Review Article

Oral Manifestations of Systemic Diseases in Cats

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Abstract

Oral lesions in cats are common disorders that adversely affect quality of life. Certain conditions (eg, stomatitis) may develop very early in life in some purebred cats. These lesions are frequently encountered in geriatric cats and in individuals that are immunosuppressed or have chronic systemic disease. Immunosuppression facilitates the involvement of opportunistic agents, disrupts the microbiota, and contributes to the development of oral lesions. In addition, infectious diseases, metabolic disorders, and multifactorial etiological factors are implicated in their pathogenesis. Many systemic diseases have oral manifestations. These manifestations can be categorized, based on histopathological/clinical features and etiology, into stomatitis, gingivitis, gingivostomatitis, chronic ulcerative stomatitis, lymphoplasmacytic gingivitis/ stomatitis, and feline chronic gingivostomatitis.

Keywords: Oral lesions, feline, feline chronic gingivostomatitis (FCGS), immunosuppression.

Introduction

Oral lesions are serious disorders that significantly impair quality of life and may ultimately lead to death. These lesions often present with common clinical signs. Halitosis is typically associated with volatile sulfur compounds produced by anaerobic bacteria secondary to periodontal disease and plaque accumulation; however, it may also occur in association with gingivitis, periodontitis, stomatitis, bacterial infections, oral neoplasms, chronic viral diseases, oronasal fistula, upper respiratory tract disease, chronic rhinitis, uremic oral lesions secondary to renal failure, eosinophilic granuloma complex, diabetes mellitus, and gastrointestinal disorders. Chronic drooling and ptyalism are most commonly related to oral irritation or dysphagia rather than increased salivary production, and are associated with oral infections, neoplasia, oropharyngeal inflammation, foreign bodies, upper respiratory tract diseases, and toxicity (Clarke and Caiafa, 2014).Additional clinical signs may include dysphagia (dropping food from the mouth, refusal of dry food), anorexia, retching, sneezing, nasal discharge, protrusion of the tongue, pain on opening the mouth, reduced grooming, oral bleeding, and facial asymmetry (Falcão et al., 2020; Wolf, 2006). Oral examination is an integral component of every health assessment and includes evaluation of presenting complaints, general and

extraoral examination, intraoral examination without anesthesia, and, when necessary, detailed assessment under anesthesia. In cats, oral lesions may be inflammatory or neoplastic, and histopathological evaluation is required for definitive diagnosis (Falcão et al., 2020).In metabolic or endocrine disorders, immunosuppression contributes to lesion development. Infections such as Feline Leukemia Virus (FeLV), Feline Immunodeficiency Virus (FIV), Feline Calicivirus (FCV), Feline Infectious Peritonitis (FIP), Feline Herpesvirus Type 1 (FHV-1), panleukopenia virus, Feline Coronavirus (FCoV) and *Hemobartonella henselae*, as well as nutritional disorders, are also associated with oral lesions, and breed predispositions have been reported (Lyon, 2005).

Classification of Oral Lesions

In cats, oral lesions can be classified into the following subcategories: stomatitis, gingivitis, gingivostomatitis, chronic ulcerative stomatitis, lymphoplasmacytic gingivitis/stomatitis, and feline chronic gingivostomatitis.

Stomatitis

Stomatitis is an inflammation of the oral mucosa that may also involve the gingiva and tongue. It is distinguished from gingivitis and periodontitis by its extension to non-gingival mucosal tissues and is typically characterized by marked mucositis in the caudal oral cavity. The reported prevalence in cats ranges from 0.7% to 12% (Jennings et al., 2015).The etiology includes dental calculus, periodontal disease, and infectious agents. Feline rhinotracheitis and FCV are associated with acute stomatitis, whereas FeLV and FIV are linked to chronic forms. In immunosuppressed cats, overgrowth of Candida spp., as well as uremia in certain metabolic disorders, may also contribute to the development of stomatitis. Clinical findings include anorexia, hypersalivation, hyperemia of the oral mucosa, epithelial friability, halitosis, and pain.

