Oral Inflammation in Small Animals

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**KEYWORDS**
- Oral inflammation
- Small mammals
- Oral cavity

**KEY POINTS**
- In mammalian tissue, inflammation is a highly integrated, elaborate response to insult or injury.
- Its primary purpose is to contain and remove offending microorganisms and necrotic tissue, preventing infection and facilitating tissue healing.
- An aberrant or accentuated inflammatory process can itself cause tissue injury and dysfunction.
- As ongoing research yields an increasing understanding of the cellular and molecular mechanisms that modulate inflammation, efforts to treat and prevent oral inflammatory diseases can become more specific, targeting the precise cells and molecules responsible.

**INTRODUCTION**

The oral cavity can be affected by a wide variety of disorders characterized by recurrent or chronic, generalized or localized inflammation of the oral mucosa and gingiva. Based on their appearance, oral inflammatory lesions may be classified as ulcerative conditions, vesiculobullous diseases, or proliferative lesions (Table 1). Because the oral mucosa has a limited repertoire of responses, however, many different diseases may produce similar manifestations. In particular, vesicles and bullae of canine and feline oral mucosa rarely persist long enough to be observed, due to constant trauma from chewing, playing, and grooming. Therefore, immune-mediated conditions normally producing vesiculobullous lesions may present as ulcerative lesions in the oral cavity. Therefore, the various inflammatory conditions are discussed according to their underlying causes: inflammation associated with dental disease, infectious conditions,

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idiopathic inflammatory responses, mucosal and cutaneous immune-mediated disorders, reactive lesions, and neoplastic conditions.

**INFLAMMATION ASSOCIATED WITH DENTAL DISEASE**

Localized ulceration or swelling limited to the gingiva and alveolar mucosa may be associated with a periodontal or endodontic abscess. Periodontal abscesses are typically associated with swelling and redness of the gingiva surrounding a single tooth, contiguous to a periodontal pocket (Fig. 1A).\(^2\) Gentle pressure on the swollen tissue generally results in expression of purulent exudate. If a draining tract is present, it is coronal to the mucogingival junction. Periodontal abscesses may be associated with regional lymph node enlargement, fever, and acute discomfort.\(^2\) Periodontal

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Fig. 1. (A) Clinical appearance of the right mandibular first and second molar teeth in a 7-year-old spayed female French bulldog. Significant ulceration of the gingiva and alveolar mucosa are evident at the second molar tooth. (B) Radiograph of the same teeth, showing combined horizontal and vertical bone loss with near total loss of attachment of the second molar tooth.
probing and intraoral radiographs confirm the presence of periodontal pockets and alveolar bone loss (see Fig. 1B). Microscopically, a periodontal abscess is a localized accumulation of neutrophils within the periodontal pocket wall. Within the overlying epithelium, intracellular and extracellular edema and leukocyte invasion are evident. Gram-negative anaerobic rods are the primary bacterial colonizers.

By contrast, an endodontic abscess (more properly termed, apical periodontitis) is typically associated with swelling and mucosal inflammation apical to the mucogingival junction, and there may be a draining tract through the mucosa, apical to the mucogingival junction (Fig. 2A). Periodontal probing usually does not reveal the presence of pockets. In dogs and cats, apical periodontitis is most commonly associated with dental fractures, but abrasion and pulp necrosis secondary to concussive trauma are also common causes of apical periodontitis. Although rare, caries may also lead to pulp necrosis and apical periodontitis in dogs.

Radiographically, a diagnosis of apical periodontitis is supported by the presence of any of the following: increased width of the periodontal ligament space in the region of the apex, changes in the trabecular bone pattern around the apex, a diffuse or well-defined periapical radiolucency (see Fig. 2B), arrested deposition of secondary dentin (indicated by a pulp cavity that is wider than that of the contralateral tooth or adjacent teeth), or inflammatory resorption of dental tissue at the apex.

