Regulatory Mechanisms in Biosystems

ISSN 2519-8521 (Print)
ISSN 2520-2588 (Online)
Regul. Mech. Biosyst.

Clinical and morphological changes associated with eosinophilic syndrome in cats

O. Piddubnyak*, N. Vovkotrub*, A. Kharchenko*, B. Bilyk*,
V. Hrytsai*, O. Bevz*, O. Yeroshenko*, N. Suslova**

** Bila Tserkva National Agrarian University, Bila Tserkva, Ukraine*
*** Dnipro State Agrarian and Economic University, Dnipro, Ukraine*

Article info

Received 18.03.2026
Received in revised form 22.04.2026
Accepted 16.05.2026

Bila Tserkva National Agrarian University, Bila Tserkva, 09100, Kyiv Oblast, Ukraine. Tel.: +38-067-407-97-91. E-mail: ovpiddubnyak73@ukr.net

Dnipro State Agrarian and Economic University, Serhii Efremov st., 25, Dnipro, 49600, Ukraine. Tel. +38-050-908-98-45. E-mail: suslova@ua.fm

Piddubnyak, O., Vovkotrub, N., Kharchenko, A., Bilyk, B., Hrytsai, V., Bevz, O., Yeroshenko, O., & Suslova, N. (2026). Clinical and morphological changes associated with eosinophilic syndrome in cats. Regulatory Mechanisms in Biosystems, 17(3), e26054. doi:10.15421/0226054

Eosinophilic syndrome in domestic cats (eosinophilic granuloma complex – EGC) has a complicated pathogenesis, and its manifestations significantly depend on the individual's sensitivity, the type and multifactorial nature of the allergen, as well as the severity of the chronic condition. This study presents the results of a comprehensive analysis of the clinical, haematological, and cytological features of eosinophilic syndrome in cats with eosinophilic granuloma complex. The course of various clinical forms of this pathology was studied, which depends on the duration of the disease, the time taken for the pet owner to consult a veterinarian and receive treatment, and the identification of potential etiological factors; a morphological blood analysis was conducted, and lesions on the skin and mucous membranes were examined. It was established that clinical symptoms ranged from localised granulomatous lesions on the lips, in the oral cavity to generalised skin and mucous membrane lesions with the development of eosinophilic gastroenteritis, accompanied by appropriate changes in the white blood cell formula: from mild eosinophilia to hypereosinophilia (8–59%). Cytological examination revealed a prevalence of eosinophils, the presence of degenerated collagen fibres, frothy macrophages and fibroblasts, with no signs of microorganisms. Numerous neutrophils, foamy macrophages with phagocytosed collagen fragments, and small lymphocytes indicated active inflammation, tissue degradation, and chronicity of the lesion. Fibrocytes and isolated fibroblasts reflected tissue remodelling, while fragmented collagen fibres suggested destruction associated with eosinophil degranulation. Haematological analysis showed an increase in the number of erythrocytes and leukocytes, as well as in haemoglobin levels, in the affected cats. The findings confirm the allergic etiology of feline eosinophilic granuloma complex and the importance of a comprehensive diagnostic approach comprising clinical, haematological, and cytological methods.

Keywords: cats; eosinophilic granuloma complex; eosinophilic ulcer; cytological examination; eosinophilia; allergic disorders.

Introduction

Due to the growth in the domestic cat population and owners' increased focus on their pets' health, the issue of timely diagnosis and effective treatment of eosinophilic syndrome has become particularly important (Miller et al., 2013; Paterson, 2016). The relevance of research into eosinophilic granulomatosis in domestic cats is due to its high prevalence, the diversity of clinical manifestations, and the complexity of differential diagnosis (Scott & Miller, 2012; Ehlers et al., 2019; Hopke & Sargent, 2019). The presence of symptoms similar to those of other skin diseases, such as bacterial dermatitis, fungal infections or autoimmune processes, complicates the timely determination of the correct diagnosis (Dokuzeylul et al., 2016; Falcão et al., 2020).

Furthermore, the absence of a specific etiological factor requires an individualised approach to treatment, which involves not only eliminating clinical manifestations but also identifying and addressing the underlying cause of this condition (Buckley & Nuttall, 2012; Miller et al., 2023). Improving diagnostic and therapeutic methods for eosinophilic granuloma is a key challenge in modern veterinary medicine, given the need to enhance treatment efficacy, prevent relapses and increase the quality of life for animals (Miller et al., 2023).

The relevance of the study is also driven by the need to raise awareness among veterinary specialists regarding the characteristics of the course and treatment of eosinophilic granuloma, which will help prevent the condition from progressing to a chronic stage, the development of secondary infections, and a deterioration in the animal's quality of life. Moreover, the systematisation of clinical data and the exploration of new therapeutic approaches will contribute to the refinement of diagnostic algorithms and treatment protocols (Bryan et al., 2010; Omelchenko et al., 2023).

Eosinophilic syndrome in cats, also known as eosinophilic granuloma complex (EGC), is a heterogeneous group of dermatoses characterised by tissue infiltration by eosinophils – cells responsible for allergic reactions and the immune response. This complex typically includes the following clinical forms: eosinophilic granuloma, eosinophilic plaque and eosinophilic ulcer ('Rodin's ulcer') (Bloom, 2006; Oliveira & Van den Broek, 2006). The pathology has a chronic course and may be complicated by lesions affecting both the skin and the mucous membranes (Fondati et al., 2001; Vogelnest, 2017).

ECG is a common problem in clinical veterinary dermatology, particularly among young animals, with females affected slightly more frequently than males (Scott & Miller, 2012). The etio-pathogenesis of the disease has not yet been definitively established, although most researchers associate it with hypersensitivity to external and internal allergens, particularly flea and mosquito bites, food allergens and inhalant irritants (Hobi et al., 2011; Diesel, 2017). Diagnosis of the syndrome is based on a comprehensive approach, which includes assessment of the clinical pattern, cytological and histological examinations, and the exclusion of other pathologies (abdominal ultrasound) (Foster, 2003).

Therefore, the aim of the study is to investigate the characteristics of the clinical forms of eosinophilic syndrome in domestic cats, identifying etiological factors and analysing changes in the blood morphology profile and lesions of the skin and mucous membranes in animals with eosinophilic granuloma.