Gingivitis

Gingivitis is an inflammation of the gingiva surrounding the teeth. In affected patients, the gingiva is hyperemic and prone to bleeding; in chronic stages, gingival recession may occur, leading to root exposure. The inflammatory process may extend to the gingiva, lips, periodontal region, and other oral tissues. (Figure 1) It commonly develops secondary to alveolar periostitis and dental calculus and may result in clinical signs such as anorexia, salivation, reluctance to eat, and halitosis. The primary cause is plaque-forming bacteria that colonize the gingival sulcus. These bacteria and their by-products penetrate from the sulcular epithelium into the underlying connective tissue, eliciting an inflammatory response. Gingivitis may be acute or chronic. Chronic stimulation can lead to gingival hypertrophy or proliferation. The condition may resolve if the underlying cause is eliminated; however, if left untreated, it can progress to periodontitis (Southerden, 2010). Gingivitis typically precedes and accompanies periodontitis. As the disease progresses, the integrity of the gingiva is compromised, ulceration develops within the sulcus, and bacteria are allowed to invade deeper periodontal structures. In cases of immunosuppression, the progression from gingivitis to periodontitis may be accelerated (Lyon, 2005).



Figure 1: Severe gingival banding in a cat (Original)

Gingivostomatitis

Gingivostomatitis is a common pathological condition, typically characterized by submucosal infiltration of plasma cells. Increased numbers of neutrophils, lymphocytes, and macrophages are also observed. Immunoglobulin levels are elevated in both serum and saliva. The immune response is evaluated based on T-lymphocyte subsets, and the CD4/CD8 ratio is generally decreased due to elevated CD8 levels, indicating an aggressive immune response.In cats, increased expression of cytokine genes, particularly elevated interleukin-4 (IL-4) levels, has been reported. A potential viral association with FCV, FHV, FIV, and FeLV has been suggested, and gene transfer has also been considered as a possible contributing factor. Although the exact etiology remains unclear, gingivostomatitis is defined as a multifactorial

disease involving bacterial, viral, genetic, nutritional, environmental, and domestication-related factors (Lyon, 2005).



Figure 2: Gingivostomatitis in a cat (Original)

Chronic Ulcerative Stomatitis

Chronic Ulcerative Stomatitis (CUS) is a mucocutaneous disorder primarily affecting mucosal surfaces and is characterized by erosive and ulcerative lesions. The tongue is the most commonly affected site, followed by the buccal mucosa and gingival tissues. Less frequently, lesions may be observed on the labial mucosa or hard palate. Clinical signs include halitosis, anorexia, pain-associated vocalization during eating or yawning, approaching the food bowl without eating, and salivation. Varying degrees of inflammation, proliferation, and ulceration are observed in the affected gingiva and oral mucosa. The mucosa is typically bright red, the tissues are fragile, and bleeding occurs easily. The inflammatory process is most often bilateral (Niemiec and FAVD, 2014).

Lymphoplasmacytic gingivitis/stomatitis

Feline lymphocytic–plasmacytic stomatitis (LPGS), also referred to as caudal stomatitis, is an inflammatory disease characterized by painful erosive lesions and proliferative changes in the oral mucosa. This persistent oral condition, with an incidence exceeding 3%, leads to severe inflammation and painful erosive lesions, particularly in the buccal region and caudal oral mucosa. Cats may develop the disease at an early age and may require lifelong management. Clinical signs include ptyalism, pain, halitosis, bleeding, weight loss, and dysphagia, and the condition may necessitate euthanasia due to markedly reduced quality of life. Genetic predisposition, environmental stress, nutritional factors, abnormal immunological responses, and viral and bacterial infections have all been associated with LPGS (Hung et al., 2014). Although LPGS is most commonly observed in middle-aged and older cats, it may occur as early as 6 months of age in Abyssinian, Somali, and certain purebred cats. Lesions may develop along the gingival margins and may be associated with Feline Odontoclastic Resorptive Lesions (FORL). In the presence of FORL, proliferative gingival tissue may be observed. In more severe cases, marked proliferative and painful hyperplastic tissues may be present within the oral cavity. Occasionally, polypoid lesions may develop on the inner surfaces of the lips. Lesions are typically bilateral, a feature that is important in differentiating this condition from oral neoplasms. In the absence of concurrent disease, complete blood count findings are usually unremarkable; however, biochemical profiles often reveal hyperglobulinemia and hyperproteinemia, which are attributed to immunological responses and lymphoid proliferation (Wolf, 2006). Histologically, the disease is characterized by a diffuse oral inflammatory response with ulcerative–proliferative lesions associated with lymphoplasmacytic cell infiltration. Although the exact etiology remains unclear, the development of oral lesions is thought to be associated with inflammation driven by an exaggerated immune response (Fontes et al., 2023).