Although periodontal abscesses and apical periodontitis may be diagnosed based on periodontal probing and radiographs, biopsy may be indicated for lesions with slightly atypical features. In some cases, tumors occurring at or near the site of a fractured tooth may go undiagnosed if the tissue swelling and ulceration are assumed inflammatory (Fig. 3).

**INFECTIOUS CONDITIONS**

Acute ulceration of the oral mucosa and/or tongue has been associated with feline calicivirus, feline herpesvirus, feline panleukopenia, feline leukemia virus, feline immunodeficiency virus, and canine parvovirus. (There are also 2 reports from India of fungal-related stomatitis in dogs.) Affected animals almost always display many other symptoms suggestive of systemic illness, such as lethargy, fever, and hematological and serum biochemical abnormalities, so diagnosis is not
based on oral findings, and biopsy is rarely indicated. Treatment is primarily supportive, including appropriate analgesia and parenteral nutrition when oral ulceration is severe. Supplementation with L-lysine has been shown to reduce replication of herpesvirus and reduce the severity of herpesvirus-related conjunctivitis, although its effects on oral symptoms have not been specifically investigated. Recent investigations using feline recombinant interferon, immune plasma, and antiviral agents, such as famcyclovir and plerixafor, have shown promise as more-specific therapeutic options. In the future, targeted gene therapy, such as the use of small interfering RNA, may also be useful in inhibiting viral replication and expediting resolution of acute viral infections.

IDIOPATHIC INFLAMMATORY CONDITIONS

Feline Chronic Gingivostomatitis

Of the oral inflammatory diseases commonly seen in veterinary practice, feline chronic gingivostomatitis (FCGS) has been the most researched, yet its etiology remains largely undetermined. Although several studies have found a higher prevalence of feline calicivirus in cats with FCGS than in nonaffected cats, and transient oral ulceration has been observed in cats with acute calicivirus infection, chronic oral inflammation has not been a sequela in either naturally occurring or experimentally induced acute calicivirus infection. Other microorganisms have been investigated as possible contributing factors to the development of FCGS, including feline immunodeficiency virus, feline leukemia virus, feline herpesvirus, Bartonella henselae, and Pasteurella multocida, but causal relationships have not been established. The presence of plaque bacteria is thought to be a major contributing factor. It is likely that the development of chronic gingivostomatitis is related to an underlying immune abnormality, specifically with regards to the inflammatory mediators produced by lymphocytes and plasma cells in response to bacterial and/or viral infection. Initial histopathologic studies of FCGS revealed that the infiltrates into affected tissue are composed of plasma cells, with varying numbers of lymphocytes, neutrophils, and macrophages present. Abnormalities of neutrophil function were
not detected in affected cats. Mast cells, although present in higher numbers in the gingiva of affected cats, seem to play only a minor role, because there seems to be no significant difference in the numbers of mast cells in the gingiva of cats with FCGS, tooth resorption, or periodontitis.

Serum biochemical changes in affected cats are typically limited to high serum globulin concentrations, composed of a polyclonal hypergammaglobulinemia, which was further classified as composed of high serum IgG, IgM, and IgA. Salivary immunoglobulin concentrations were also evaluated, with the result that cats with FCGS were found to have much higher salivary IgG, moderately higher salivary IgM, and lower salivary IgA than unaffected cats. These findings coincide with recent findings that the majority of plasma cells in the oral mucosa of cats with FCGS were of the IgG isotype. In that same investigation, severity of inflammation was positively correlated with the number of CD97a+ cells (mostly plasma cells), CD3+ T cells, and L1+ cells (primarily neutrophils) and expression of MHC class II proteins in affected tissue. All these studies suggest an underlying aberration in the immune response. The investigators also noted, however, that CD8+ T cells (cytotoxic T cells) greatly outnumbered CD4+ (helper T) cells, suggesting that intracellular pathogens, such as viruses, play a role in the pathogenesis of FCGS.

