibodies (IgG or IgM) that promote extravascular and/or intravascular RBC destruction through phagocytosis or complement activation. Clinically, affected animals exhibit pallor, icterus, tachycardia, splenomegaly, and in severe cases, hemoglobinuria. Diagnostic evaluation includes hematology, Coombs' testing, blood smear analysis, and molecular screening to differentiate idiopathic from secondary forms. Prognosis remains guarded, with mortality rates ranging from 18-44% within the initial months after diagnosis, primarily due to thromboembolic complications. Immunosuppressive therapy (glucocorticoids with or without adjunctive agents) forms the cornerstone of treatment, supported by blood transfusion and antithrombotic therapy. Secondary IMHA requires addressing the underlying cause, such as infection or neoplasia. Recent studies emphasize the role of genetic susceptibility, cytokine dysregulation, and complement pathways in disease progression. Future directions include the development of targeted immunotherapies, improved molecular diagnostics, and long-term monitoring tools for relapse prediction. Overall, early recognition, comprehensive diagnostic workup, and multimodal therapy significantly improve survival outcomes in dogs and cats with IMHA.

Keywords: Immune-mediated hemolytic anemia, dogs, cats, autoantibodies, hemotropic mycoplasma, immunosuppressive therapy, thromboembolism, veterinary hematology

1. Introduction
Definition and Classification
1.1 What is IMHA
Immune-mediated hemolytic anemia (IMHA) is a disorder in which the patient's immune system targets and destroys its own red blood cells (RBCs), producing a clinically important hemolytic anemia (Garden *et al*., 2019) [4]. IMHA may present acutely with severe anemia and systemic signs, or subacutely/chronically with more subtle clinical signs (Balch & Balch & Mackin, 2007) [2].

1.2 Primary (Idiopathic) versus Secondary IMHA
Primary (idiopathic) IMHA is defined when no underlying trigger can be identified and autoimmunity appears to be the principal disease process (Balch & Balch & Mackin, 2007) [2]

Secondary IMHA occurs when antibody-mediated RBC destruction is associated with an identifiable cause such as infectious agents, neoplasia, drugs, vaccines or other immune disorders (Garden *et al*., 2019) [4].

1.3 Types of immune-mediated RBC destruction (mechanistic classification)

IMHA in dogs is most commonly mediated by warm-reacting immunoglobulins (predominantly IgG), whereas cold-reacting antibodies (often IgM) are less common but can be clinically important (Balch & Balch & Mackin, 2007) [2]. RBC destruction may occur extravascularly (phagocytosis by splenic and hepatic macrophages producing spherocytes) or intravascularly (complement-mediated lysis with hemoglobinemia/hemoglobinuria), and some cases show mixed mechanisms (Garden *et al*., 2019) [4].

2. Epidemiology and Predisposing Factors

2.1 Species, Breed, Age and Sex Predilections

IMHA is an important cause of morbidity and mortality in dogs and occurs less commonly in cats (Garden *et al*., 2019) [4]. Certain dog breeds are overrepresented for IMHA, with English Cocker Spaniels, American Cocker Spaniels, Springer Spaniels, Poodles and Old English Sheepdogs commonly reported as predisposed (Balch & Mackin, 2007) [2]. Genetic associations have been demonstrated in some predisposed breeds, for example DLA and immune-regulatory gene polymorphisms in Cocker Spaniels, suggesting a heritable component to susceptibility. IMHA most often affects young adult to middle-aged dogs, though it can occur at any age; no strong, consistent sex predilection is universally reported, although some studies note a slight female predominance in certain cohorts (Todays Veterinary Practice, 2020).

2.2 Geographic and Seasonal Trends

Geographic differences in reported IMHA incidence are influenced by the regional prevalence of infectious triggers and vector-borne diseases, which can be associated with secondary IMHA (Garden *et al*., 2019) [4]. Some retrospective studies and clinical impressions suggest seasonal variation in IMHA presentations, possibly linked to seasonal vector activity and infection rates, but findings are not consistent across all regions (Garden *et al*., 2019) [4].