Feline Chronic Gingivostomatitis

Feline chronic gingivostomatitis (FCGS) is a destructive, chronic, and complex inflammatory mucosal disease affecting approximately 26% of domestic cats. It causes severe oral pain and may be potentially life-threatening in at least 10% of cases (Soltero-Rivera et al., 2024). Affected cats may exhibit decreased appetite, dysphagia, and weight loss (Southerden, 2010). The disease may manifest in three stages: early gingival inflammation during the eruption of deciduous teeth, inflammation triggered by the eruption of permanent teeth, and the most common form observed in cats with a mean age of 7–10 years (Abbasi et al., 2025). It is more frequently reported in Persian, Siamese, Abyssinian, Himalayan, and Burmese breeds, with no reported sex predilection (Sánchez-Vallejo et al., 2025).Recurrence is common, and effective treatment options are limited. Surgical intervention, including extraction of premolar and molar teeth, is often recommended to reduce bacterial load and control inflammation. Ulcerative and/or proliferative lesions are typically localized lateral to the palatoglossal folds (Lee et

al., 2020). Lesions are usually bilateral and are most commonly observed in the glossopalatine arches, fauces, and alveolar mucosa adjacent to the teeth.(Figure 3) Less frequently, they may be found at the base of the tongue, pharynx, palate, and rostral regions of the lips (Fontes et al., 2023). Clinically, proliferative and ulcerative phenotypes are observed. In advanced cases, ulceration, erosion, and marked inflammation of the tongue may develop; tongue lesions can impair eating and drinking, and in some cases, proliferative tissues may prevent tongue retraction (Lee et al., 2020; Sánchez-Vallejo et al., 2025).



Figure 3: Images of Feline Chronic Gingivostomatitis in 2 Different Cats (Original)

The pathogenesis may involve bacteria (*Pasteurella multocida, Bartonella spp., Fusobacterium nucleatum*), viruses (FIV, FHV1, FCV, FeLV), periodontal disease, and upper respiratory tract infections. In addition, $CD8^+$ T cell–mediated immune responses, IL-6 and IL-17–associated pathways, and myeloid cells contribute to disease development; alterations in the oral microbiome facilitate pathogen colonization and modulate the local immune response (Sánchez-Vallejo et al., 2025). Histopathologically, FCGS is characterized by lymphoplasmacytic infiltration. Immunohistochemical studies demonstrate $CD3^+$ T lymphocyte infiltration in the epithelium and subepithelial stroma, whereas $CD20^+$ B lymphocytes are largely confined to the subepithelial stroma. Increased $CD8^+$ T cell levels and a decreased CD4/CD8 ratio suggest a cytotoxic cell-mediated response and immune dysregulation (Lee et al., 2020).In the chronic phase, antibodies derived from plasma cells may activate the complement system, leading to damage of the stratified squamous epithelium, increased vascular permeability, and gingival recession. The typical distribution of oral lesions often supports clinical diagnosis. The disease is a multifactorial inflammatory condition that develops as a result of an inappropriate host immune response to chronic oral antigenic stimulation (Druet and Hennet, 2017).

Infectious Causes of Oral Lesions

Feline Calicivirus Infection (FCV)

Feline calicivirus (FCV) is a virus belonging to the genus Vesivirus within the family Caliciviridae (Hurley and Sykes, 2003). It is widespread and highly contagious within the feline population. FCV can cause severe pneumonia in kittens and, less commonly, in adult cats. It has been associated with feline chronic gingivostomatitis, and infection with virulent systemic FCV (VS-FCV) may result in epizootic spread, particularly in multi-cat environments, and is often fatal (Hofmann-Lehmann et al., 2022).Clinical manifestations vary depending on the viral strain, age, and husbandry conditions. The most common findings include oral ulcerations, particularly on the tongue and palate, along with mild upper respiratory tract signs (Hurley and Sykes, 2003).(Figure 4)



Figure 4: Linguinal ulcer in two separate cats (Original)

While infection may remain subclinical in some cats, others may develop the characteristic lingual ulcer syndrome (Hofmann-Lehmann et al., 2022). An association has been demonstrated between FCV and the lymphoplasmacytic gingivostomatitis (LPGS) complex, with FCV positivity reported in approximately 80% of affected cats (Radford et al., 2007).