Clinically, FCGS may appear as generalized or localized areas of ulceration (Fig. 4A) or proliferation (see Fig. 4B) within the oral cavity. Because periodontitis and tooth

Fig. 4. (A) Left maxillary gingiva and buccal mucosa of a 3.5-year-old neutered male domestic shorthair cat who was presented for evaluation of ulcerative stomatitis. (B) Maxillary gingiva and buccal mucosa of a 10-year-old neutered male domestic medium-hair cat who was presented for evaluation of severe proliferative stomatitis. (C) Caudal oral mucosa of a 2.5-year-old spayed female domestic shorthair cat presented for treatment of severe, generalized stomatitis.
resorption may also be associated with generalized or localized inflammation, full-mouth radiographs and dental charting are important to distinguish between these 3 conditions. The presence of inflammation in the caudal oral cavity (ie, the areas lateral to the palatoglossal folds, sometimes incorrectly referred to as the fauces) and/or the oropharynx is one of the distinguishing characteristics of FCGS (see Fig. 4C).

Patients with periodontitis and/or tooth resorption alone do not have inflammation in these caudal areas. All 3 diseases, however, can be present concomitantly (Fig. 5).

Successful treatment of FCGS requires minimizing oral bacteria. Because daily plaque removal by mechanical means (eg, toothbrushing) is difficult in these painful patients, reduction of plaque-retentive surfaces by extracting teeth has proved the most effective way to minimize plaque and reduce oral inflammation. It has been demonstrated that 60% to 80% of cats with lymphocytic-plasmacytic gingivitis stomatitis significantly improve after extraction of all premolar and molar teeth, and surgical treatment is, therefore, the current standard of care for cats with FCGS. Those cats that do not respond to premolar/molar or full-mouth extractions, however, present a therapeutic challenge. Because glucocorticoids have immunosuppressive effects (which include decreasing neutrophil diapedesis, redistributing lymphocytes to extravascular compartments, and down-regulating maturation of antigen-presenting cells) and are easily accessible and inexpensive, they remain the most commonly prescribed medication for management of refractory stomatitis. The beneficial effects of steroid administration are inconsistent, however, and may be accompanied by deleterious effects, such as behavior changes, thinning of the skin, polyuria, polydipsia, and potential for development of diabetes mellitus. Therefore, alternative treatments are sought. One option is cyclosporine, which has recently been Food and Drug Administration–approved for use in cats and is available in a liquid suspension (Atopica for Cats, Novartis Animal Health, Greensboro, North Carolina) which allows more precise dosing and is easier to administer than the capsule form commonly prescribed for dogs (Atopica for Dogs, Novartis Animal Health, Greensboro, North Carolina). In one retrospective analysis featuring 8 cats with FCGS who received oral cyclosporine (Sandimmune solution, Novartis Pharmaceutical Corporation, East Hanover, New Jersey) at 30 mg to 50 mg daily, 50% achieved remission of inflammation after 90 days, and the remaining 50% showed fair to good improvement of 40% to 70%. Although it was reported that all cats in this study had previously received injectable steroids, it was not noted whether these cats had previously undergone periodontal treatment or dental extractions. In a prospective, placebo-controlled study of 16 cats with refractory FCGS (ie, those who had not completely responded to premolar/molar or full-mouth extractions), approximately 78%
demonstrated improvement of 40% or more after 6 weeks of treatment compared with 14% of control cats; mean improvement in the cyclosporine group was 52.7% after 6 weeks. Side effects are usually mild and consist primarily of transient vomiting or diarrhea. Cyclosporine administration is not without risk, however, particularly for outdoor cats, because disseminated toxoplasmosis has been reported.

In 2011, a multicenter, controlled, double-masked European investigation revealed that recombinant feline interferon omega delivered transmucosally was as effective as prednisolone in decreasing clinical lesions and pain scores. With no significant deleterious side effects reported, this product is expected to be widely used once it becomes commercially available in the United States.