2.3 Risk Factors & Underlying Triggers (Secondary IMHA)

Secondary IMHA may be precipitated by infectious agents (for example Babesia, Mycoplasma/hemotropic organisms, Ehrlichia and other vector-borne pathogens), and therefore regions or seasons with higher vector activity may show more secondary cases (Garden *et al*., 2019) [4]. Drugs, vaccines and certain neoplasms have been implicated as triggers for secondary IMHA in individual case reports and series, and a careful drug and vaccine history is therefore important in every case (Garden *et al*., 2019) [4]. Concurrent immune-mediated diseases (e.g., immune thrombocytopenia or systemic autoimmune disease) and prior immune dysregulation are associated with increased risk of IMHA or with more complex, refractory disease courses (PLOS One gene expression / cohort studies). A recent clinical case report documented immune-mediated hemolytic anemia secondary to *Mycoplasma haemofelis* infection in a cat, where diagnosis was confirmed by PCR and a positive Coombs' test, and successful recovery was achieved with doxycycline, prednisolone, and supportive care (Abhijith *et al*., 2025) [1].

3. Pathophysiology

3.1 Immunologic mechanisms-autoantibodies and hypersensitivity

IMHA is classically a Type II hypersensitivity disorder in which autoantibodies bind to erythrocyte surface antigens, marking the cells for immune destruction (Balch & Mackin, 2007) [2].

Autoantibodies are most often IgG in warm-type IMHA, whereas IgM (and IgG + complement) are more typical in cold-reactive cases; IgM fixes complement more efficiently and therefore is more likely to produce intravascular hemolysis (Balch & Mackin, 2007) [2]. Antibody-RBC binding leads to Fc receptor-mediated phagocytosis by splenic and hepatic macrophages or to complement activation and direct lysis, depending on antibody isotype, affinity and complement involvement (Garden *et al*., 2019) [4].

3.2 Sites and mechanisms of RBC destruction-extravascular vs intravascular

Extravascular hemolysis occurs when opsonized erythrocytes are removed by macrophages in the spleen and liver, which frequently produces spherocytes on blood smear due to partial phagocytosis (Balch & Mackin, 2007) [2]. Intravascular hemolysis results from complement-mediated membrane attack and rupture of erythrocytes within the circulation, producing hemoglobinemia and hemoglobinuria in severe cases (Balch & Mackin, 2007) [2]. Many clinical cases show mixed mechanisms (both extravascular and intravascular components), and the dominant pathway influences clinical signs and laboratory findings (Garden *et al*., 2019) [4].

3.3 Complement, immune complexes and secondary amplification

Complement activation (classical pathway) amplifies erythrocyte destruction when C3b/C3d bind to RBCs and promote phagocytosis or membrane attack complex formation (MSD Manual; Balch & Mackin, 2007) [2]. Immune complexes and circulating free hemoglobin can further stimulate inflammation and contribute to downstream organ effects such as endothelial activation and prothrombotic changes (Garden *et al*., 2019) [4].

3.4 Bone marrow response and erythropoiesis

If marrow function is intact, hemolysis typically induces a regenerative response with increased reticulocytosis and erythroid hyperplasia in the marrow (Garden *et al*., 2019) [4]. Severe or prolonged immune attack can sometimes target erythroid precursors (pure red cell aplasia or erythroid hypoplasia), producing a nonregenerative anemia despite peripheral destruction (Balch & Mackin, 2007) [2].

3.5 Systemic consequences: Inflammation, hypercoagulability and organ injury

Massive hemolysis releases hemoglobin and other intracellular contents that can scavenge nitric oxide, promote vasoconstriction, and contribute to endothelial dysfunction (Garden *et al*., 2019) [4]. Dogs with IMHA commonly develop a prothrombotic state; microvascular thrombosis and clinically important thromboembolic disease (for example pulmonary thromboembolism) are recognized major

complications and important determinants of outcome (Garden *et al*., 2019) [4].

