A Differential for Oral Ulcers in Cats

Susan C. Stover

Ulceration of the oral mucosa in cats can be a sign of many different diseases or conditions. Possible causes include chemical irritation, trauma, neoplasia, eosinophilic granuloma complex, uremia, viral infections of the upper respiratory tract, gingivitis and periodontal disease, acute necrotizing ulcerative gingivitis, mycotic infection, pemphigus, systemic lupus erythematosus, and plasma cell pododermatitis. These conditions can be very difficult to distinguish on clinical appearance alone, and even the results of appropriate tests may be inconclusive.

The Oral Environment

The normal flora of the oral cavity includes a combination of aerobes, anaerobes, facultative organisms, and spirochetes. Therefore, bacterial culture of an oral lesion is often clinically meaningless. The organisms which grow on culture may not be those initially responsible for the lesion. There is also evidence that the alkaline pH and lysosomal activity of saliva combine to produce a bactericidal action. If a microorganism is suspected as the cause of the lesion, antibiotics should be selected with consideration of the normal oral flora.

The Oral Exam

The signs which may prompt an owner to present a cat for an examination are anorexia, dysphagia, halitosis, hypersalivation, pawing at the mouth, and other signs of pain. The onset and progression of signs may be helpful. However, cats may show very few signs, especially with early lesions.

When a cat is presented for an oral lesion, a detailed history and a complete physical exam will help determine systemic conditions and specific syndromes. Carefully examine other mucocutaneous junctions for the presence of other lesions, and check the size of the mandibular lymph nodes. Also, inspect the mouth for foreign bodies, lacerations, burns, and other signs of trauma. Because of a cat’s temperament or response to pain, sedation or anesthesia may be necessary to do a thorough oral cavity exam.

The nature of the lesions (i.e. position, extent, and symmetry) can help to differentiate various conditions. Diseased periodontium can cause ulcers on the buccal mucosa. Ulcers scattered over the mucosa usually indicate a viral infection or chemical irritation. A single asymmetric ulcerated lesion may be caused by neoplasia.

A complete blood count, feline leukemia test, renal function tests, and blood glucose level may be beneficial. If there is adequate suspicion, bacterial or mycotic cultures can be taken. However, be careful not to misinterpret
the results with respect to the normal oral flora.

Radiographs may be beneficial in demonstrating neoplastic disease. A biopsy for routine histopathology and fluorescent antibody testing may provide additional information.

**Chemical Irritation and Trauma**

Although cats are usually fastidious eaters, they can acquire oral lesions as a result of their curiosity or grooming habits. Careful questioning of the owner can often provide a history of plant ingestion or exposure to unusual substances.

*Diffenbachia house plants* can cause severe irritation and ulceration in the mouth. The combination of calcium oxalate crystals and a histamine-releasing substance produces hyper-salivation and pharyngeal edema which leads to dyspnea and dysphagia. Antihistamines may be beneficial, and tube feeding may be necessary for a few days until the lesions resolve.

Chewing on pine needles or drinking water from the base of a Christmas tree stand can result in *pine tar ingestion*. This causes stomatitis, pharyngitis, gastroenteritis, emesis, diarrhea, lethargy, and inanition. Treatment consists of removing any remaining material from the gastrointestinal tract and providing supportive therapy.

*Poinsettias* also contain an irritant sap which is reported to cause stomatitis, keratoconjunctivitis, and gastroenteritis.

*Strong acids or bases* which are licked from the cat’s hair can cause mucosal ulceration. Treatment consists of removing the agent from the hair and skin by washing with large amounts of water. The residue can be neutralized with either vinegar or sodium bicarbonate, as appropriate. Protect damaged skin with an antibiotic ointment and bandages as necessary. Supportive care can include parenteral fluids, tube feeding, and B vitamins until the lesions have healed. Oral lesions can be flushed with kaolin bismuth or mineral oil.

