Artykuł przeglądowy *Review*

Pathogenesis and management of feline eosinophilic granuloma complex

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Summary

Feline eosinophilic granuloma complex (FEGC) is a heterogeneous inflammatory disorder characterized by diverse clinical manifestations and multifactorial etiopathogenesis. This review summarizes the current knowledge on the etiopathogenesis, clinical presentation, diagnostic approach, and therapeutic management of FEGC based literature evidences. Currently, FEGC is understood as an immunological reaction pattern that most commonly develops secondarily to allergic diseases. Consequently, the diagnosis of FEGC should be considered the starting point for identifying the underlying cause. Effective management is based primarily on recognition and elimination of the triggering factor, whereas anti-inflammatory and immunomodulatory therapy is used to control clinical signs and should accompany treatment of the underlying disease whenever possible. Appropriate identification of the primary cause together with an individualized therapeutic approach is essential for achieving long-term remission and reducing the risk of recurrence.

Keywords: Feline eosinophilic granuloma complex, pathogenesis, diagnosis, treatment

Feline eosinophilic granuloma complex (FEGC) is a heterogeneous disease with a multifactorial etiology and variable clinical presentation. The complex includes three major clinical forms manifesting on the skin, mucocutaneous junctions, or within the oral cavity: eosinophilic granuloma (EG), eosinophilic plaque (EP), and eosinophilic ulcer (EU; indolent ulcer, IU). These lesions may occur individually or coexist in the same animal, differing in location, clinical appearance, and the severity of pruritic and inflammatory responses. Until the end of the 20th century, the pathogenesis of feline eosinophilic granuloma complex was poorly understood, and eosinophilic granuloma was regarded as a distinct skin disease of unknown etiology (30, 39). Early studies mentioned the possible involvement of infectious (27), autoimmune (17), and environmental factors. Currently, the studies on immunopathogenesis have shown FEGC as a multifactorial pattern of inflammatory response affecting the skin and mucous membranes, most commonly associated with hypersensitivity reactions to flea and mosquito bites, food, or environmental allergens, with evidence supporting the involvement of autoallergens, autoantibodies, and hereditary predisposition in some groups of cats (5, 14, 16, 41, 44, 59). The term Feline Eosinophilic Granuloma Complex (FEGC) emphasizes the coexistence of several types of cutaneous lesions with a similar pathogenesis.

Etiology and pathogenesis

Current concepts emphasize that FEGC represents a pattern of immune response that most commonly develops in the course of allergic diseases (4, 5, 41). In numerous cases, no unequivocal factors triggering the development of the lesions have been identified; however, in many cats, lesions identified as FEGC have been clearly associated with hypersensitivity reactions to insect bites (fleas, mosquitoes), environmental allergens, and food allergens. Disease development is associated with both immediate (type I) and delayed (type IV) hypersensitivity reactions, which lead to eosinophil recruitment and activation and the release of inflammatory mediators in response to antigenic stimulation (7). A study of cats with FASS (Feline Atopic Skin Syndrome), which is one of the main causes of lesions belonging to FEGC, demonstrated increased expression of Th2 cytokines, such as IL-4, IL-5, and IL-13, and the chemokine CCL17, as well as

IL-17A and IL-10, in eosinophilic plaques, confirming the predominance of a Th2-type inflammatory response in the skin (57). It should be emphasized that this cytokine profile deals with eosinophilic plaques in cats with FASS, in which they constitute one of the FEGC variants. FASS, however, is an etiological diagnosis, whereas FEGC represents a pattern of cutaneous and mucosal lesions that may develop in the course of various diseases (18). It is most commonly observed in cats with FASS, flea-bite hypersensitivity, adverse food reactions, and mosquito-bite hypersensitivity, and less commonly in the course of other disorders. Therefore, a diagnosis of FEGC should be regarded as an indication for further diagnostic investigation aimed at identifying the underlying disease.

Food hypersensitivity is considered to be of particular importance in the etiopathogenesis of FEGC. The most commonly reported food allergens in cats include beef, fish, and chicken; however, virtually any dietary protein has the potential to trigger hypersensitivity reactions in susceptible animals. Chronic exposure to food antigens leads to eosinophil recruitment and the development of a pattern of cutaneous lesions that match the eosinophilic granuloma complex. In some cats, an adverse food reaction may be manifest exclusively as dermatological lesions, without concurrent gastrointestinal signs. Confirmation of food as a cause of lesions in the course of FEGC requires implementation of an elimination diet followed by a dietary provocation challenge (23).