Material and methods

During the experiments, compliance with the requirements of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1986), the General Ethical Principles of Performing Research in Animals approved by the National Congress on Bioethics (Kyiv, 2001), the provisions of the Law of Ukraine "On the Protection of Animals from

Cruelty" (2006), as well as the provisions of the European Union Directive 2010/63/EU dated September 22, 2010, was ensured. The research program was approved by the Local Ethics Committee of the Bila Tserkva National Agrarian University (conclusion 21/21 dated 08/28/25) and written consent was obtained from the animal owners.

The study group was formed by domestic cats (n = 12) who were admitted to the veterinary clinic of the Faculty of Veterinary Medicine of the Bila Tserkva National University. All animals underwent a comprehensive clinical examination, which included: collection of anamnesis data, a complete general examination, laboratory blood tests (complete blood count, serum biochemical profile), dermatological examination, material selection using scrapings and swabs from the skin and mucous membranes for cytological analysis, testing for the presence of ectoparasites (fleas, ticks), exclusion of fungal, bacterial and viral infections using appropriate methods (PCR, cultures, Wood's lamp, etc.). Elimination diets and hypersensitivity tests were used to determine the allergic component. The animals coming to the clinic had varying degrees of disease severity: some had a mild condition, some were moderate, and others had a severe form of the disease. The mild form was characterised by lesions found only on the lips; in the moderate form, ulcers were diagnosed on the lips and in the oral cavity. Animals with severe disease showed signs of lesions affecting the coat, the skin of the head, neck and trunk, visible mucous membranes, as well as gastrointestinal dysfunction. According to the principle of analogs, a control group consisting of clinically healthy domestic cats (n = 10) was formed.

Anamnesis revealed that the animals (8 female cats and 4 male cats) belonged to different breeds, including British Shorthair (50%), Maine Coon, Sphynx, and Scottish Fold (25% each), and ranged in age from 6 months to 9 years. According to the owners, the diet of 66.7% of the cats consisted of home-prepared food (boiled poultry and beef meat, by-products, dairy products, and fish). In addition, the animals were frequently fed processed meat products and table scraps. Only 33.3% of the cats consumed diets based on commercially prepared dry feed.

Blood samples were collected from the lateral brachial vein into vacuum tubes containing a stabilizer. The total number of leukocytes, erythrocytes, haemoglobin content, haematocrit, and 'red blood cell' indices – mean corpuscular haemoglobin (MCH) and mean corpuscular volume (MCV) – were determined using the Mindray BC-2800 Vet haematology analyser (Mindray, China, 2016). Leukograms were evaluated by counting 100 cells in stained blood smears using light microscopy at ×1000 magnification. Rapid staining of blood smears and tissue imprint smears was performed using LEUCODIF 200 (CZ) stains (Erba Lachema s.r.o., Czech Republic).

Sample preparation and determination of the respective parameters were performed according to the manufacturer's instructions for the analyzer and reagents. Statistical analysis of the obtained results was performed using Statistica 10 software (StatSoft Inc., USA). Differences between mean values were assessed using Tukey's test, and differences were considered statistically significant at $P < 0.05$ for all data (Petrovska et al., 2022).

Results

A clinical examination revealed that 33.3% of diseased cats had swellings with dense white or yellow nodules in the upper lip area, rarely in the oral cavity (Fig. 1). They were firm to the touch and up to 2 mm in diameter. The general condition of the animals in the study was good, and their appetite was unaffected. According to the pet owners, a 'blister' on the upper or lower lip appeared in cats within 2–3 days and immediately caused distress to the animals. They constantly scratched it with their paws, licked it, or rubbed their snouts on surrounding objects. During morphological blood analysis, the cats showed mild eosinophilia (8–11%, compared to a normal range of 2–7). Clinical examination showed that 41.7% of cats had unilateral or bilateral ulcers on the upper lip, which were clearly demarcated and coloured from yellowish-brown to bright red (Figs. 2, 3).

During the examination, the diseased cats attempted to avoid being manipulated. Generally, the ulcers on the mucous membranes did not bleed, but if they were damaged, minor bleeding, hypersalivation, halitosis, pain, and itching were observed on palpation. During an oral examination, lesions were observed on the mucous membranes of the lips, cheeks, gums, tongue, palate and pharynx, appearing as petal-like formations that impeded swallowing and the flow of food (Fig. 4).