Feline Herpesvirus Infection (FHV-1)

The causative agent of viral rhinotracheitis in cats is widespread worldwide and is currently designated as Felid alphaherpesvirus-1 (FHV-1) (Gaskell et al., 2007). The natural routes of entry for FHV-1 are the nasal, oral, and conjunctival mucosa.

Following infection, viral replication during the acute phase occurs primarily in the mucosa of the nasal septum, turbinates, nasopharynx, and tonsils. The conjunctiva, mandibular lymph nodes, and upper trachea are also frequently involved. Infectious virus can be detected in oropharyngeal and nasal swabs approximately 24 hours after infection (Gaskell et al., 2007).FHV-1 is shed in ocular, nasal, and oral secretions, and transmission occurs predominantly through direct contact. Both acutely infected cats and carrier cats represent important sources of infection. Early clinical signs include depression, sneezing, anorexia, and hyperthermia, followed by ocular and serous nasal discharge. Although oral ulceration may occur in FHV-1 infection, it is less common compared with FCV infection (Gaskell et al., 2007).

Feline Immunodeficiency Virus (FIV)

Feline immunodeficiency virus (FIV) is an enveloped RNA virus belonging to the genus Lentivirus within the family Retroviridae (Sykes, 2013). Adult cats are more susceptible than kittens. The virus is present in saliva and is most commonly transmitted through bite wounds. In addition, transmission may occur via blood, saliva-contaminated fomites, grooming of infected animals, as well as through the placenta and milk. Free-roaming male cats and older individuals are at increased risk.The virus replicates in lymphoid tissues and salivary glands and subsequently disseminates throughout the body. As long as the immune system remains competent, clinical signs may not be evident for years. Clinical manifestations are typically observed in the terminal stages and are often associated with secondary disorders, and infected cats remain lifelong carriers. In immunosuppressed cats, infections such as FCV, FHV-1, *Toxoplasma spp., Chlamydia spp.,* and *Hemobartonella spp.* may occur. In these animals, stomatitis, gingivitis, and gingival banding are commonly observed (Figure 5). Some FIV strains exhibit high replication in lymphocytes with limited replication in macrophages, whereas others replicate similarly in both cell types; these differences are associated with variations in clinical presentation (Hartmann, 1998).



Figure 5: Gingival banding in an FIV-positive cat (Original)

Feline Leukaemia Virus (FeLV)

Feline leukemia virus (FeLV) is an enveloped RNA virus belonging to the genus Gammaretrovirus within the family Retroviridae. Three major subtypes have been identified: FeLV-A, FeLV-B, and FeLV-C, each utilizing different cellular receptors for entry. All cats infected with FeLV-B or FeLV-C are also infected with FeLV-A, and FeLV-A is the only subtype transmissible between cats. These subtypes may influence the clinical course of the disease.Clinically, FeLV infection is characterized by immunodeficiency, including leukopenia, lymphopenia, varying degrees of anemia, progressive weight loss, persistent diarrhea, and opportunistic or secondary infections. Chronic and recurrent bacterial or viral upper respiratory tract infections, gingivitis, chronic cutaneous infections, FCV, and FIP may also be observed. In progressive FeLV infection, clinical findings vary depending on the stage of disease and the presence of secondary conditions. While some cats remain asymptomatic, others may exhibit hypothermia, mucosal pallor, petechial hemorrhages, dehydration, peripheral lymphadenomegaly, cachexia, stomatitis, gingivostomatitis, subcutaneous abscesses, and upper respiratory tract signs.The principal clinicopathological abnormalities associated with retroviral infections include anemia, leukopenia, neutropenia, lymphopenia, decreased albumin-to-globulin ratio, and hypergammaglobulinemia. The clinical and pathological features of FeLV and FIV syndromes largely overlap and cannot be reliably distinguished based solely on clinical findings. In cases of co-infection, a synergistic effect on immunosuppression and disease progression may be observed (Hoover and Mullins, 1991). In FeLV infection, opportunistic infections may develop as a result of myelosuppression or acquired cell-mediated immunodeficiency. Impaired neutrophil function further exacerbates the effects of neutropenia, predisposing affected cats to infections such as bacterial infections of the upper and lower urinary tract, hemoplasmosis,