Parasitic factors that may play a role in the etiopathogenesis of feline eosinophilic granuloma complex include not only fleas but also other ectoparasites, such as *Lynxacarus radovskyi*. This fur mite is predominantly reported in tropical and subtropical regions, including Brazil, Australia, Hawaii, Florida, and several islands of the Pacific and Caribbean, although sporadic cases have also been described elsewhere. This mite inhabits the hair coat of domestic cats and may induce a hypersensitivity reaction leading to the development of eosinophilic dermatitis. Infestation with *Lynxacarus radovskyi* may be associated with the occurrence of lesions belonging to FEGC, constituting a potential trigger for this cutaneous reaction. Besides of lesions typical of FEGC, the presence of this ectoparasite has also been associated with miliary dermatitis, flea allergy dermatitis (FAD), and non-flea, non-food hypersensitivity dermatitis (NFNFHD). Therefore, the presence of *L. radovskyi* should be considered in the differential diagnosis, particularly in cats presenting with pruritus, scaling, and deterioration of coat quality (a "salt-and-pepper" appearance) (19, 46).

In addition to the classical mechanisms of hypersensitivity to environmental allergens, food allergens, and insect bites, the involvement of autoantibodies in the pathogenesis of FEGC has also been mentioned in the literature. Antibodies directed against components of the cat's own epidermis have been detected in blood of some cats with eosinophilic granuloma complex, suggesting a possible autoimmune component in the development of cutaneous lesions. It should be emphasized, however, that autoantibodies may arise secondarily as a consequence of epithelial damage and the release of tissue antigens, and their presence does not necessarily indicate a primary autoimmune process (17, 32). The retrospective study by Gelberg et al. (1985) detected autoantibodies in 68% of cats but did not provide convincing evidence supporting an autoimmune basis of FEGC. During the following four decades, evidence supporting the autoimmune nature of the disease remained scarce. Moreover, the Fel d 1 allergen (*Felis domesticus* allergen I), known primarily as a feline allergen in human allergic disease, has been shown to act as an autoallergen, promoting persistence of the chronic inflammatory response through modification of the CD4/CD8 lymphocyte ratio in the skin of cats with eosinophilic granuloma complex (59).

Other studies suggest that infectious agents, such as viruses, bacteria, or fungi, are involved in the etiology of FEGC. However, the consensus regarding this concept is lacking. One study described an attempt of autologous transfer of eosinophilic plaques to other sites of the body in five cats. The inability to induce new lesions as a result of this procedure led to conclusion that these lesions were probably not infectious in nature and therefore not transmissible (32). Nevertheless, bacteria have been demonstrated within lesions in both cytological and histological examinations, including oral eosinophilic granulomas, eosinophilic plaques, and eosinophilic ulcers (47). At the same time, it is emphasized that FEGC lesions facilitate microbial colonization of damaged tissues; therefore, the role of bacteria in the disease is most likely secondary. Nevertheless, routine cytological examination is recommended during the diagnostic work-up and, in justified cases, bacterial culture with antimicrobial susceptibility testing should also be performed, enabling confirmation of a possible bacterial infection and guiding treatment (4).

The involvement of viral factors in the pathogenesis of FEGC has also been considered. In some cases, feline herpes virus type 1 (FHV-1) antigens have been demonstrated in cutaneous and oral eosinophilic lesions using immunohistochemical methods (34); however, the virus was detected in only a small proportion of cases, suggesting that its role in the development of EGC is limited. Nevertheless, case reports emphasize the importance of including FHV-1 infection in the differential diagnosis and excluding it as a potential etiological factor, which may be important for selecting appropriate therapeutic strategy and increasing the likelihood of a favorable treatment outcome (27, 42).