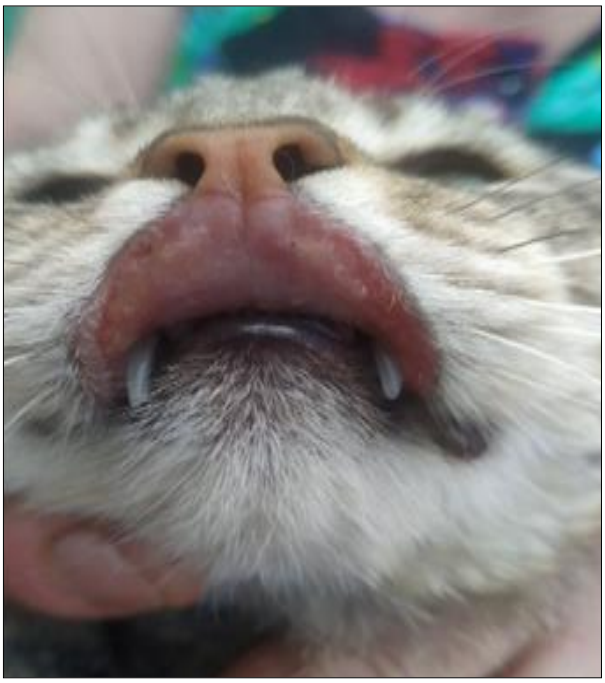

Fig. 1. Bilateral eosinophilic granuloma on the upper lip in a cat

Fig. 2. Unilateral eosinophilic plaque on the upper lip in a cat

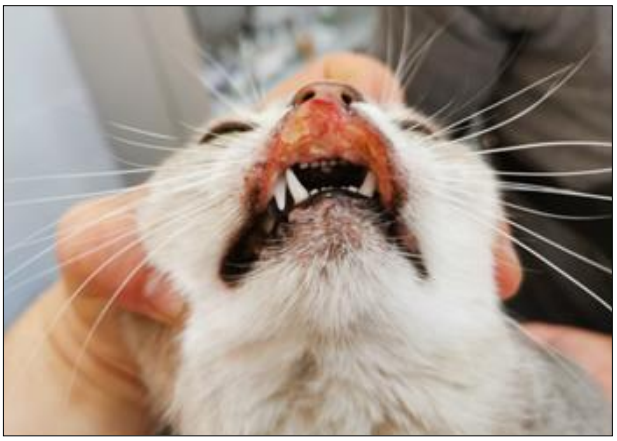

Fig. 3. Bilateral eosinophilic ulcer on the upper lip in a cat

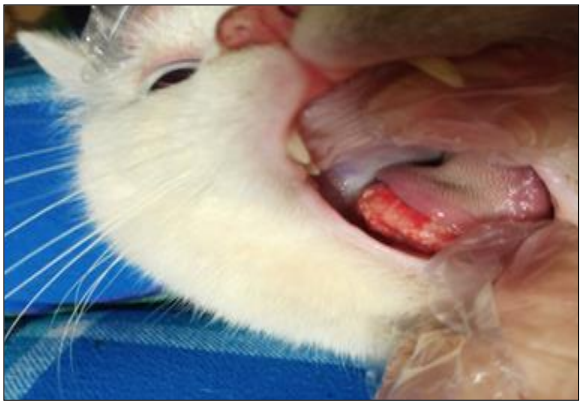

Fig. 4. Eosinophilic granuloma in the oral cavity in a cat

The owners noted that the cats had become lethargic and inactive, found it much more difficult to eat dry food, and most of them refused it. In the sick cats, body temperature, defecation and urination were in the normal range; no abdominal pain was noted on palpation, and peristalsis was sufficient. A leukogram analysis revealed moderate eosinophilia (12–21%).

The other 25% of cats were diagnosed with a generalised form of eosinophilic syndrome, characterised not only by the formation of ulcers on the lips and in the oral cavity, but also by plaques and ulcers on the head and body (Figs. 5–9). The lesions appeared as thick, red or dark pink crusts on hairless areas, which the animals constantly licked. These animals exhibited a reduced appetite, vomiting after feeding, and semi-liquid, unformed faeces. Hypereosinophilia (22–59%) was detected in the blood of the diseased cats.

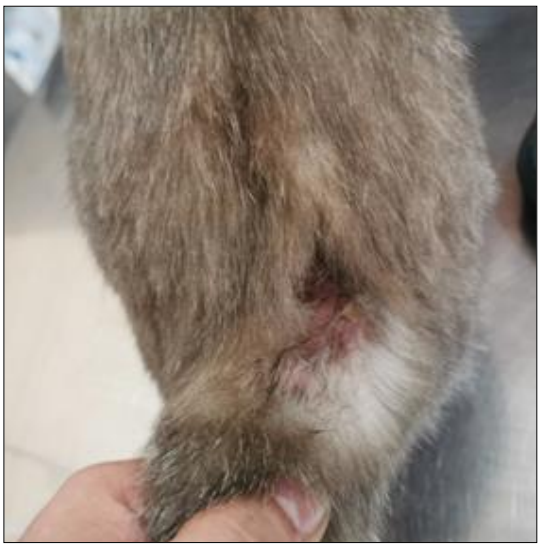

Fig. 5. Eosinophilic plaque at the base of the tail in a cat

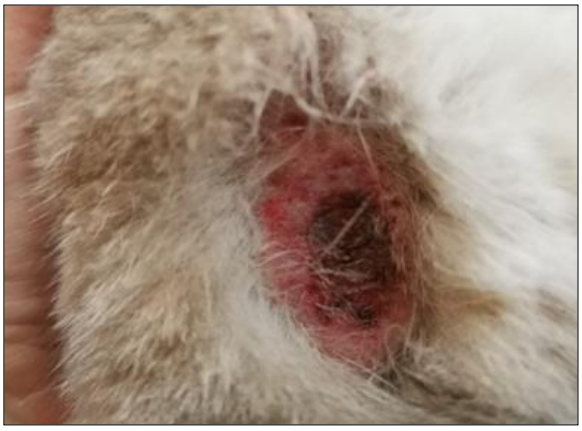

Fig. 6. Eosinophilic ulcer in the back area of a cat

Fig. 7. Eosinophilic ulcerative lesions on neck of a cat

Fig. 8. Eosinophilic ulcerative lesions on facial region

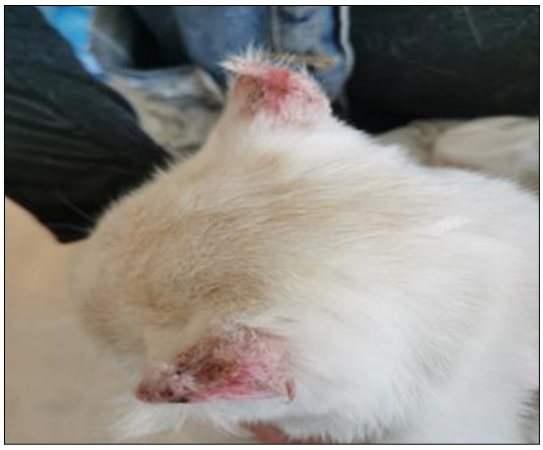

Fig. 9. Eosinophilic ulcerative lesions on the ears of a cat

The impression smears showed a predominance of hypercellular inflammatory cells, accompanied by areas of fibrous collagenous stroma with signs of degeneration, cholesterol crystals, eosinophil granules and granular protein debris. In stained preparations, the dominant cell population consisted of eosinophils, which had well-defined bright red granules, sometimes partially degranulated. Eosinophils were found both singly and in clusters. A significant population of non-degenerative neutrophilic granulocytes was visualised, with clear visualisation of nuclear segments. At the same time, a significant proportion of degenerative neutrophils with swollen, indistinct nuclear contours was detected, indicating areas of tissue degradation in this area. Active foamy macrophages were also identified – large, round cells with an oval nucleus of peripheral or central topography and

vacuolated cytoplasm, within which phagocytosed eosinophilic granules and fragments of degraded collagen were visualised, indicating active phagocytic activity. Isolated small lymphocytes were detected in impression smears, indicating the development of a chronic lesion. Cytological examination of samples from the affected areas revealed the presence of fibroblastic cells of a spindle-shaped, elongated form with rod- and cigar-shaped nuclei (mature fibrocytes) and isolated fibroblasts. The presence of fibroblastic cells in the collagenous stroma indicates tissue remodelling and the progression of a chronic process. The collagen fibres in the smear were unevenly stained blue-violet, had irregular, frayed edges and substantial fragmentation, which is evidently associated with the effect of major basophil granule protein (MBP), which during degranulation destroys collagen fibres and cell membranes, followed by the release of cholesterol crystals (Fig. 10).