upper respiratory tract infections, FIP, chronic stomatitis, toxoplasmosis, dermatophytosis, and cryptococcosis (Sykes and Hartmann, 2013).

Candidiasis

Oral candidiasis is a fungal infection most commonly caused by *Candida albicans*. The organism is a normal opportunistic component of the flora of the oral mucosa, nasopharynx, gastrointestinal tract, and external genital tract. Although rare in cats, it may occur in regions such as the lips and tongue. It is typically localized to mucocutaneous tissues, remains confined to the epithelium, and does not usually invade deeper structures.The most common predisposing factors include immunosuppression and prolonged antibiotic or corticosteroid therapy. Consequently, it may be more frequently observed in cats undergoing treatment for gingivostomatitis. Oral and perioral candidiasis represent the most common forms of acute mucocutaneous candidiasis. The condition is characterized by small lesions or whitish papules forming a creamy, adherent layer on the tongue, buccal mucosa, and palate. Lesions may occasionally extend to the tongue, palate, and pharynx and reach the commissures of the mouth. Typical findings include white plaques and a tendency for bleeding in the surrounding ulcerated areas (Soto, 2020).

Fungal Cryptococcosis

Cryptococcosis is a deep mycosis that occurs sporadically worldwide in humans and animals and represents the most common systemic fungal infection in cats. The causative agents are most commonly *Cryptococcus neoformans* and *Cryptococcus gattii* (Trivedi et al., 2011). Although the exact mode of transmission is not fully elucidated, infection is believed to occur primarily via inhalation. In cats, the nasal cavity is the initial site of infection, and asymptomatic colonization has been reported (Trivedi et al., 2011).The nasal form is the most common presentation and may disseminate to the skin, subcutaneous tissues, bones, and regional lymph nodes. The resulting fleshy mass may protrude from one or both nostrils. Proliferative or ulcerative lesions, as well as gingival proliferation, may be observed in the oral cavity or pharynx. In some cases, oral lesions may be severe enough to interfere with mouth closure and feeding, necessitating surgical removal (Pennisi et al., 2013).

Bartonella Infection

The most commonly isolated Bartonella species in cats is *Bartonella henselae*. Less frequently, infections with *Bartonella clarridgeiae* or *Bartonella koehlerae* may occur. Bartonella species are Gram-negative bacteria that inhabit erythrocytes and endothelial cells. Isolation is typically achieved by bacteriological culture of blood following partial lysis of erythrocytes (Sykes et al., 2010). Domestic cats serve as the primary reservoir for *B. henselae*. Seroepidemiological studies have demonstrated a worldwide distribution of infection, with antibodies detected in 4–80% of cats. Cats may remain bacteremic for several weeks to several years, with a higher prevalence observed in younger cats (<1 year of age). Direct transmission in flea-free environments and vertical transmission from queen to kitten have not been demonstrated. Warm and humid climates are associated with increased infection rates and ectoparasite infestations. The cat flea (*Ctenocephalides felis*) plays a critical role in transmission (Chomel et al., 2006).Bartonella infection may be associated with gingivostomatitis and is considered to represent a secondary manifestation related to immunosuppression.

Fusobacterium Infection

Fusobacteria are Gram-negative, non-spore-forming, rod-shaped anaerobic bacteria that produce butyric acid as the primary end product of fermentation. *Fusobacterium necrophorum* is an opportunistic pathogen that is part of the normal flora of the oral cavity and gastrointestinal tract and is one of the most commonly isolated anaerobes from abscesses and respiratory tract infections in animals. *F. necrophorum* is associated with a range of necrotic conditions collectively referred to as necrobacillosis. This condition most commonly affects the liver, feet, mammary glands, as well as the oropharynx and oral mucosa. In the oral cavity, it may result in caudal stomatitis characterized by focal lesions ranging in color from white to brown (Nagaraja et al., 2005).