Similarly, with regard to fungal factors, studies conducted to date have not demonstrated a direct role of fungi in the development of FEGC; however, fungal infections should be included in the differential

diagnosis of cutaneous and mucosal lesions because some dermatomycoses may clinically resemble EGC (21). Infection with *Pythium insidiosum* has been shown to cause severe, multifocal, eosinophilic granulomatous inflammation of the oral cavity. Pythiosis is a potentially fatal but non-contagious disease that is rarely reported in cats and typically causes granulomatous disease of the skin or gastrointestinal tract (15).

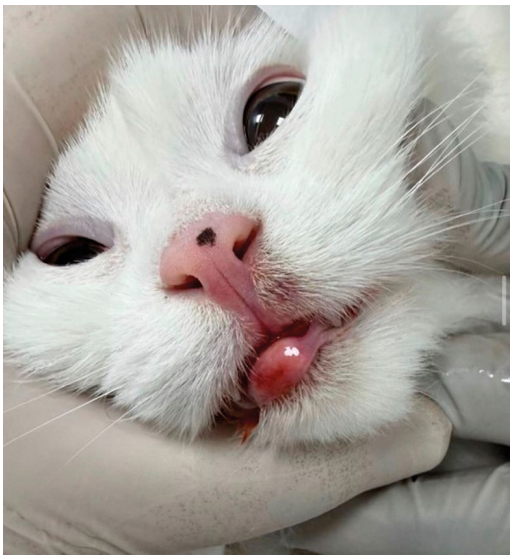

Fig. 1. Eosinophilic granuloma of the lower lip in a 14-year-old female Persian cat. A focal, nodular lesion undergoing ulceration is visible. It is characterized by raised, erythematous, well-demarcated swelling with central ulceration and mild bleeding. Source: Dr. Moustafa Elsobky. Reproduced with permission

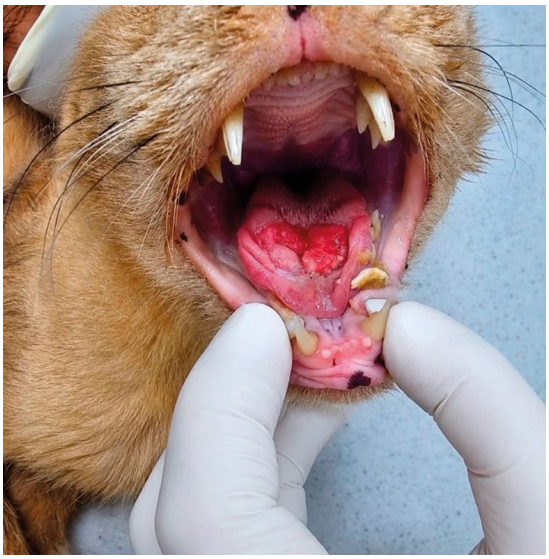

Fig. 2. Eosinophilic granuloma of the tongue in a cat. A proliferative, erythematous, ulcerative lesion, clearly demarcated from healthy tissues, is visible. Source: Kacper Śróda. Copyright © 2026 Kacper Śróda

Research into the etiology of feline eosinophilic granuloma complex suggests a potential hereditary predisposition (43, 44, 50). Although specific genetic mutations or underlying molecular mechanism remains unidentified, clinical observations have documented familial clustering of the disease, indicating a likely inherited component. In a study involving 17 related Norwegian Forest cats, a high prevalence of the linear form of eosinophilic granuloma was identified, with lesions observed in several individuals from the same litter and in cats sharing a common ancestor. Pedigree analysis demonstrated an increased frequency of lesions within this population, which may suggest a hereditary predisposition to the disease. In addition, the authors indicated that because six of fourteen kittens from the same litter developed similar lesions, the inheritance pattern may indicate a recessive predisposition (28).

It is currently considered that, in most cats, FEGC represents a cutaneous reaction pattern that develops secondary to allergic diseases; however, in some patients, the triggering factor cannot be unequivocally identified.

Clinical signs

Feline eosinophilic granuloma complex may occur in three different forms that are histologically distinct but share some common features (13). These lesions are most commonly located in the skin (87.1%) and less frequently involve mucocutaneous junctions (12.9%) (40). However, a study that included oral lesions found that lesions occurred most commonly in the oral cavity (40%), then on the skin (33.3%), and then at mucocutaneous junctions (26.6%) (9). In a retrospective study of oral locations, lesions were most frequently observed on the lips (36.4%), followed by the dorsal and ventral surfaces of the tongue (11). Another study, however, found that the tongue was the most commonly affected site in cats with FEGC, followed somewhat less frequently by the lip and pharyngeal regions, with half of the lesions having an ulcerative gross appearance (54). A case of eosinophilic panuveitis in a cat has also been described (35).