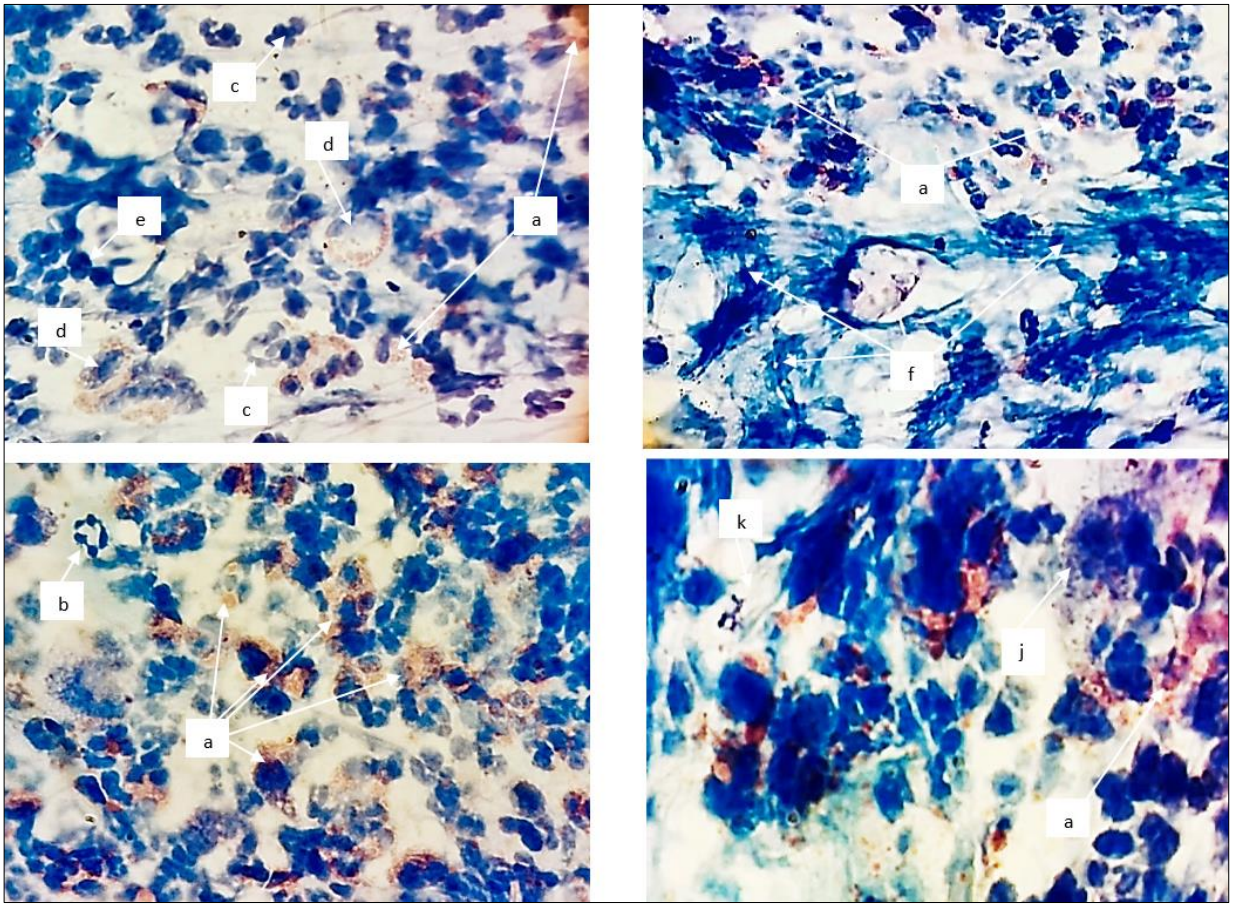

Fig. 10. Cytological features of smears: *a* – eosinophils; *b* – non-degenerative neutrophil; *c* – degenerative neutrophils; *d* – macrophages with phagocytized eosinophilic granules and collagen fragments; *e* – lymphocytes, *f* – fibrocytes and clusters of fragmented collagen fibers; *j* – granular protein debris; *k* – cholesterol crystals; Leukodif 200 (LDF 200)

This type of collagenous fibrous component is a typical cytological feature of eosinophilic granuloma. Its localisation, accompanied by a significant amount of granular protein debris, was the result of localised areas of necrosis with tissue destruction caused by eosinophilic cytotoxins. No microorganisms were detected in the impression smears. The cytological findings are compatible with a typical eosinophilic granuloma: predominantly eosinophilic infiltration, degenerated collagen fibres, a mixed chronically active cell population – macrophages, neutrophils, lymphocytes, stromal remodelling cells – fibroblastic series, absence of microflora.

Haematological examination revealed that the mean red blood cell count in cats with eosinophilic syndrome was $8.30 \pm 0.27 \times 10^{12}$, which differed significantly from the control group ($7.40 \pm 0.17 \times 10^{12}$; $P < 0.01$, Table 1). The haemoglobin level in the blood of diseased cats averaged 147.0 ± 5.8 g/L, which was significantly higher than the averaged indicator in healthy animals ($P < 0.05$; Table 1). Hyperchromia (152.0–178.0 g/L) was detected in 33.3% of the sick animals. There was no statistically significant difference between the experimental and control groups in haematocrit levels and red blood cell indices (MCV and MCH) (Table 1; $P < 0.05$).

However, the myeloid lineage of the red bone marrow and the peripheral immune system underwent certain changes in cats with eosinophilic granuloma. In particular, the total white blood cell count in affected cats was significantly elevated (Table 2; $P < 0.05$) and averaged $11.5 \pm 0.46 \times 10^{9}$/L. Analysis of the leukogram showed that the number of eosinophils in the blood of all affected animals was significantly increased (Fig. 11).