4. Clinical Signs and Presentation

4.1 History and onset

IMHA can develop acutely with sudden collapse and life-threatening anemia, or subacutely with progressive lethargy and weakness over days to weeks (Balch & Mackin, 2007) [2]. Owners often report nonspecific signs such as inappetence, exercise intolerance, or decreased activity prior to overt anemia (Garden *et al*., 2019) [4]. In cats, secondary causes are more common, so the presenting complaint may reflect the underlying infectious or neoplastic disease rather than anemia alone (Kohn *et al*., 2006) [6].

4.2 General clinical signs

Pale mucous membranes are a hallmark finding, reflecting reduced oxygen-carrying capacity due to anemia (Balch & Mackin, 2007) [2]. Icterus is frequently observed because extravascular hemolysis leads to increased bilirubin production from heme breakdown (Garden *et al*., 2019) [4]. Many dogs show tachycardia, tachypnea, and bounding pulses as compensatory responses to anemia (Balch & Mackin, 2007) [2]. Lethargy, collapse, and anorexia are common nonspecific systemic manifestations (Kohn *et al*., 2006) [6].

4.3 Specific physical examination findings

Splenomegaly and hepatomegaly may occur secondary to increased erythrophagocytosis and extramedullary haematopoiesis (Balch & Mackin, 2007) [2]. Fever is reported in a subset of cases and may reflect cytokine release or concurrent infection (Garden *et al*., 2019) [4]. Hemoglobinuria can occur in intravascular hemolysis, producing dark red to brown urine and sometimes leading to renal compromise (Kohn *et al*., 2006) [6]. A systolic heart murmur may be auscultated due to anemia-related high-output circulation (Balch & Mackin, 2007) [2].

4.4 Cat-specific considerations

IMHA is less common in cats than in dogs, and when present is usually secondary to FeLV, hemotropic Mycoplasma infection, or neoplasia (Kohn *et al*., 2006) [6]. Cats often show less dramatic regenerative responses and less consistent icterus compared with dogs, making clinical recognition more challenging (Garden *et al*., 2019) [4]. Clinical signs may overlap with those of the underlying trigger, so diagnostic workup is especially important in feline cases (Kohn *et al*., 2006) [6]. Clinical signs such as pallor, icterus, lethargy, tachycardia, and splenomegaly were observed in a cat with IMHA secondary to haemotropic mycoplasmosis, highlighting the overlap between infectious and immune-mediated pathology (Abhijith *et al*., 2025) [1].

5. Diagnostics

5.1 Hematology and blood smear findings

Most dogs with IMHA present with a moderate to severe regenerative anemia, characterized by polychromasia, anisocytosis, and reticulocytosis (Balch & Mackin, 2007) [2]. Spherocytosis, due to partial phagocytosis of RBC membranes by macrophages, is a key morphological feature supportive of IMHA in dogs (Garden *et al*., 2019) [4]. Leukocytosis, often neutrophilia with a left shift, is frequently reported and reflects inflammatory cytokine release (Kohn *et al*., 2006) [6]. Thrombocytopenia may occur concurrently, either from a consumptive process or as part of Evans' syndrome (IMHA with immune thrombocytopenia). (Balch & Mackin, 2007) [2]. Laboratory findings in this report included anemia, spherocytosis, reticulocytosis, leukocytosis with neutrophilia, and hyperbilirubinemia, which collectively supported a diagnosis of secondary IMHA (Abhijith *et al*., 2025) [1].

5.2 Biochemical and urinalysis abnormalities

Hyperbilirubinemia and bilirubinuria are common due to increased heme catabolism in extravascular hemolysis (Garden *et al*., 2019) [4]. Intravascular hemolysis leads to hemoglobinemia and hemoglobinuria, which can contribute to renal tubular injury (Balch & Mackin, 2007) [2]. Mild increases in liver enzymes are sometimes seen, often secondary to hypoxia or hepatic congestion (Kohn *et al*., 2006) [6]. Azotemia may develop from hypoxic renal injury or concurrent conditions. (Balch & Mackin, 2007) [2].