Eosinophilic granuloma (EG) is a condition also described as feline collagenolytic granuloma and feline linear granuloma. Physical examination allows to suspect an eosinophilic granuloma; however, it should be confirmed histopathologically. Lesions may occur anywhere on the body, including inside the oral cavity. Classical eosinophilic granuloma occurs in a characteristic linear arrangement on the caudal or medial aspect of the thighs. Lesions are frequently located at the lip margins and on the chin (Fig. 1). In the oral cavity, typical locations include the tongue (Fig. 2), hard palate or palatine arches. Eosinophilic granulomas are pink, raised, and alopecic. Foci ranging in color from white to orange-yellow may occur within the lesions. Both cutaneous and oral eosinophilic granulomas are ulcerative and may undergo cavitation while remaining clearly demarcated from the surrounding healthy tissues. Oral lesions may lead to erosions and may bleed; moreover, cats with oral lesions may suffer from dysphagia (24).

Eosinophilic plaques (EP) occur primarily on the abdomen and inner thighs. They may also be found on the skin of the head and neck. They usually cause severe pruritus (55). These lesions present as focal areas of alopecia with marked erythema (Fig. 3). Clinically, they range in severity from poorly demarcated, small macules to large, well-demarcated, glistening, raised

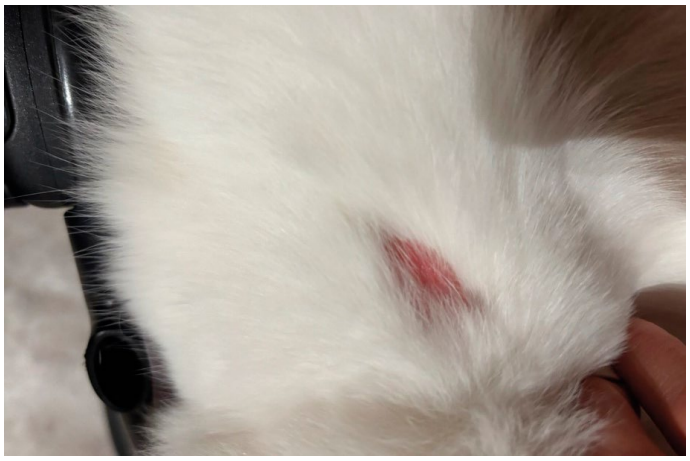

Fig. 3. Eosinophilic plaque in the scapular region of a 5-year-old Maine Coon cat. A focal, well-demarcated alopecic skin lesion with pink coloration and a moist, glistening surface, accompanied by erythema and severe pruritus. Source: Kacper Śróda. Copyright © 2026 Kacper Śróda

plaques (44). Eosinophilic plaques occur more frequently in younger cats between two and six years of age, with no breed or sex predisposition (16).

Eosinophilic ulcer (EU; indolent ulcer; rodent ulcer) is a well-demarcated, solitary or symmetrical ulceration (55) occurring in the region of the philtrum or adjacent to the upper canine tooth. EU may occur alone or in association with other eosinophilic diseases. Pruritus is absent in this clinical form (44). The margins of the lesions are raised and surround a pink-yellowish ulcerated surface. Large lesions may result in deformation of the facial tissues (16).

Diagnosis

Clinical recognition of lesions associated with feline eosinophilic granuloma complex is relatively straightforward, whereas determination of their etiology may constitute a significant diagnostic challenge. The lack of unequivocal diagnostic guidelines and initiation of treatment before the cause has been identified may complicate interpretation of diagnostic tests. The course of the disease and response to therapy are variable, probably as a result of differences in etiopathogenesis. The diagnostic approach should focus on confirming lesions consistent with feline eosinophilic granuloma complex and identifying the primary underlying disease (4).