**Contact Mucositis (Plaque-reactive Stomatitis)**

Although cats are more frequently affected by stomatitis, an increasing number of dogs is being presented with symptoms, such as ptyalism, halitosis, decreased ability to prehend hard food, and reluctance to play with chew toys. General physical examination may reveal atrophy of the masticatory muscles and weight loss. Patients may be extremely reluctant to allow oral examination without sedation or anesthesia. Oral examination typically reveals ulceration of the vestibular (buccal) mucosa in areas that contact the tooth surfaces, particularly at the carnassial and canine teeth. Although histopathologic analysis of the lesions may reveal lymphocytes and plasma cells in the epithelium and lamina propria similar to that found in cats with FCGS, this syndrome (previously referred to as chronic ulcerative paradental stomatitis) differs from feline gingivostomatitis in that the lesions are almost exclusively localized to the areas in contact with the teeth and do not typically involve the caudal oral mucosa or the oropharyngeal mucosa. In some cases, severe ulceration occurs in the absence of significant periodontitis (Fig. 6). In other cases, periodontitis may be evident based on clinical findings of severe gingival recession and/or radiographic findings of bone loss (Fig. 7).

As with cats, treatment of contact mucositis in dogs relies on effective plaque control. Although many dogs are amenable to daily toothbrushing, the discomfort associated with contact mucositis may make these patients uncooperative for home care. Professional periodontal treatment is essential, including extraction of any teeth demonstrating significant bone loss. This should be followed by administration of analgesic and anti-inflammatory medications, which may provide enough relief to enable

![Fig. 6](image-url) (A) Ulcerative stomatitis featuring gingivitis, buccal mucositis, and glossitis in a 7-year-old spayed female shepherd mix. The lateral margins of the tongue were affected in the regions that contacted the lingual surfaces of the mandibular teeth. (B) Intraoral radiograph of the right maxillary premolar and molar teeth of the same dog. Replacement resorption is evident at the first premolar tooth, and there is evidence of inflammatory resorption at the third and fourth premolar teeth, but periodontal bone levels are near normal, with approximately 1 mm of horizontal bone loss apparent.
initial attempts to remove plaque using wet gauze on a finger. Chlorhexidine-based rinses and gels or drinking water additives that have demonstrated efficacy in reducing plaque (eg, products that have received the Seal of Acceptance by the Veterinary Oral Health Council) may be helpful adjuncts to toothbrushing. If these measures fail to resolve the areas of inflammation, medical management (as discussed previously, cyclosporine has fewer side effects than glucocorticoids and is, therefore, preferred) and/or surgical treatment by removing all teeth in the affected areas may be required. In the author’s experience, a combination of selective extractions, professional dental cleaning at 3-month to 6-month intervals, and diligent home care is usually sufficient to prevent recurrence of contact ulcers. In several canine patients for whom home care was not feasible and long-term immunosuppressive medication was undesirable, however, extraction of all premolar and molar teeth was performed, resulting in resolution of the inflammation (Fig. 8).

**MUCOSAL AND CUTANEOUS DISEASES**

**Eosinophilic Granuloma Complex**

Eosinophilic granuloma complex is a common disorder in cats, affecting the skin (eosinophilic plaque), upper lip (indolent ulcer) (Fig. 9), palate, and/or tongue (eosinophilic granuloma). Although rare, eosinophilic lesions have been described in the oral cavity of dogs as well, who may be presented with symptoms of clearing the throat, difficulty swallowing, coughing during and after eating, or difficulty eating. Rather than a disease, eosinophilic dermatoses should be thought of as a reaction pattern to a variety of different stimuli. Histologic findings are typical, with an eosinophilic infiltrate and a variable number of mast cells, histiocytes, and lymphocytes.
Feline herpesvirus-1 may occasionally result in skin or oral lesions resembling eosinophilic plaques or ulcers. The most common underlying cause is a hypersensitivity reaction to environmental antigens, foods, or parasites, and it is important to attempt to identify and address the underlying cause before administering immunosuppressive medications, such as glucocorticoids or cyclosporine. In some cases, treatment with amoxicillin trihydrate–potassium clavulanate alone may result in near-resolution of eosinophilic plaques or indolent ulcers.