**Neoplasia**

The most common tumor which occurs in the oral cavity of the cat is *squamous cell carcinoma*. This tumor usually occurs on the gingiva and tongue. It is locally invasive and will usually recur after excision. The tumor can be both proliferative and ulcerative and causes blood tinged saliva, halitosis, pain, and reluctance to eat. In the early stages, squamous cell carcinoma may be white or pink nodules, appear as a proliferation of gingival tissue with loosening of teeth, or resemble granulation tissue, eosinophilic granuloma, or even trauma.4

The early stages, however, are usually asymptomatic and go unnoticed. In the later stages, nodules are usually white to yellow to pink, very firm, and often ulcerated.1 One needs to differentiate these lesions from gingival hypertrophy which can also become quite large. Definitive diagnosis is possible with a biopsy. Radiographs may be suggestive if there is sufficient bony involvement. Treatment choices are surgical excision, cryosurgery, and mandibular resection.4 7 1

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Feline Health Topics

*A publication for veterinary professionals*

The ultimate purpose of the Cornell Feline Health Center is to improve the health of cats everywhere, by developing methods to prevent or cure feline diseases, and by providing continuing education to veterinarians and cat owners. All contributions are tax-deductible.

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Two less common oral tumors in cats are fibrosarcoma and ameloblastoma. Fibrosarcoma may occur on the mandibular gingiva or in the region of the upper molars. Ameloblastoma arises from dental primordium of the enamel organ and dental lamina. The ameloblastoma usually ulcerates only if it becomes very large.

Eosinophilic Granuloma Complex

The eosinophilic granuloma complex is an arbitrary grouping of several skin and mucous membrane diseases of cats. Neither tissue eosinophilia or blood eosinophilia are necessarily characteristic of these conditions.

The indolent ulcer, eosinophilic ulcer or rodent ulcer is a well circumscribed, red-brown alopecic, glistening ulcerated lesion on the upper lip of cats. It can also occur on the oral mucosa, lower lip, tongue, and skin. The lesion is usually non-pruritic and not painful. It is most often seen in female cats five to six years old.

The cause of the lesion is unknown. Suggested causes for the lesions are constant irritation (i.e. rough tongue or sharp canine tooth), low-grade bacterial infection, or chronic allergy leading to an erosion and eventually an ulcer. The lesion could also be a precancerous lesion which is capable of undergoing malignant transformation to squamous cell carcinoma. No pathogenic bacteria, fungi, or viruses have been isolated from the lesions.

The lesion’s clinical features and its location on the lip is indicative of indolent ulcer. Be sure to examine the cat thoroughly for other skin lesions. It is important to differentiate indolent ulcers from squamous cell carcinoma.

Indolent ulcers usually respond to corticosteroid therapy, especially if treated early. Oral prednisolone or methylprednisolone acetate injected subcutaneously can be used as required. Recurrent lesions may require treatment with doses every other day of oral prednisolone.

The lesions of eosinophilic plaque usually occur on the abdomen and medial thigh, but they can also occur in the oral cavity. Tissue and blood eosinophilia are usually found, although the lesions closely resemble psychogenic dermatitis. The lesions are red, ulcerated, and distinctly circumscribed. There may be several smaller lesions which coalesce into larger ones.

Treatment of eosinophilic plaque is similar to indolent ulcer, although higher doses may be required.

Linear granulomas are well circumscribed, firm, raised, yellow-pink plaques. They occur in a distinct line on the caudal aspect of the cat’s hind limbs. They may also occur on the lips, tongue, gums, and palate. The prognosis is guarded when peripheral eosinophilia occurs with oral lesions.

If cutaneous lesions are present, the condition can be diagnosed by its clinical appearance. However, diagnosis is difficult if only oral lesions are present. Treatment is similar to that for eosinophilic granuloma. Some linear granulomas may regress spontaneously, but if oral lesions are present, high doses of prednisolone are required. Some cats may require intermittent lifetime therapy.

Uremic Ulcers

Uremic ulcers result from any condition which causes renal failure. The high levels of urea in the blood diffuse into body secretions such as saliva. The action of oral microorganisms changes the urea into ammonia. The ammonia combined with the dry mouth and clotting deficiencies of the dehydrated uremic patient causes mucosal erosions and eventual ulcerations.

The presence of uremic ulcers may heighten suspicion of renal failure even before laboratory data is available.

Viral Infections

Several investigators now claim that mucosal ulcers are probably caused by a dual infection with calicivirus, even though the clinical signs are consistent with herpes virus infection. Clinical signs aid in diagnosis of viral upper respiratory infections.