5.3 Coombs' test and autoagglutination

The direct antiglobulin (Coombs') test detects antibodies or complement bound to erythrocytes and supports a diagnosis of IMHA when positive (Balch & Mackin, 2007) [2]. False negatives can occur if antibody levels are low or if immunosuppressive therapy has already been initiated. (Garden *et al*., 2019) [4]. The saline agglutination test (SAT) is a rapid, in-clinic test that detects persistent RBC agglutination after dilution with saline, and a positive result is strongly supportive of IMHA (Kohn *et al*., 2006) [6]. Accurate diagnosis using a combination of blood smear, PCR and Coombs test is critical (Willi *et al*., 2007; Tasker, 2010) [11, 10]. Coomb's-positive results in many affected cats, indicating the presence of erythrocyte bound antibodies (Hartmann *et al*., 2015) [5]. The clearance of these antibody-coated erythrocytes is primarily facilitated by splenic macrophages through extravascular haemolysis (Tasker, 2010; Messick, 2003) [10, 7].

5.4 Advanced and confirmatory methods

Flow cytometry can identify antibody-coated erythrocytes with high sensitivity and specificity, but it is not widely available in general practice (Garden *et al*., 2019) [4].

PCR panels for vector-borne pathogens are useful in ruling out secondary infectious causes, especially in endemic regions. (Kohn *et al*., 2006) [6]. Bone marrow aspiration may reveal erythroid hyperplasia in regenerative cases or selective erythroid hypoplasia in precursor-targeted IMHA (Balch & Mackin, 2007) [2].

5.5 Screening for secondary causes

Because secondary IMHA is common, especially in cats, diagnostic workup should include infectious disease testing (FeLV, FIV, hemotropic Mycoplasma, Babesia, Ehrlichia) (Garden *et al*., 2019) [4]. Imaging such as thoracic radiographs and abdominal ultrasound may help identify neoplasia or systemic disease that could trigger secondary IMHA (Kohn *et al*., 2006) [6].

Drug and vaccination history is essential to identify possible iatrogenic causes. (Balch & Mackin, 2007) [2].

6. Complications and Prognostic Indicators

6.1 Major complications

The most clinically important complication of IMHA in dogs is thromboembolism, particularly pulmonary thromboembolism, which is a leading cause of death (Garden *et al*., 2019) [4]. Thrombosis is driven by systemic inflammation, endothelial activation, and procoagulant

changes associated with hemolysis (Balch & Mackin, 2007) [2]. Other complications include disseminated intravascular coagulation (DIC), which may manifest as petechiae, ecchymoses, or uncontrolled bleeding (Kohn *et al*., 2006) [6]. Acute kidney injury can occur as a result of hemoglobinuria in intravascular hemolysis or secondary to hypoxia (Balch & Mackin, 2007) [2]. Hepatic dysfunction, pancreatitis, and secondary infections due to immunosuppression are additional risks in severe or chronic cases. (Kohn *et al*., 2006) [6].

6.2 Prognostic indicators in dogs

Hyperbilirubinemia has repeatedly been shown as a negative prognostic indicator, correlating with both severity of hemolysis and mortality risk (Duclos *et al*., 2024) [3]. Concurrent thrombocytopenia is associated with worse survival, especially when combined with anemia (Evans' syndrome), (Duclos *et al*., 2024) [3]. High illness severity scores, such as those derived from the Canine Hemolytic Anemia Objective Score (CHAOS), predict increased mortality risk. (Garden *et al*., 2019) [4]. Dogs with intravascular hemolysis or autoagglutination at presentation tend to have more severe disease and poorer outcomes (Balch & Mackin, 2007) [2].