Table 1
Dynamics of erythropoiesis parameters in cats with eosinophilic syndrome (x ± SD)

Hematological parameters	Group of cats			
	clinically healthy (n = 10)		patients with EGC (n = 12)	
	x ± SD	Lim	x ± SD	Lim
RBC, 10^{12}/L	7.40 ± 0.17	6.20–8.90	8.30 ± 0.27**	7.20–9.80
Haemoglobin, g/L	133.0 ± 3.6	121.0–147.0	147.0 ± 5.8*	128.0–178.0
Hematocrit, %	42.2 ± 2.07	38.0–47.0	45.5 ± 3.82	41.0–54.0
MCH, pg	18.1 ± 1.26	16.2–19.0	17.8 ± 1.31	16.5–18.9
MCV, μm³	57.2 ± 4.07	55.6–59.5	54.9 ± 2.86	51.9–58.3

Notes: * – $P < 0.05$, ** – $P < 0.01$ – compared with clinically healthy cats.

However, it should be noted that the degree of increase in these granulocytes in the peripheral blood depended on the stage of the eosinophilic granulomatous complex. Thus, in the asymptomatic stage of this pathology, mild eosinophilia was observed – 8–11% (against a normal range of 2–7). During the stage of granulomatous ulcer formation on the oral mucosa, and the tongue – moderate eosinophilia (12–21%), and in the generalised form of eosinophilic syndrome (involvement of the oral mucosa, skin of the head and body) – marked hypereosinophilia – 22–59%.

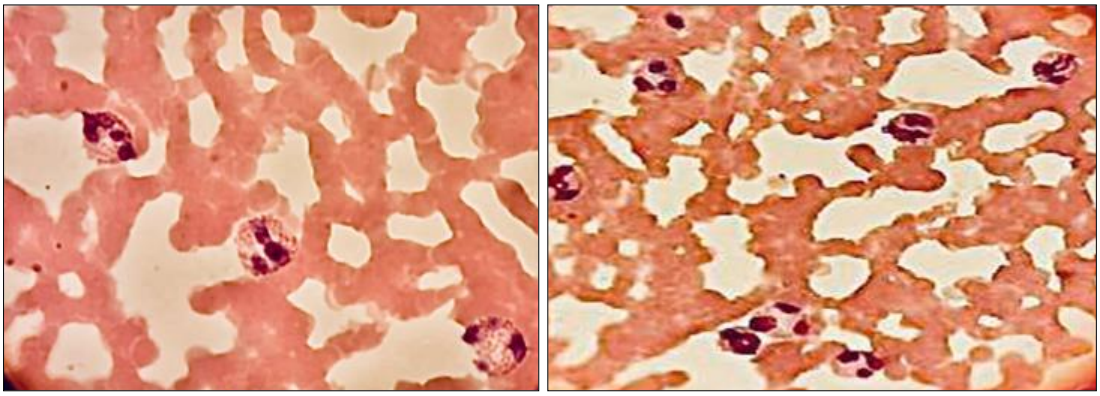

Fig. 11. Hypereosinophilia in a blood smear from the sick cat. Leukodiff 200 (LDF 200)

Table 2
Dynamics of leukocytopoiesis indices
in cats with eosinophilic syndrome (x ± SD)

Leukocyte differential	Group of cats			
	clinically healthy (n=10)		patients with EGC (n=12)	
	x ± SD	Lim	x ± SD	Lim
Leukocytes, 10^9/L	9.72 ± 0.59	8.1–12.3	11.5 ± 0.46*	8.0–15.8
Eosinophils, %	2.8 ± 0.48	2.0–4.0	24.6 ± 0.26***	8.0–59.0
Basophils, %	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
Juvenile neutrophils, %	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0
Band neutrophils, %	4.2 ± 0.41	3.0–6.0	1.4 ± 0.83**	1.0–3.0
Segmented neutrophils, %	44.0 ± 1.56	41–48	46.6 ± 2.16	17–62
Lymphocytes, %	46.6 ± 0.88	42–50	25.8 ± 2.26**	17–36
Monocytes, %	2.4 ± 0.52	2.0–3.0	0.6 ± 0.37*	1.0–7.0

Notes: * – P < 0.05, ** – P < 0.01, *** – P < 0.001 – compared with clinically healthy cats.

Analysing changes in other types of leukocytes, it should be noted that the percentage of segmented neutrophils, lymphocytes and monocytes decreased significantly (Table 2; P < 0.001; P < 0.001; P < 0.05, respectively), indicating the development of relative eosinophilia in the context of general depletion of the leukocyte pool.

Discussion

Recently, due to the rising prevalence of allergies not only among the human population but also among animals, as well as an increase in the incidence of autoimmune and oncological diseases in animals and the activation of parasitic infections, the frequency of eosinophilic syndrome has risen. This syndrome often accompanies these diseases and may influence the characteristics of their course, prognosis, the development of complications and the choice of treatment methods.

The results obtained in our study allow for a more detailed characterisation of the features of the various clinical forms of eosinophilic syndrome in domestic cats and highlight the importance of a comprehensive approach to the diagnosis of this pathology.

The data we have obtained matches the current understanding of feline eosinophilic granuloma (FEG) as a multifactorial allergy-based condition, the course of which greatly depends on the animal's individual response and external factors, especially allergens. Clinical data on the influence of housing and feeding conditions are also of significant importance. A considerable proportion of cats whose diet included human food or by-products may indicate a possible dietary etiological component in the development of allergic reactions. The high prevalence of cases in British and Scottish breeds may be linked to genetic predisposition. Our data on breed susceptibility to eosinophilic granulomatosis in cats correlate with the findings of Pressanti & Cadiergues (2015), who described cases of eosinophilic dermatitis in kittens from the same litter, which directly points to a genetic link to the disease.