Confirmation of eosinophilic granuloma complex

Clinical history. Particular attention should be paid during history-taking on the presence of pruritus, seasonality of clinical signs, and their possible association with exposure to environmental or food allergens. Information regarding antiparasitic prophylaxis (especially flea control), diet (including recent changes of food), previous episodes of similar skin lesions, and response to treatment is important. Environmental factors and stress, which may exacerbate clinical signs, should also be taken into account.

Clinical examination. Clinical examination includes a detailed assessment of the skin and mucous membranes, with particular attention to the typical forms of FEGC: eosinophilic ulcer (most commonly on the upper lip), eosinophilic plaque (frequently on the abdomen or thighs), and eosinophilic granuloma (e.g., on the caudal aspect of the thighs, in the oral cavity, or on the tongue). Lesions may appear as ulcerations, erosions, erythema, or raised, well-demarcated foci. The severity of pruritus, the presence of secondary infections, and the patient's general condition should be assessed. During examination of the oral cavity, identification of proliferative lesions that may impair food intake is important.

Cytological examination. This involves evaluation of specimens collected from affected skin, for example by impression smear or adhesive tape techniques. The presence of very large numbers of eosinophils in cytological specimens makes one of the forms of feline eosinophilic granuloma complex highly likely. To obtain material from an eosinophilic ulcer, the surface of the lesion may be gently scraped with the blunt edge of a scalpel, after which the collected material is spread onto a microscope slide. Larger ulcers, plaques, or nodules may require histopathological examination to exclude non-allergic differential diagnoses, particularly neoplasia. The presence of intracellular bacteria (indicative of active phagocytosis) or extracellular bacteria together with degenerated neutrophils in a cytological specimen may suggest bacterial infection or colonization. It should be emphasized, however, that antimicrobial therapy – topical or systemic – should be initiated only when infection has been confirmed by cytological and culture tests. Bacterial culture and antimicrobial susceptibility testing are indicated, particularly in animals previously treated repeatedly with antibiotics, when empirical treatment has failed, or when rod-shaped bacteria are identified on cytological examination (4, 22).

Histopathological examination. The principal histopathological features include varying degrees of epidermal hyperplasia and erosion or ulceration, together with a prominent eosinophilic infiltrate within the dermis. So-called "flame figures," i.e., small foci of collagen fibers surrounded by degenerate eosinophils, are frequently observed (1, 13).

Identification and treatment of the underlying disease

A detailed history and clinical examination are key elements in identifying potential etiological factors.

Flea-bite hypersensitivity is one of the most common causes of dermatological lesions in cats, including cutaneous lesions associated with feline eosinophilic granuloma complex (7). This is of particular importance during spring and summer because seasonal variation in the occurrence of FEGC lesions has been demonstrated – their severity increases during the

warmer months, and disease onset often correlates with periods of increased ectoparasite activity (40). Diagnosis is based on clinical examination, including combing the coat to detect fleas or flea feces, which acquire a reddish-brown coloration on contact with water. It should be remembered, however, that their absence does not exclude flea allergy dermatitis (FAD), because of the intensive grooming behavior of cats. In such cases, a therapeutic trial with flea-control products is helpful. Effective control requires an integrated approach involving both the animal and the environment because most developmental stages of fleas occur outside the host. Regular use of products targeting different developmental stages of the parasite is crucial, including adulticides (e.g., fipronil, imidacloprid, selamectin), isoxazolines (fluralaner, afoxolaner, sarolaner, and lotilaner), and insect growth regulators (e.g., lufenuron, pyriproxyfen). Rapid reduction in flea numbers and feeding time is important because it reduces allergen exposure. It is currently considered that a substantial reduction in infestation, rather than complete elimination, may be sufficient to control clinical signs (8, 53). It should be emphasized that flea-bite hypersensitivity is not the only possible cause of lesions within the eosinophilic granuloma complex. Other ectoparasites, such as mites, may play an important role in pathogenesis; therefore, diagnostic testing for these parasites should be provided, including hair examination (trichography), adhesive tape examination, and skin scraping.