6.3 Prognostic indicators in cats

Idiopathic IMHA is rare in cats, and outcomes largely depend on whether an underlying cause (such as FeLV or hemotropic Mycoplasma infection) can be identified and treated. (Kohn *et al*., 2006) [6]. Cats with secondary IMHA due to neoplasia or chronic systemic illness typically have a guarded to poor prognosis. (Garden *et al*., 2019) [4]. As in dogs, regenerative responses in cats are variable, and nonregenerative anemia at presentation is associated with a worse prognosis (Kohn *et al*., 2006) [6].

6.4 Survival statistics

Reported mortality rates in canine IMHA range from 18-44% within the first 1-2 months after diagnosis, reflecting high risk of early death from anemia or thrombosis (Duclos *et al*., 2024) [3].

In long-term survivors beyond 3 months, prognosis improves substantially, with median survival times exceeding several years in some studies (Duclos *et al*., 2024) [3].

7. Treatment and Management

7.1 General therapeutic goals

The mainstay of IMHA treatment is suppression of the immune-mediated destruction of red blood cells while providing supportive care to stabilize the patient (Balch & Mackin, 2007) [2]. Therapy must also address complications such as thromboembolism and provide treatment for any identified underlying cause in secondary IMHA (Garden *et al*., 2019) [4].

Supportive care includes oxygen therapy, fluid therapy, and nutritional support in severely affected patients. (Kohn *et al*., 2006) [6].

7.2 Immunosuppressive therapy

Glucocorticoids, primarily prednisone or prednisolone, are the first-line immunosuppressive drugs, reducing macrophage phagocytosis of antibody-coated RBCs and suppressing autoantibody production (Balch & Mackin, 2007) [2]. If response to corticosteroids alone is inadequate, adjunctive immunosuppressants such as azathioprine, cyclosporine, mycophenolate mofetil, or leflunomide may be added (Garden *et al*., 2019) [4]. In cats, azathioprine is generally avoided due to hepatotoxicity and myelotoxicity, with chlorambucil considered safer. (Kohn *et al*., 2006) [6]. Early combination immunosuppression is sometimes used in severe cases to hasten remission (Garden *et al*., 2019) [4]. Early antimicrobial and immunosuppressive therapy can achieve favourable outcomes (Hartmann *et al.*, 2015; Sykes, 2014) [5].

7.3 Anti-thrombotic therapy

Given the high risk of thromboembolism, antithrombotic therapy is a cornerstone of IMHA management (Garden *et al*., 2019) [4]. Low-dose aspirin, clopidogrel, and unfractionated or low molecular weight heparin are commonly used agents to reduce platelet activation and clot formation (Duclos *et al*., 2024) [3]. Clopidogrel has been shown to be more effective than aspirin in preventing platelet aggregation in dogs (Garden *et al*., 2019) [4].

7.4 Blood transfusion

Packed red blood cell transfusions are often necessary in cases of severe or life-threatening anemia (Balch & Mackin, 2007) [2]. Cross-matching should be performed before transfusion, especially in cats, due to naturally occurring alloantibodies (Kohn *et al*., 2006) [6]. Repeated transfusions may be required but carry the risk of sensitization and transfusion reactions, particularly in immune-mediated disease (Garden *et al*., 2019) [4].

7.5 Adjunctive and alternative therapies

Intravenous immunoglobulin (IVIG) has been used in refractory cases to block Fc receptor-mediated phagocytosis of RBCs (Balch & Mackin, 2007) [2]. Plasmapheresis may be considered in specialized centers for fulminant, treatment-resistant IMHA (Garden *et al*., 2019) [4]. Erythropoiesis-stimulating agents may support red cell production in nonregenerative cases, though their use remains controversial (Kohn *et al*., 2006) [6].

7.6 Management of secondary IMHA

In secondary cases, identification and removal of the underlying trigger (infection, neoplasia, drug exposure) is critical for successful management (Kohn *et al*., 2006) [6].

Antimicrobial therapy is warranted when hemotropic Mycoplasma or other infectious triggers are identified (Garden *et al*., 2019) [4]. Oncologic therapy may be necessary in neoplastic-associated IMHA, though prognosis remains guarded (Balch & Mackin, 2007) [2].