Metabolic Factors

Uraemia and Azotaemia

Uremia is a clinical syndrome characterized by increased blood urea levels and is associated with renal failure and lower urinary tract diseases. It develops as a result of decreased excretion of metabolites in the urine and is accompanied by systemic lesions that disrupt fluid balance and enzymatic processes. The severity and distribution of uremic lesions may vary among species and are generally related to the duration of exposure to uremic toxins (Ambrosio

et al., 2020).In uremia resulting from tubular necrosis, accumulated toxins lead to parenchymal damage and ischemia. Degeneration of the tubular epithelium occurs, and desquamated cells obstruct the tubular lumen. Increased intraluminal pressure triggers tubuloglomerular feedback, leading to afferent arteriolar constriction and a reduction in glomerular filtration rate. Consequently, oliguria or anuria develops, resulting in acute renal failure. In advanced stages, the glomerular filtration rate may decrease to less than 5% of normal, leading to signs of intoxication. Accumulation of uremic toxins, along with disturbances in acid–base and water–electrolyte balance, plays a critical role in the syndrome. Urea accumulating in tissues and mucosa is converted to ammonia by bacterial urease activity, causing mucosal irritation and inflammation, and frequently resulting in erosive–ulcerative stomatitis in cats.Azotemia is characterized by increased concentrations of urea, creatinine, and other nitrogenous compounds in the blood and is most commonly associated with impaired renal filtration. It is classified as prerenal, renal, or postrenal. Prerenal azotemia results from decreased renal perfusion; renal azotemia is associated with parenchymal damage and reduced glomerular filtration rate; and postrenal azotemia occurs due to obstruction of urine flow distal to the kidneys. Increased resistance leads to hydronephrosis and elevated tubular pressure, enhancing urea reabsorption; prolonged obstruction damages the tubular epithelium and decreases the urea-to-creatinine ratio. As in uremia, erosive–ulcerative stomatitis may develop secondary to mucosal irritation in azotemia, with lesions typically localized to the caudal oral region.

Diabetes mellitus

Diabetes mellitus is a metabolic disorder characterized by persistent hyperglycemia, regardless of the underlying cause, and is one of the most common endocrine diseases in cats. Its prevalence varies among populations (Feldman et al., 2014). The disease occurs as type 1 and type 2 diabetes, with immune-mediated type 1 being rare (Nelson and Couto, 2019). The most common form in cats is type 2 diabetes, which develops as a result of a combination of insulin resistance and β-cell dysfunction. In obese cats, the risk of diabetes is increased by approximately 3.9-fold. Clinical signs include polyuria, polydipsia, polyphagia, and weight loss.Oral lesions develop as a consequence of glucotoxicity associated with chronic hyperglycemia. Increased mucosal glucose levels promote bacterial and fungal proliferation, while hyperglycemia impairs neutrophil chemotaxis and phagocytosis, weakening host defenses against anaerobes, *Candida spp.*, and other pathogens. This may result in gingivitis, stomatitis, and glossitis. Microvascular alterations, increased capillary permeability, and impaired perfusion reduce mucosal blood supply, leading to non-healing ulcerative and necrotic lesions.Elevated glucose levels further enhance fungal growth, particularly *Candida albicans*, disrupting the microbial balance; halitosis, plaque accumulation, and tooth loss may occur. Dehydration and reduced salivary defense factors, such as lysozyme, render the mucosa more susceptible to trauma. Increased protein catabolism slows oral tissue regeneration and delays healing. Consequently, gingivitis, ulcerative stomatitis, glossitis, gingival enlargement, periodontal disease, and halitosis may develop.

Multifactorial aetiological factors

In addition to infectious and metabolic factors, multifactorial determinants also play a role in the development of oral lesions (Nunn, 2003). As in humans, the onset and progression of oral lesions in cats are influenced by multiple determinants, including individual characteristics, social and behavioral factors, genetic and systemic influences, features specific to the oral mucosa and microbiota, microbial alterations within dental plaque, and other emerging risk factors.