Erythema Multiforme

Erythema multiforme (EM) is a rare disorder, leading to vesicular and ulcerative lesions on skin and mucous membranes, with some histologic findings typical of pleocellular inflammation but featuring characteristic keratinocyte apoptosis and lymphocyte satellitosis. It is hypothesized that EM results from a host-specific cytotoxic T-lymphocyte attack on keratinocytes expressing nonself antigens, typically microbes and drugs. In many cases, EM is initiated by administration of medications. In some cases, viral infection of skin and mucosal epithelial cells results in activation of cytotoxic T lymphocytes, which then induces keratinocyte apoptosis. In dogs, the oral cavity is involved in approximately one-third of cases with EM, and dysphagia and/or ptalism secondary to oral ulceration may be the primary complaint.
at presentation. In some cases, it may be difficult to distinguish EM from epitheliotrophic lymphoma, and immunohistochemistry may be required to identify neoplastic intraepithelial CD3+ T cells within a focus of pleocellular inflammation. Proper diagnosis is crucial, because the prognosis for epitheliotrophic lymphoma is poor, whereas EM may resolve once the triggering factor is removed. In the future, administration of intravenous immunoglobulins may comprise part of the treatment plan for EM patients.

**Pemphigus Foliaceous**

Pemphigus foliaceous is most common antibody-mediated, autoimmune skin disease of dogs, but mucosal lesions are rare, reported in approximately 2% of dogs with pemphigus foliaceous. A major antigen responsible was recently identified as desmocollin-1, a calcium-dependent transmembrane glycoprotein involved in intercellular adhesion. Clinical features include facial and footpad lesions consisting of vesicles and pustules (which evolve rapidly to erosions covered with crusts). Histopathology reveals acantholytic keratinocytes accompanied by neutrophilic infiltration and a variable number of eosinophils. The prognosis is generally good, because patients usually respond to immunosuppressive doses of corticosteroids (2 mg/kg/d).

**Pemphigus Vulgaris**

Although rare, lesions of pemphigus vulgaris may first develop in the oral cavity or at mucocutaneous junctions and then spread to haired skin. German shepherd dogs and collies may be predisposed, and male dogs predominate. Vesicles evolve rapidly into irregular erosions and areas of sloughing. The prognosis is guarded and referral should be made to an internist or dermatologist to discuss appropriate treatment options.

**Mucous Membrane Pemphigoid**

Vesicles, erosions, and ulcers are seen primarily in or around the oral cavity, nasal plane, eyes, ear canals, anus, and genitals. German shepherd dogs may be predisposed to mucous membrane pemphigoid. IgG autoantibodies directed against basement membrane proteins result in subepidermal and submucosal vesiculation, with few inflammatory cells.

**Epidermolysis Bullosa Acquisita**

Epidermolysis bullosa acquisita is characterized by severe clinical signs, including sloughing of the oral epithelium and footpads, and carries a poor prognosis. Great Danes are overrepresented. Autoantibodies target collagen VII, resulting in subepidermal and submucosal vesicles without inflammation or with subepidermal alignment of neutrophils. Immunohistochemistry is required to differentiate epidermolysis bullosa acquisita from mucous membrane pemphigoid or bullous pemphigoid (which does not usually present with oral mucosal ulceration).

**Systemic Lupus Erythematosus**

It is unusual for patients with systemic lupus erythematosus to present with primarily oral signs, because joint pain and stiffness (attributable to polyarthritis), together with dermatitis, are more common presenting complaints. Erythematous, crusty skin lesions on the face, and ulceration of the lip margins may be apparent (Fig. 10). Affected animals test positive for circulating antinuclear antibody.
REACTIVE LESIONS

In addition to the intrinsic immune disturbances (described previously), many external stimuli can result in oral inflammation. When attempting to determine the underlying cause of an oral inflammatory lesion, whether the lesion is localized or generalized within the oral cavity and whether it is ulcerative or proliferative should be considered.