8. Monitoring and Follow-up

8.1 In-hospital monitoring

Close monitoring is critical during the acute phase of IMHA because rapid deterioration can occur (Garden *et al*., 2019) [4]. Packed cell volume (PCV), total solids, and bilirubin levels should be checked at least daily in hospitalized patients (Balch & Mackin, 2007) [2]. Monitoring for thromboembolic events-such as sudden dyspnoea or limb pain is essential, as early detection improves prognosis (Duclos *et al*., 2024) [3].

8.2 Outpatient follow-up

After discharge, frequent re-evaluation (every 3-7 days initially) is required to assess PCV, reticulocyte counts, and serum biochemistry. (Balch & Mackin, 2007) [2]. As the patient stabilizes, monitoring intervals can be extended, but relapses remain a possibility for months to years. (Garden *et al*., 2019) [4]. Owners should be instructed to watch for pale

mucous membranes, jaundice, lethargy, or sudden collapse at home. (Kohn *et al*., 2006) [6].

8.3 Monitoring drug effects and side effects
Corticosteroid therapy can cause adverse effects such as polyuria, polydipsia, polyphagia, muscle wasting, and gastrointestinal ulceration. (Balch & Mackin, 2007) [2]. Azathioprine may induce hepatotoxicity or myelosuppression, requiring routine liver enzyme panels and CBC monitoring. (Garden *et al*., 2019) [4]. Cyclosporine blood levels may need to be monitored to optimize immunosuppressive effect and minimize toxicity (Kohn *et al*., 2006) [6].

8.4 Criteria for tapering therapy
Glucocorticoids and other immunosuppressants should be tapered gradually once remission are confirmed, typically over 3-6 months. (Garden *et al*., 2019) [4]. Tapering too quickly increases the risk of relapse, while prolonged use predisposes to adverse drug effects. (Balch & Mackin, 2007) [2]. Sustained remission is generally defined as stable PCV and reticulocyte counts without transfusion support for several weeks (Duclos *et al*., 2024) [3].

9. Prognosis and Outcomes
9.1 General outlook
The prognosis for IMHA in dogs and cats is guarded, with high short-term mortality but potential for long-term survival in responders. (Garden *et al*., 2019) [4]. Early mortality is most often due to severe anemia or thromboembolic complications. (Balch & Mackin, 2007) [2]. In cats, prognosis depends heavily on identification and management of underlying causes, as idiopathic IMHA is rare (Kohn *et al*., 2006) [6].

9.2 Reported survival rates in dogs
Overall survival to discharge ranges from 55-80% in dogs, depending on severity and treatment protocols (Duclos *et al*., 2024) [3]. One-to two-month mortality rates are often reported between 20-45%. (Garden *et al*., 2019) [4]. Dogs surviving the initial crisis period have a good chance of long-term survival, with many living several years after diagnosis. (Duclos *et al*., 2024) [3].

9.3 Prognostic markers
Hyperbilirubinemia and autoagglutination at presentation predict worse outcomes. (Duclos *et al*., 2024) [3]. Concurrent thrombocytopenia, especially when severe, is a poor prognostic indicator. (Balch & Mackin, 2007) [2]. Response to initial immunosuppressive therapy within 7-10 days correlates with higher survival. (Garden *et al*., 2019) [4].

9.4 Outcomes in cats
Cats with IMHA generally have a less favorable prognosis than dogs because secondary causes, such as FeLV infection or neoplasia, often carry poor outcomes. (Kohn *et al*., 2006) [6]. Those with infectious causes like hemotropic Mycoplasma may respond well to antimicrobial therapy combined with immunosuppression. (Garden *et al*., 2019) [4]. Idiopathic feline IMHA, when confirmed, can sometimes achieve remission similar to canine cases. (Kohn *et al*., 2006) [6].