Age

In humans, aging is associated with stomatitis and periodontal diseases, and this relationship is thought to be linked to progressive periodontal destruction and increased susceptibility to disease over time (Nunn, 2003). Similarly, in cats, advancing age contributes to the development of oral lesions through increased dental plaque accumulation and inadequate oral hygiene.Lymphoplasmacytic gingivostomatitis (LPGS) is most commonly observed in middle-aged and older cats. Feline chronic gingivostomatitis (FCGS) may manifest in three life stages: early gingival inflammation associated with the eruption of deciduous teeth, inflammation triggered by the eruption of permanent teeth, and the most common form observed in cats with a mean age of 7–10 years.

Race and Genetics

Breed represents a complex risk factor for

periodontal disease. In humans, individuals aged 65 years and older of Black ethnicity have been reported to have a higher likelihood of periodontal destruction compared with White individuals, with Prevotella intermedia identified as a risk factor (Nunn, 2003). Differences in the distribution of genetic risk factors among populations may contribute to variations in disease prevalence and severity.Similarly, in cats, certain breeds appear to be predisposed to oral diseases. Lymphoplasmacytic gingivostomatitis (LPGS) is more frequently observed in Abyssinian, Somali, and some purebred cats, whereas feline chronic gingivostomatitis (FCGS) has been reported to be more prevalent in Persian, Siamese, Abyssinian, Himalayan, and Burmese breeds.

Gender

The relationship between sex and periodontal disease is complex. Studies adjusted for oral hygiene, socioeconomic status, and age have demonstrated greater bone loss in males compared with females. In postmenopausal women, estrogen has been suggested to play a role in periodontal disease, with reduced gingival bleeding and inflammation reported in those receiving estrogen therapy (Nunn, 2003). In cats, however, a clear association between sex and periodontal diseases or other oral lesions has not yet been established.

Nutrition

The relationship between nutrition and periodontal disease has been investigated, particularly with respect to calcium and vitamin C intake. According to NHANES III data, low calcium intake is associated with an increased risk of periodontal disease in young men, young women, and older men. Additionally, a study conducted in individuals aged 65 years and older demonstrated that calcium and vitamin D supplementation administered to prevent osteoporosis significantly reduced tooth loss. Within the NHANES III framework, a weak but statistically significant dose–response relationship was identified between vitamin C intake and periodontal disease, with low vitamin C intake reported to increase risk, particularly in smokers (Nunn, 2003).Similarly, in cats, nutritional imbalances may contribute to the development of oral lesions. Wet diets and soft foods may promote tartar formation, thereby accelerating plaque accumulation and the development of infection. Furthermore, vitamin D has been suggested to be associated with gingivostomatitis in cats, and deficiency may represent a risk factor in disease onset; however, further research is required to clarify this relationship (Abbasi et al., 2025).

Social Factors

In humans, socioeconomic status can influence the importance attributed to oral hygiene, and it is well established that smoking and alcohol consumption contribute to its deterioration (Nunn, 2003). Similarly, oral lesions are more frequently observed in cats living in multi-cat households or communal feeding environments. This may be related to the facilitation of transmission of infectious agents through close contact between animals, as well as the generally lower quality of nutrition in such settings.

Microbial Risk Factors

Microbial dental plaque has long been regarded as the primary initiator of periodontal disease. However, not all bacteria within the plaque have been consistently associated with disease progression. The etiological bacteria most commonly identified in periodontal destruction associated with chronic periodontitis include Actinobacillus actinomycetemcomitans, Bacteroides forsythus, and Porphyromonas gingivalis (Nunn, 2003). In a study conducted in cats, the oral microbiota was evaluated, and it was determined that certain microorganisms exhibited increases or decreases in different regions of the oral cavity in cats with feline chronic gingivostomatitis (FCGS).

In conclusion, the recurrent and persistent nature of oral lesions in cats is attributable to the presence of multifactorial etiological factors, including host, agent, and immune-related components. The onset and progression of oral lesions are influenced by numerous determinants, such as individual characteristics, social and behavioral factors, genetic and systemic influences, features specific to the oral mucosa and microbiota, microbial alterations within dental plaque, and other emerging risk factors.Unless the underlying cause is identified and eliminated, lesions tend to recur and become chronic. Therefore, it is essential to address the root cause and establish a treatment protocol accordingly.

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