Da Rocha et al. (2019) found a clear link between infestation with the tick *Lynxacarus radovskyi* and the development of EGC. The presence of ectoparasites, as well as contact with external allergens, may act as triggering factors, as evidenced by the positive response of some animals to elimination diets and antihistamine or antiparasitic treatments. The variability of the clinical changes we diagnosed (ranging from local granulomatous lesions on the lips to generalised skin lesions) confirms the heterogeneity of the syndrome and the varying degrees of immune-inflammatory response (Bryan et al., 2010; Omelchenko et al., 2023). The local lesions observed in one-third of the cats were characterised by mild eosinophilia (8–11%) and a relatively stable, acceptable general health status of the animals, which likely indicates an early stage of the disease or limited exposure to the allergen. In contrast, significant lesions of the oral cavity and generalised plaques on the head and trunk of cats, observed in almost 50.0% and 25.0% of animals respectively, were accompanied by moderate (12–21% eosinophils) and hypereosinophilia (22–59%), as well as changes in general condition, appetite and enteropathy. This correlation between the degree of lesions and the leukogram parameters confirms the role of eosinophils as key effector cells in the pathogenesis of EGC.

According to Vogelnest (2017), skin lesions are often just a marker of cats' general health and systemic diseases. Our research found signs of enteropathy (vomiting, loose stools) in 25% of animals with the generalised form. This is consistent with the findings of Tucker et al. (2014), who documented ultrasonographic signs of eosinophilic enteritis, and Kim et al. (2023), who described cases of lympho-plasmacytic and eosinophilic enterocolitis. In addition, Cerna (2024) notes the development in cats of eosinophilic sclerosing fibropathy of the gastrointestinal tract, manifested by the formation of eosinophilic masses associated with the digestive tract and surrounding abdominal lymph nodes, which are often localised near the pylorus or the ileocecal junction.

In contrast to our findings, where cats predominantly showed skin and mucosal lesions in the head area, other authors have reported different localisations of pathological changes in eosinophilic syndrome. For instance, Balicka et al. (2021) and Lucyshyn et al. (2021) describe eosinophilic keratitis and keratoconjunctivitis in the context of EGC, whilst Lee et al. (2020) indicate the possibility of eosinophilic respiratory tract inflammation in cats. Soltero-Rivera et al. (2023) reported in their studies that concurrent gastrointestinal signs were seen in 44% of cats, 37% of patients showed skin-related lesions, and 33% showed respiratory signs. Taken together, 39% of cats experienced a combination of clinical signs. Such findings allow eosinophilic granulomatosis in cats to be characterised as a systemic polyorgan syndrome.

Based on the literature, eosinophilia in feline eosinophilic granulomatosis arises in response to increased differentiation of precursor cells and proliferation of eosinophils in the bone marrow due to the production of interleukins (IL-5, -3, -4, -13). It is also associated with activation of interactions between eosinophils and endothelial cells, leading to increased adhesion and migration of eosinophils. In addition, it is associated with enhanced chemokine interactions with eosinophils and activation and destruction of their mature forms (Miller et al., 2013; Diny et al., 2017).

Allergic reactions are the pathogenetic mechanism in the development of eosinophilia, which involves genetic, inflammatory and cytotoxic components. A susceptibility to atopy is inherited by 20–

30% of animals, which is associated with the overproduction of IgE antibodies to environmental antigens (Diesel, 2017; Santoro et al., 2021). The genetic component of eosinophilia involves relevant genes responsible for IgE production and the expression of their receptors, as well as loci at the IL-4 and IL-5 genes on chromosome 5q31 (Halliwell et al., 2021). It has been established that eosinophilia is also genetically determined by genes of the histocompatibility complex on chromosome 6 (Pressanti & Cadiergues, 2015; Halliwell et al., 2021).

The inflammatory component of eosinophilic syndrome is caused by certain mechanisms that lead to the pathological process entering a chronic phase. Following increased IgE synthesis, tissue basophils produce the cytokines IL-1 and TNF-α, thereby inducing inflammation (Banovic et al., 2025). Concurrently, Th-2 lymphocytes (which are essential cells of the late phase of the allergic response) become involved in the process, as they produce the cytokines IL-4 and IL-13 and regulate IgE synthesis and IL-5 production. The late phase of the allergic response is accompanied by an inflow of inflammatory cells to the target tissues as early as 3–4 hours after contact with the allergen (Diesel, 2017). In this phase, the synthesis of Th1 cells, which produce IFN-γ and TNF-β, is also regulated by tissue basophils and Th2 cells (Halliwell et al., 2021; Banovic et al., 2025). Tissue basophils primarily activate Th2 cells and eosinophils, secrete inflammatory mediators that induce the synthesis of chemokines by epithelial cells and trigger eosinophil migration, ensure the development and maintenance of inflammation in tissues, and also cause tissue eosinophilia by secreting IL-3, IL-5 or GM-CSF, which stimulate these cells (Diny et al., 2017). Eosinophils, in turn, enhance the expression of numerous receptors for cytokines, immunoglobulins and complement components on their membranes (Miller et al., 2013; Diny et al., 2017).

The third stage in the development of eosinophilic syndrome is cytotoxic. It is primarily caused by cytotoxic substances. It has been established that eosinophils produce unique toxic inflammatory mediators following their activation (Bardagí et al., 2003). Their granules contain a crystalline substance comprising large basic and cationic proteins and eosinophil peroxidase. These aggregates widen the pores of cell membranes, facilitating the penetration of other toxic molecules. They also significantly increase the sensitivity of M2 muscarinic receptors in smooth muscle and induce degranulation of tissue basophils. In addition, they promote the production of large quantities of cysteine-derived leukotrienes, including LTC4, LTD4, and LTE4. These mediators increase vascular permeability, enhance mucus secretion, stimulate prolonged smooth muscle contraction, and destroy cell membranes. Finally, activated eosinophils begin to produce cytotoxic cytokines (Bardagí et al., 2003; Diny et al., 2017).

In view of the above, a cytological examination should be conducted, as it is of particular importance for confirming the diagnosis. In impression smears from cats with eosinophilic syndrome, a large number of eosinophils, phagocytic macrophages, degenerative collagen fibres and protein debris were detected, which clearly corresponds to the typical morphological features of eosinophilic granuloma. Our data correlate with the findings of Bhagya et al. (2025), who, during a cytological examination of an eosinophilic ulcer in a cat, revealed the presence of a predominant number of eosinophils, ruptured eosinophils leaving naked nuclei, eosinophilic granules in the background, and infiltration of neutrophils in some areas. Gowri et al. (2022) previously reported that dense infiltration of eosinophils along with a few neutrophils was frequently observed on impression smears of feline eosinophilic granuloma complex.