Calicivirus infection can produce two different forms of ulcers. Low virulence strains may produce few clinical signs -- ulcers of the
Feline Health Topics

tongue, hard palate, and nose, and little or no pyrexia. High virulence strains produce signs of pyrexia, anorexia, depression, dyspnea, pneumonia, oral lesions, and death.

Cats develop the disease upon initial exposure to the calicivirus, but may also relapse following stress or immunosuppressive disease. One theory is that the infection sets up a chronic ulcerative glossitis. This is characterized by recurrent bouts of hypersalivation and halitosis. During this time the cat may not eat or eat only soft food and may not groom itself. The glossitis may be treated once the viral infection is past with thorough teeth cleaning and an antiseptic mouthwash for several days.

Administer antibiotics (ampicillin or amoxicillin drops) to prevent bacterial infections secondary to calicivirus infections. Also, feeding warm, tantalizing liquid food encourages eating.

Oral ulcers are usually seen in herpes virus infection only when there are severe signs of upper respiratory infection. These signs include fever, blepharospasm, paroxysmal sneezing, head shaking, serous to mucopurulent nasal discharge, hypersalivation, anorexia, and malaise. Countereact secondary bacterial infection, dehydration, respiratory distress, and inanition by providing vigorous supportive therapy. This includes antibiotics, fluids, vitamins, cleansing of the nose and eyes, steam vaporization, and good nursing care which may be more beneficial at home.

Gingivitis and Periodontal Disease

Gingivitis is an inflammation of the perialveolar gum margins due to an accumulation of dental plaque and tartar. The dental tartar results from the accumulation and deposition of by-products from bacterial breakdown of food. Its development depends on diet, the resident microflora, and the animal’s chewing habits. The excessive accumulation leads to gingival damage, and an alteration in the number and type of microflora. The mucosal integrity is damaged and ulcerations form, or gingival hyperplasia can lead to traumatized tissue which then bleeds and becomes ulcerated. The best treatment is scaling of tartar and extraction of damaged teeth. Sharp excision of hypertrophied tissue is not recommended.

Acute Necrotizing Ulcerative Gingivitis

Acute necrotizing ulcerative gingivostomatitis (ANUG) is usually seen in association with an acquired immunosuppressive disease such as diabetes mellitus, persistent neutropenia, feline T-lymphotropic virus infection or feline leukemia virus infection. The prominent signs are a reluctance to eat, hypersalivation, halitosis and pain on opening the mouth. On physical exam there may be a mass of gray necrotic tissue covering an ulcerated area that bleeds easily when touched.

Many organisms have been cultured from ANUG lesions and tend to parallel the usual organisms found in the mouth. Therefore, such cultures are not helpful. Diagnosis is by clinical findings, the presence of excessive dental tartar, and evidence of immunosuppressive systemic disease on routine testing (hematology, biochemistry, and FeLV tests). Treatment consists of dealing with the primary systemic disease if present, removing diseased teeth and tartar, flushing with 1% hydrogen peroxide, and administering antibiotics (ampicillin, clindamycin).

Occasionally ANUG may lead to a chronic stomatitis that is both proliferative and ulcerative. This is usually seen in the fauces. On histopathology, the lesions show lymphoplasmacytic or eosinophilic infiltrates. The signs are similar, but the lesion is unresponsive to therapy. The chronic nature of the disease results in proliferation of granulation tissue which may require surgical removal and biopsy to rule out neoplastic disease. Removal of the most caudal molars (upper and lower) may relieve some associated pain. Some lesions may develop into squamous cell carcinoma.

Symptomatic treatment consists of a hypoallergenic diet, antibiotics, levamisole, progesterone, and topical mouthwashes. As a last resort, a local injection of corticosteroids may be used.

Mycotic Stomatitis

Candidiasis is a rare but severe cause of ulcer-
ative stomatitis. It usually occurs in debilitated or immune-suppressed animals or after long term use of corticosteroids or antibiotics. The lesions are irregular ulcerated areas surrounded by a zone of inflamed mucosa. They may be diagnosed by fungal culture. Ketoconazole is the current drug of choice.

Autoimmune Disease

Pemphigus is an autoimmune disease that results from the production of an autoantibody against the glycocalyx of keratinocytes. When the autoantibody binds to the keratinocytes, a type II hypersensitivity reaction results which causes hydrolysis of the glycocalyx and intra-epidermal acantholysis. The clinical result is a chronic blistering disease of the skin and mucous membranes. Most patients are presented for chronic ulceration and inflammation of oral mucosa, skin, nasal philtrum, and mucocutaneous junctions.