Adverse food reactions should be investigated using a strict elimination diet for at least 8 weeks. The most reliable method for investigating food allergy in cats is the use of a diet based on a single protein source previously unknown to the animal. A second, commercially available option is a hydrolyzed diet, which contains proteins broken down into smaller fragments, thereby reducing their antigenicity. If marked clinical improvement is achieved during the elimination diet, a provocation challenge is recommended by reintroducing the previous diet for 10-14 days to assess recurrence of clinical signs. Alternatively, individual dietary components, such as treats or flavored preparations, may be introduced gradually, maintaining 10-14-day intervals between successive challenges (3).

Treatment

It should be emphasized that the prognosis for most cats with FEGC is good, particularly when the triggering factor can be identified and eliminated. In many patients, effective ectoparasite control, an appropriately conducted elimination diet, or short-term anti-inflammatory therapy allows long-term remission of lesions. A multimodal approach is currently recommended for the treatment of feline eosinophilic granuloma complex, including implementation of a hypoallergenic diet, control of ectoparasites, administration of systemic glucocorticoids or other immunosuppressive drugs, use of antimicrobial agents, and, in selected cases, surgical intervention. However, the efficacy of these methods varies and is often short-term. Cats with eosinophilic lesions involving the palate more frequently exhibit respiratory signs and have a poorer response to treatment (54).

Glucocorticoids. Glucocorticoids are usually the drugs of first choice for controlling lesions in cats with eosinophilic granuloma complex. Drugs in this group are used to rapidly suppress inflammation associated with FEGC and to control pruritus. Cats generally tolerate glucocorticoid therapy well; however, adverse effects may occur, including polyuria, polydipsia, polyphagia, weight gain, diabetes mellitus, iatrogenic Cushing's syndrome, congestive heart failure, demodicosis, and gastric ulceration. Prednisolone is the recommended oral formulation for cats and is well tolerated by most healthy cats when used short-time for anti-inflammatory or immunosuppressive purposes. However, the long-term safety of prednisolone requires close monitoring of metabolic parameters. The initial dose of prednisolone is 1-2 mg/kg every 24 h for 7 days, followed by administration every second day; however, in some cases, a higher dose, up to 4 mg/kg, may be required. Lesions should be reassessed after 7-14 days and, if possible, daily doses should be gradually reduced or therapy should be administered every second day. Another recommended glucocorticoid is dexamethasone administered at 0.1-0.2 mg/kg every 24 h (induction dose) until the lesions resolve, followed by administration every 72 h at 0.05-0.1 mg/kg (maintenance dose) to maintain the lowest effective dose that sustains lesion remission. It should be noted that dexamethasone has a stronger diabetogenic effect than prednisolone at equivalent doses (29). If oral administration is impossible or considerably difficult, the use of long-acting glucocorticoid preparations, such as methylprednisolone acetate, may be considered at a dose of 20 mg per cat (4 mg/kg) every two or three weeks. After remission, maintenance doses may be administered every six to twelve weeks. Because the drug cannot be rapidly discontinued and the dose cannot be gradually reduced, depot preparations should be used with caution and primarily in appropriately selected patients. Whenever possible, treatment with oral prednisolone is preferred because it allows individual dose adjustment and gradual tapering to the lowest effective dose (33, 40).

In addition to standard systemic therapy, topical treatment using topical glucocorticoids and calcineurin inhibitors has also been described in cats with allergic dermatoses. Studies of feline allergic dermatoses have shown that a topical spray containing 0.0584% hydrocortisone aceponate may significantly reduce the severity of skin lesions and pruritus in cats with FEGC. It may be used as an adjunctive treatment or as an alternative to high doses of systemic glucocorticoids (33, 48). Individual case reports have also demonstrated the

efficacy of topical calcineurin inhibitors (tacrolimus) used in combination with systemic prednisolone in cats with recurrent eosinophilic granuloma, indicating the potential of topical therapy to reduce recurrence and the dose of systemically administered glucocorticoids (31).