Generalized ulcerative lesions may result from viral infections (in particular, feline calicivirus, as discussed previously), chemical exposure, administration of pancreatic enzyme supplements, or uremia. Obtaining a thorough history, which includes travel history, exposure to toxins, known traumatic incidents, and administration of any dietary supplements as well as prescription medications, is important whenever patients present with evidence of oral ulceration. In most patients with generalized oral ulceration caused by viral infection, chemical exposure, or uremia, the oral signs are not the primary complaint, and patients display other significant symptoms that direct the path of diagnostic testing.

Generalized proliferative lesions may include viral papillomatosis, drug-induced gingival enlargement, and familial gingival hyperplasia.

A localized ulcer may result from a penetrating wound, an electrical injury (Fig. 11), or eosinophilic indolent ulcer.

Conditions presenting with localized proliferation of oral mucosa include focal fibrous hyperplasia, foreign body reaction, and sublingual mucosal hyperplasia (Fig. 12). Sublingual mucosal hyperplasia may be both proliferative and ulcerated, depending on whether there is masticatory trauma, and is usually bilaterally symmetric, although one side may be larger than the other. Excisional biopsy not only yields a diagnosis but also in most cases is curative.

Treatment depends on the underlying cause. Localized ulcerative lesions may respond well to débridement and supportive care. In the future, stem cell therapy may play a role in the treatment of oral inflammation, because local injection of mesenchymal stem cells derived from bone marrow was found to accelerate the healing of chemically induced oral ulcers in an experimental model using dogs.
NEOPLASTIC LESIONS

Although many oral neoplasms appear as masses, several may first present as ulcerations or even simply as erythematous areas on the gingiva or oral mucosa. Squamous cell carcinoma (Fig. 13) in cats and epitheliotrophic lymphoma in dogs are 2 malignant neoplasms that commonly have an ulcerative rather than exophytic appearance. Biopsy and histopathologic analysis is recommended for any abnormal-appearing tissue in the oral cavity, in particular, nonhealing extraction sites; early diagnosis and appropriate intervention may mean the difference between a satisfactory and unsatisfactory outcome.

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**Fig. 11.** (A) Ulcerative, necrotic gingival, and mucosal lesion affecting the left mandibular fourth premolar and first molar teeth of a 10-year-old neutered male Weimaraner. Radiographs were unremarkable. The lesion was biopsied and débrided. Histopathology revealed no evidence of neoplasia, with changes suggestive of electrical cord injury. (B) Despite complete loss of the attached gingiva and alveolar mucosa at the buccal aspect of the fourth premolar and first molar teeth, 6 weeks later the area had healed completely and appeared almost normal.

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**Fig. 12.** Sublingual mucosal hyperplasia in a 10-year-old spayed female bichon frise. These lesions are usually bilateral and tend to occur in small breed dogs. Surgical excision is usually only performed if they are inflamed and/or are traumatized during mastication.88
SUMMARY

In mammalian tissue, inflammation is a highly integrated, elaborate response to insult or injury. Its primary purpose is to contain and remove offending microorganisms and necrotic tissue, preventing infection and facilitating tissue healing.\(^8\) An aberrant or accentuated inflammatory process, however, itself can cause tissue injury and dysfunction. As ongoing research yields an increasing understanding of the cellular and molecular mechanisms that modulate inflammation, efforts to treat and prevent oral inflammatory diseases can become more specific, targeting the precise cells and molecules responsible.

REFERENCES


Fig. 13. (A) Clinical and (B) radiographic appearance of an ulcerative lesion at the site of a recent tooth extraction (the right maxillary third premolar tooth, P3). The radiograph reveals subtle permissive and subtle moth-eaten osteolysis in the region extending from the region of the missing P3 to the (also missing) canine tooth site. Histopathologic analysis of bone and soft tissue collected from the P3 site confirmed a diagnosis of squamous cell carcinoma.