Biopsies of early lesions (if available) will aid in diagnosis. Collect two sets of tissues—one in formalin and one in Michel's fixative. Direct immunofluorescence of tissue is preferred; since low or negative antibody titers in the serum may be difficult to interpret in an indirect test.

Pemphigus vulgaris is a vesiculobullous disorder of the oral cavity, lips, nostrils, and philtrum. The vesicles are fragile and easily ruptured to leave erosions and ulcers bordered by epidermal collarettes. As with any disruption of the mucosa, a secondary pyoderma is common. Severely affected cats may be anorectic, depressed, and febrile.

Pemphigus foliaceous usually begins on the face and feet, then generalizes. Only rarely does it produce oral lesions. Also, the vesicles are rarely seen. Secondary signs include erythema, oozing, scales, alopecia, and erosions with epidermal collarettes. Routine laboratory tests are non-specific. Diagnosis is by history, physical exam, direct smears (non-degenerative neutrophils and numerous clustered acanthocytes), mucosal biopsy, or immunofluorescence testing. Once pemphigus foliaceous has been diagnosed, the prognosis is not clear since too few cases have been followed.

Treatment for both types of pemphigus includes systemic glucocorticoids, immunomodulating drugs such as cyclophosphamide, azathioprine, and chlorambucil, or gold.

Systemic lupus erythematosus is a disease with several possible etiologies. Suggested factors are genetic, immune, and viral. The pathogenesis of the disease involves a type III hypersensitivity reaction characterized by a wide variety of autoantibodies and widespread organ involvement. The cutaneous manifestations of the disease are diverse and include seborrhea, cutaneous and/or mucocutaneous vesiculobullous ulcers of the nostrils or lips, footpad ulcers and hyperkeratosis, discoid lupus erythematosus, secondary pyoderma, and panniculitis. One case report of systemic lupus describes the chief complaint as a chronic, non-healing, ulcerative stomatitis.

Diagnosis of this condition relies on the clinical picture together with diagnostic elimination for other diseases and positive results on antinuclear antibody testing and/or positive fluorescence on a direct immunofluorescence antibody test of a tissue specimen.

Treatment of systemic lupus erythematosus is similar to that for pemphigus.

Plasma Cell Pododermatitis

Plasma cell pododermatitis is a rare disorder of cats with an unknown etiology. However, an immunologic basis is suspected based on the presence of plasma cells in the tissues, its seasonal recurrence, and response to immunomodulating treatment.

The footpad lesions begin as soft, painless swellings. These lesions progress to ulcerations and exuberant granulation tissue. There may be concurrent oral lesions or oral lesions alone which are characterized by a bilateral proliferative ulceration at the commissures of the mouth.

A thorough exam of the entire cat may help differentiate plasma cell pododermatitis lesions (continued on page 8)
Megestrol Acetate Therapy in the Cat

Susan M. Szczotka

Megestrol acetate is manufactured in the United States by Schering (Ovaban, 5 and 20 mg tablets) and by Mead Johnson (Megace, 20 mg tablets). It was first used in veterinary medicine in 1975 to control estrus in the bitch. Since then, the application of this drug has expanded tremendously. It is now used for a wide variety of diseases and behavior problems in several animal species, including the cat. However, its use in the cat is not legally approved.

Uses

Reproduction: Megestrol acetate is used in cats for estrus suppression, estrus prevention, and the possible prevention of conception after mating. Administration of megestrol acetate after the onset of clinical signs of estrus may shorten the clinical course of estrus, and if continued, can prevent subsequent estrus cycles. If administered shortly after an undesired mating, megestrol acetate has been reported to prevent pregnancy.

Behavior Modification: Progestin therapy has been used to treat spraying and marking, male aggression, sexual behavior in toms, hyperactivity, and other forms of abnormal behavior. It has been most successful in treating behavior problems that are related to sexually dimorphic behavior. The anti-androgenic effects of the drug are very similar to the effects of castration. Therefore, progestins may help reduce or eliminate male roaming, fighting, and urine marking.

Dermatologic Disorders: Megestrol acetate has been widely used in treating eosinophilic granuloma complex, feline endocrine alopecia, feline acne, stud tail, psychogenic alopecia, and milimentary dermatitis.