Immunomodulatory drugs. Cyclosporine is one of the treatment options for feline eosinophilic granuloma complex (FEGC), particularly in cases refractory to glucocorticoid therapy or when glucocorticoid use is contraindicated, for example because of congestive heart failure or the severe adverse effects during previous glucocorticoid therapy. In selected patients with diabetes mellitus, cyclosporine may constitute an alternative to glucocorticoids because it has a weaker diabetogenic effect. It should be remembered, however, that this drug may affect glucose metabolism and impair pancreatic β-cell function; therefore, blood glucose monitoring is recommended during therapy (6). Because of the potent immunosuppressive effects of cyclosporine, its use – similarly to glucocorticoids – is contraindicated in animals with severe, uncontrolled bacterial, viral, or fungal infections. The mechanism of action of cyclosporine involves inhibition of T-lymphocyte proliferation and activation. The drug also affects cells that initiate the immune response and effector cells of the allergic response, inducing changes in the function of eosinophils, keratinocytes, Langerhans cells, mast cells, and macrophages, thereby modulating both the initiation and effector phases of the immune response and limiting inflammatory and allergic reactions. Cyclosporine administered at doses ranging from 3.6 to 13.3 mg/kg every 24 h has been shown to be effective in the treatment of cutaneous lesions associated with feline eosinophilic granuloma complex (37, 38, 58). The recommended dose of cyclosporine for cats is 7 mg/kg orally every 24 h with food until the lesions resolve, followed by an attempt to reduce the frequency of administration to every second day. Whenever possible, the frequency of administration should be gradually reduced until the lowest effective maintenance dose is achieved. Cyclosporine is generally well tolerated, and adverse effects are most often limited to mild gastrointestinal disturbances, more severe symptoms including hypersalivation, excitement, decreased appetite, or gingival hyperplasia are observed less frequently (20, 26). There are also reports suggesting a possible association between cyclosporine and reactivation of latent toxoplasmosis (2, 25) and neoplastic processes; however, these reports mainly concern cases of combination therapy with prednisolone, particularly in marked immunosuppression (49). Therefore, concomitant use of cyclosporine and glucocorticoids requires caution. It should also be remembered that cyclosporine interacts with certain drugs, including some antibiotics (e.g., doxycycline, erythromycin) and antifungal drugs (ketoconazole, itraconazole, and fluconazole) (45). Because of the relatively slow onset of action of cyclosporine, its use may be limited in patients requiring rapid control of clinical signs, such as painful oral lesions or extensive, intensely pruritic skin lesions.

Chlorambucil is an alkylating cytostatic drug and a nitrogen mustard derivative. Although feline eosinophilic granuloma complex is not considered a neoplastic disease, chlorambucil may be used because of its immunosuppressive properties rather than its antineoplastic activity, particularly in cats with refractory disease. It is not licensed for use in cats; however, it may be considered for use in cats with cutaneous lesions associated with eosinophilic granuloma complex that are refractory to steroid therapy. Chlorambucil is most commonly used alone at a dose of 0.1-0.2 mg/kg body weight every 24 h or in combination with glucocorticoids every second day. Therapy lasts four to eight weeks. Adverse effects of chlorambucil include vomiting, diarrhea, anorexia, and bone marrow suppression. Therefore, patient monitoring and assessment of hematological parameters every 2 weeks are necessary (4).

It has also been reported that subcutaneous injections of 2.5 MU of interferon omega once or twice weekly may be effective and well tolerated in cats with lesions associated with eosinophilic granuloma complex (4). Because of the limited number of studies and the absence of controlled clinical trials, its use is not currently considered a standard treatment and may be considered only in selected cases.

Oclacitinib. Oclacitinib is a selective Janus kinase (JAK) inhibitor licensed exclusively for use in dogs; however, it may constitute an off-label alternative for the treatment of allergic pruritus in cats in which other drugs were ineffective or may cause serious adverse effects. The initial dose of oclacitinib ranges from 0.6 to 1.6 mg/kg every 12 h, with a median dose of 1.1 mg/kg every 12 h (56). In a study involving long-term administration of oclacitinib, most adverse effects were mild; however, the authors emphasized the need for periodic monitoring of hematological and biochemical parameters and the need for further studies evaluating the safety of long-term therapy in cats. Available studies suggest that higher initial doses than those routinely used in dogs may be effective in cats, which may result from differences in the pharmacokinetic properties of the drug (12, 36). An advantage of oclacitinib is its relatively rapid onset of action, making it a potential alternative in patients requiring rapid control of the inflammatory process. Clinical reports also indicate its potential efficacy in the treatment of selected eosinophilic diseases in cats, including idiopathic periocular eosinophilic furunculosis and vasculitis. Data on the use of oclacitinib in cats with FEGC remain limited and are based mainly on clinical case reports; therefore, further studies are required to evaluate its efficacy and safety in these animals (10).