The presence of fibroblasts and fibrocytes, which we detected in the smears, indicates a chronic process, consistent with clinical cases of longstanding lesions. Describing the condition and pathologies of the skin, particular emphasis is now placed on its microbiome. Older et al. (2023) investigated the relationship between skin health and its microbiome. The authors concluded that the bacterial population of the skin plays an important role in the development of dermatopathies and skin changes in response to both infectious and non-infectious factors. Significantly, no microflora was detected in the samples during our study, which allows us to exclude the infectious nature of the pathology and confirms the immunoallergic mechanism of the disease's development. This is consistent with the published data, according to which eosinophilic granuloma develops as a result of hypersensitivity to external or internal allergens, and secondary infections occur only in cases of significant damage to the skin or mucous membranes.

Haematological monitoring has shown a number of typical changes associated with eosinophilic syndrome. In particular, an increase in red blood cell count and haemoglobin concentration may be associated with the body's stress responses, dehydration, or compensatory mechanisms aimed at maintaining tissue oxygenation in cases of chronic inflammation. In contrast, changes in the leukocyte formula (a decrease in segmented neutrophils, monocytes and lymphocytes with an increase in the eosinophil population) are a typical response of the immune system to allergic stimulation. Such changes indicate a disruption of homeostasis in the leukocyte compartment and the development of relative eosinophilia, which is a key diagnostic feature of EGC. However, blood abnormalities such as increased white blood cell count and hypereosinophilia (up to 59%), as observed in our study, should be distinguished in patients with the generalised form of the disease from feline eosinophilic leukaemia. According to Gelain et al. (2006) and Sharifi et al. (2007), feline eosinophilic leukaemia is characterised by marked leukocytosis (>10 G/L) with 77% eosinophils (many of them immature), severe non-regenerative anaemia and significant thrombocytopenia.

This study not only expands existing understanding of the course of EGC in domestic cats but also emphasizes the need for further investigation of this pathology, particularly regarding the search for new diagnostic markers, the identification of genetic susceptibility, and the refinement of treatment protocols. Raising awareness among veterinary specialists regarding the specific manifestations of eosinophilic syndrome will facilitate timely diagnosis, more effective treatment and the prevention of chronic forms of the disease.

Conclusions

Eosinophilic syndrome in domestic cats is characterised by significant clinical variability, appearing as eosinophilic granuloma, ulcers and plaques, which may be found on the mucous membranes of the oral cavity as well as on the skin of the head and trunk. The severity of clinical symptoms depends on the individual's immune response and the type of allergen. In 33.3% of animals, local granulomatous lesions with mild eosinophilia (8–11%) were diagnosed; in 41.7%, ulcerative lesions of the oral cavity with moderate eosinophilia (12–21%); and in 25.0% – a generalised form with severe hypereosinophilia (22–59%), which indicates a clear correlation between the severity of the disease and changes in the leukogram. Cytological studies confirmed typical features of eosinophilic granuloma, including a prevalence of eosinophils, the presence of degraded collagen fibres, foamy macrophages and fibroblasts, as well as the absence of microorganisms. This emphasizes the immunological and allergic nature of the pathology and excludes bacterial or fungal origins of the lesions. Blood tests revealed a significant increase in white and red blood cell counts and haemoglobin levels in the cats of the study group, which may be associated with a stress response or compensatory mechanisms in chronic inflammation. Changes in the leukocyte differential count indicate the development of relative eosinophilia with a decrease in other white blood cell populations. The results obtained emphasize the necessity of a comprehensive diagnostic approach, which should include clinical examination, cytological studies, haematological analysis, exclusion of infectious agents, and assessment of the allergic component. This will allow increased diagnostic accuracy and therapeutic efficacy, as well as prevent the progression of the condition to a chronic state and recurrence.

The authors declare no conflict of interest.

References

Balicka, A., Lapšanská, M., Fekiačová, V., & Trbolová A. (2021). Feline eosinophilic keratitis – a review. Folia Veterinaria, 65(3), 75–79.