9.5 Relapse and long-term outcomes
Relapses are common, occurring in up to 30-40% of canine cases, particularly during drug tapering (Balch & Mackin, 2007) [2]. Long-term survival requires careful follow-up, individualized immunosuppressive tapering, and management of relapses. (Garden *et al*., 2019) [4]. Cats may relapse if the underlying cause is not eliminated or if immunosuppression is withdrawn too soon (Kohn *et al*., 2006) [6].

10. Future Directions and Research Needs
10.1 Improved understanding of pathogenesis
Further research is needed to clarify genetic and molecular mechanisms that predispose certain breeds or individuals to IMHA. (Garden *et al*., 2019) [4]. Identification of specific autoantigens and immune dysregulation pathways could allow for more targeted therapies. (Balch & Mackin, 2007) [2]. In cats, understanding why secondary IMHA is far more common than idiopathic disease may provide insights into species-specific immune regulation. (Kohn *et al*., 2006) [6].

10.2 Advances in diagnostics
There is a need for more sensitive and specific diagnostic tests that can differentiate IMHA from other causes of anemia and identify early disease (Garden *et al*., 2019) [4]. Biomarkers of immune activation, hemolysis, and thrombosis could improve prognostication and guide therapy. (Duclos *et al*., 2024) [3]. Point-of-care molecular testing for infectious triggers in cats could speed diagnosis and treatment. (Kohn *et al*., 2006) [6]. Strandberg *et al.* (2023) [8] reported the first case of haemophagocytic syndrome in a cat infected with *Mycoplasma haemofelis*, diagnosed via PCR despite no organisms visible cytologically.

10.3 Novel therapeutic strategies
Development of targeted immunomodulators, such as monoclonal antibodies or cytokine inhibitors, may provide alternatives to broad immunosuppression (Garden *et al*., 2019) [4].
Safer and more effective antithrombotic strategies are needed to reduce mortality from thromboembolism (Duclos *et al*., 2024) [3]. Gene therapy and personalized medicine approaches could eventually be applied in predisposed breeds (Balch & Mackin, 2007) [2].

10.4 Long-term disease monitoring
Longitudinal studies are required to evaluate relapse rates, optimal treatment durations, and predictors of sustained remission. (Garden *et al*., 2019) [4]. Improved monitoring tools for drug toxicity, especially with long-term immunosuppression, are essential for enhancing quality of life. (Kohn *et al*., 2006) [6]. Studies on client education and home-monitoring strategies could also contribute to earlier detection of relapses (Balch & Mackin, 2007) [2].

11. Conclusion
Immune-mediated hemolytic anemia (IMHA) is one of the most common and life-threatening immune-mediated disorders in dogs, and though less common in cats, it remains clinically significant. The disease arises from immune destruction of red blood cells, often through autoantibody binding and complement activation. It may be primary (idiopathic) or secondary to infections, neoplasia, drugs, or vaccines. Clinical presentation is variable, but anemia, icterus, and lethargy are frequent features, with cats often showing subtler signs. Diagnosis requires a combination of hematology, biochemical tests, and immunological assays, supported by exclusion of secondary causes. Major complications such as thromboembolism, disseminated intravascular coagulation, and organ dysfunction strongly

influence outcomes. Management relies on immunosuppressive drugs, antithrombotic therapy, blood transfusions, and treatment of underlying causes in secondary IMHA. Prognosis is guarded, with significant early mortality, although long-term survival is possible in patients that respond well to therapy. Monitoring is essential for assessing remission, preventing relapse, and detecting adverse drug effects. Future research focusing on pathogenesis, novel therapeutics, and improved diagnostic and prognostic tools will enhance management and survival outcomes for both dogs and cats with IMHA.

Conflict of Interest
Not available

Financial Support
Not available

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How to Cite This Article
Abhijith SP, Apoorva HJ, Kalam AA, Chandrakumar D, Sanjay HV. Immune-mediated hemolytic anemia in dogs and cats: Comparative pathophysiology, diagnosis and management. International Journal of Veterinary Sciences and Animal Husbandry. 2025;10(11):201-206.