Antihistamines. Antihistamines may have limited efficacy in cats with allergic diseases (30, 52).

Although the available data concern primarily feline atopic skin syndrome (FASS), in which the therapeutic effect of antihistamines is considered modest, they may also be used as adjunctive treatment in feline eosinophilic granuloma complex (FEGC). The best effects are probably observed in mild or early stages of the disease and in combination with glucocorticoids to maintain their therapeutic effect, rather than during disease exacerbations. Chlorpheniramine is the most commonly recommended drug in this group; however, it should be emphasized that the efficacy of antihistamine treatment in FEGC has not been unequivocally confirmed and its role remains limited. In addition to its antipruritic effect as an H1-receptor blocker, chlorpheniramine may cause sedation and indirectly contribute in reducing stress that may exacerbate clinical signs. For the treatment of pruritus in cats, an initial dose of 2 mg chlorpheniramine per cat twice daily for 14 days is recommened; subsequently, if improvement is achieved, the frequency of administration may be reduced to 2 mg/cat once daily (33).

Polyunsaturated fatty acids. Polyunsaturated fatty acids (PUFAs), particularly omega-3 fatty acids (EPA and DHA), exert anti-inflammatory effects by modulating arachidonic acid metabolism and reducing the synthesis of pro-inflammatory eicosanoids. Their use has been widely described in the treatment of inflammatory and allergic dermatological diseases in cats, including feline eosinophilic granuloma complex, although the available data are mainly indirect (51). In clinical practice, PUFAs are used as adjunctive therapy, particularly in chronic or mild cases. Fatty acid supplementation has been shown to contribute the reduction of the severity of pruritus and improving skin condition in cats with allergic diseases, which supports their use in FEGC as well. It should be emphasized, however, that their efficacy is moderate and the response to treatment varies among individuals (33).

In some cases, spontaneous regression of FEGC lesions is observed without pharmacological treatment, sometimes only after several months (39, 40). Seasonal fluctuations in the severity of clinical signs have also been described, with a tendency toward deterioration during spring and summer, often associated with flea exposure. This suggests that elimination of the triggering factor may lead to spontaneous remission of lesions even without pharmacological therapy (40). The cornerstone of FEGC treatment is the identification and elimination of the triggering factor. Anti-inflammatory and immunomodulatory pharmacotherapy is intended to control clinical signs and should be used concurrently with the treatment of the underlying disease or when its complete elimination is not possible. Despite the possibility of occasional spontaneous resolution of lesions, current recommendations emphasize the importance of early diagnosis and active treatment of FEGC because the disease may be associated with pain and pruritus and may follow a chronic or recurrent course requiring long-term therapeutic control (4, 40, 58).

Feline eosinophilic granuloma complex does not represent a single disease entity but rather a group of cutaneous and mucocutaneous inflammatory reaction patterns with diverse etiologies. Current evidence indicates that these lesions most commonly develop secondarily to allergic diseases, including flea allergy dermatitis, food hypersensitivity, and feline atopic skin syndrome. Although earlier studies demonstrated the presence of autoantibodies against epithelial components in some cats and suggested a possible role of autoallergens, their primary or causal role in the development of FEGC has not been confirmed. The presence of autoantibodies may instead represent a secondary consequence of tissue damage and chronic inflammation. Therefore, FEGC should be regarded as an immune-mediated inflammatory reaction pattern rather than a primary autoimmune disease.

The interpretation of currently available evidence is limited by the relatively small number of controlled studies investigating the pathogenesis and treatment of FEGC, as well as by inconsistent terminology, diagnostic criteria, and methods used to assess treatment response. These limitations make comparisons between studies difficult and highlight the need for standardized diagnostic criteria and well-designed prospective clinical trials.

Clinically, the diagnosis of FEGC should be regarded as a starting point for identifying and, whenever possible, eliminating the underlying disease or triggering factor. Anti-inflammatory and immunomodulatory therapies are intended to control the inflammatory response, and their clinical efficacy should not be interpreted as evidence supporting a primary autoimmune etiology of FEGC.

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