Banovic, F., Gomes, P., & Trainor, K. (2025). Feline immune-mediated skin disorders: Part 2. Journal of Feline Medicine and Surgery, 27(4), 1–15.
Bardagí, M., Fondati, A., Fondevila, D., & Ferrer, L. (2003). Ultrastructural study of skin lesions in feline eosinophilic granuloma complex. Veterinary Dermatology, 14, 297–303.
Bhagya, B. K., Shivakumar, V., Kavitha, K., & Shivakumar, M. (2025). Cytological diagnosis and successful treatment of indolent ulcer in a Persian cat: A case report. Indian Journal of Veterinary and Animal Sciences Research, 54, 86–90.
Bloom, P. B. (2006). Canine and feline eosinophilic skin diseases. Veterinary Clinics of North America, Small Animal Practice, 36(1), 141–160.
Bryan, J., & Frank, L. A. (2010). Food allergy in the cat: A diagnosis by elimination. Journal of Feline Medicine and Surgery, 12(11), 861–866.
Buckley, L., & Nuttall, T. (2012). Eosinophilic granuloma complex(s) in cats: Some clinical clarifications. Journal of Feline Medicine and Surgery, 14(7), 471–481.
Cerna, P. (2024). Understanding feline gastrointestinal eosinophilic sclerosing fibroplasia. Companion Animal, 29, 2–6.
Da Rocha, C. M., Farias, P. C. G., Gorza, L., Soares, F. E. F., Ferraz, C. M., Souza, R. L. O., Renon, L. B. S., & Braga, F. R. (2019). Association between infestation by *Lynxacarus radovskyi* (Acari: Lystrophoridae) and the occurrence of feline eosinophilic granuloma complex. Journal of Parasitic Diseases, 43(4), 726–729.
Diesel, A. (2017). Skin hypersensitivity dermatoses in feline patients: A review of allergic skin diseases in cats. Veterinary Science, 4(2), 25.
Diny, N. L., Rose, N. R., & Čiháková D. (2017). Eosinophils in autoimmune diseases. Frontiers in Immunology, 8, 484.
Dokuzeylul, B., Kayar, A., & Or, M. E. (2016). Prevalence of systemic diseases in cats with oral lesions. Veterinární Medicína, 61(4), 219–223.
Ehlers, L. P., Slaviero, M., Vargas, T. P., Argenta, F. F., Driemeier, D., Amorim da Costa, F. V., Pavarini, S. P., & Sonne, L. (2019). Epidemiologic and pathologic aspects of feline eosinophilic granuloma complex. Acta Scientiae Veterinarie, 47(1), 98316.
Falcão, F., Faísca, P., Viegas, I., De Oliveira, J. T., & Requicha, J. F. (2020). Feline oral cavity lesions diagnosed by histopathology: A 6-year retrospective study in Portugal. Journal of Feline Medicine and Surgery, 22(10), 977–983.
Fondati, A., Fondevila, D., & Ferrer, L. (2001). Histopathological study of feline eosinophilic dermatoses. Veterinary Dermatology, 12, 333–338.
Foster, A. (2003). Clinical approach to feline eosinophilic granuloma complex. In Practice, 25(1), 2–9.
Gelain, M. E., Antoniazzi, E., Bertazzolo, W., Zaccolo, M., & Comazzi, S. (2006). Chronic eosinophilic leukemia in a cat: Cytochemical and immunophenotypical features. Veterinary Clinical Pathology, 35(4), 454–459.
Gowri, B., George, A., Aasthika, E., Rajkumar, S. S., & Kavitha, S. (2022). Clinical and cytopathological diagnosis of feline eosinophilic granuloma complex. The Pharma Innovation, 11(9), 2777–2779.
Halliwell, R., Pucheu-Haston, C. M., Olivry, T., Prost, C., Jackson, H., Banovic, F., Nuttall, T., Santoro, D., Bizikova, P., & Mueller, R. S. (2021). Feline allergic diseases: Introduction and proposed nomenclature. Veterinary Dermatology, 32(1), 8-e2.
Hobi, S., Linek, M., Marignac, G., Olivry, T., Beco, L., Nett, C., Fontaine, J., Roosje, P., Bergvall, K., Belova, S., Koebrich, S., Pin, D., Kovalik, M., Meury, S., Wilhelm, S., & Favrot, C. (2011). Clinical characteristics and causes of pruritus in cats: A multicentre study on feline hypersensitivity-associated dermatoses. Veterinary Dermatology, 22, 406–413.
Hopke, K. P., & Sargent, S. J. (2019). Novel presentation of eosinophilic granuloma complex in a cat. Journal of Feline Medicine and Surgery Open Reports, 5(2), 1–7.
Kim, J. Y., Hwang, T. S., Jung, D. I., Song, K. H., & Song, J. H. (2023). Lymphocytic-plasmacytic and eosinophilic enterocolitis presented with marked eosinophilia and basophilia in a cat: Case report. Frontiers in Veterinary Science, 10, 1153702.
Lee, E. A., Johnson, L. R., Stern, J. A., & Vernau, W. (2020). Clinical features and radiographic findings in cats with eosinophilic, neutrophilic, and mixed airway inflammation (2011–2018). Journal of Veterinary Internal Medicine, 34(3), 1291–1299.
Lucyshyn, D. R., Vernau, W., Maggs, D. J., Murphy, C. J., & Leonard, B. C. (2021). Correlations between clinical signs and corneal cytology in feline eosinophilic keratoconjunctivitis. Veterinary Ophthalmology, 24, 620–626.
Miller, J., Simpson, A., Bloom, P., Diesel, A., Friedeck, A., Paterson, T., Wisecup, M., & Yu, C. M. (2023). AAHA management of allergic skin diseases in dogs and cats guidelines. Journal of the American Animal Hospital Association, 59(6), 255–284.
Miller, W. H., Griffin, C. E., & Campbell, K. L. (2012). Muller and Kirk's small animal dermatology. 7th edition. Saunders, Elsevier Health Sciences, Saint Louis.
Older, C. E., Hoffmann, A. R., & Diesel, A. B. (2023). The feline skin microbiome: Interrelationship between health and disease. Journal of Feline Medicine and Surgery, 25(7), 1–9.
Oliveira, A., & Van den Broek, A. (2006). The feline eosinophilic granuloma complex. Companion Animal, 11(1), 48–55.
Omelchenko, H., Avramenko, N., Kulynych, S., Petrenko, M., Volosovets, V., Volosovets, N., & Woźniakowski, G. (2023). Some aspects of the diagnosis and treatment of eosinophilic granuloma in cats. Journal of Veterinary Research, 67(4), 619–626.
Paterson, S. (2016). Eosinophilic granuloma complex in the cat. Companion Animal, 21(5), 256.
Pressanti, C., & Cadiergues, M. C. (2015). Feline familial pedal eosinophilic dermatosis in two littermates. Journal of Feline Medicine and Surgery Open Reports, 1(1), 1–6.
Santoro, D., Pucheu-Haston, C. M., Prost, C., Mueller, R. S., & Jackson, H. (2021). Clinical signs and diagnosis of feline atopic syndrome: Detailed guidelines for a correct diagnosis. Veterinary Dermatolology, 32(1), 26-e6.
Scott, D. W., & Miller, W. H. (2012). Idiopathic eosinophilic granuloma in cats: A retrospective study of 55 cases. The Japanese Journal of Veterinary Dermatology, 18(1), 13–18.
Sharifi, H., Nassiri, S. M., Esmaelli, H., & Khoshnegah, J. (2007). Eosinophilic leukaemia in a cat. Journal of Feline Medicine and Surgery, 9(6), 514–517.
Soltero-Rivera, M., Quinones, M. P. D. T., Arzi, B., & Vapniarsky, N. (2023). Importance of early diagnosis, multimodal treatment, and a multidisciplinary approach for oral eosinophilic lesions in cats: a retrospective study of 38 cases (1997–2022). Journal of the American Veterinary Medical Association, 261(2), 70–78.
Tucker, S., Penninck, D. G., Keating, J. H., & Webster, C. R. L. (2014). Clinicopathological and ultrasonographic features of cats with eosinophilic enteritis. Journal of Feline Medicine and Surgery, 16(4). 270–276.
Vogelnest, L. J. (2017). Skin as a marker of general feline health: Cutaneous manifestations of systemic disease. Journal of Feline Medicine and Surgery, 19(9), 948–960.