Miscellaneous: Other diseases treated with megestrol acetate are eosinophilic keratitis, feline hyperesthesia syndrome, and chronic unresponsive feline urologic syndrome.

Side Effects

There are a myriad of side effects associated with megestrol acetate therapy in the cat. Most of these adverse reactions are seen with high doses and long periods of treatment. However, side effects have also been reported with short term use and low dosage levels. Changes in temperament are very frequent. Cats may exhibit increased affection, friendliness and tranquility, sometimes as profound as to be considered lethargy or depression. Increased appetite with subsequent weight gain is common, as is polyuria and polydipsia. Megestrol acetate decreases spermatogenesis, thereby reducing fertility. Hence, its use in breeding toms is contraindicated. Toms on progestogen therapy may lose social order within large colonies. This loss of status was not seen in toms that were castrated.

A serious side effect is pyometra or stump pyometra with associated cystic endometritis. These uterine changes are seen in intact females or in females where incomplete ovariohysterectomies have been performed. Usually there is also a history of long term treatment with progestogens. However, some reports indicate that progestogen treatment had not been excessive in either dose or duration. The incidence of pyometra associated with progestogen use is great enough to warrant extreme caution in the use of this drug, especially in intact females.

Feline mammary hypertrophy is a benign, but abnormal, rapid growth of mammary tissue. It may occur in early pregnancy, in females experiencing their first estrus period, and in both males and females after long term administration of progestogen compounds.

Identification of steroid hormone receptors in hypertrophied mammary tissue indicated the presence of progesterone receptor patterns similar to those seen in feline mammary adeno-
carcinomas. This finding demonstrates the apparent influence of progesterone and related compounds in the development of mammary hypertrophy and suggests a possible progression to mammary adenocarcinoma. In a study conducted at the Animal Medical Center in New York, 12 percent of the 50 cats treated with megestrol acetate developed mammary tumors. Mammary disease occurred in cats maintained on 5 mg doses three times weekly for periods ranging from 6 weeks to one year. The incidence rate in this study clearly demonstrates a causal relationship between megestrol acetate administration and mammary tumors, especially when compared to the annual incidence rate of mammary tumors in all female cats of only 0.025 percent.

Another very serious side effect of megestrol acetate therapy is the development of diabetes mellitus. Its occurrence does not seem to be related to dose or duration of treatment. The disease has been seen with as little as two weeks of progestogen therapy. Some cases are transient, disappearing after the cessation of therapy; others require insulin therapy; and a few have been shown to be insulin resistant. The antagonistic effect of progestogens on insulin may precipitate diabetes in animals that are borderline or prediabetic prior to progestogen therapy.

Adrenocortical suppression has been documented in several experiments involving megestrol acetate administration in the cat. The results of Chastain's study proved that even recommended doses of megestrol acetate in the cat do have significant and potentially dangerous side effects on adrenocortical function. The results of this study support the recommendation that megestrol acetate not be used indiscriminately. Furthermore, in cases where its use is necessary, one must consider concurrent stresses which may create the need for glucocorticoid supplementation.

Summary

Megestrol acetate is being used in increasing frequency in the treatment of diseases of the cat, despite the lack of federal approval for its use in this species. Side effects have been noted in the past, but it is not until recently that an effort is being made to scientifically and experimentally characterize the harmful effects of this drug. Undoubtedly, there are many disease conditions which respond favorably to progestogen therapy; but this drug should not be used as panacea. In many cases there are reasonable therapeutic substitutes with less associated risks. In those cases where an alternate treatment is unavailable, use megestrol acetate sparingly and judiciously according to the animal's particular needs and conditions. Also, closely monitor the animal for early signs of any life-threatening side effects.

References:


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Oral Ulcers (continued from page 5)

from infections, eosinophilic granulomas, tumors, and other autoimmune disorders. Some lesions may regress spontaneously and recur seasonally. Others have responded to aurothioglucose. Most lesions have not responded well to glucocorticoid therapy.4,7

Conclusion

Several different conditions can cause oral ulcers in cats, thereby, making diagnosis and treatment of oral ulcers complex. However, with a differential list in mind and a systematic diagnostic plan to follow, you can successfully treat the most perplexing conditions.

References:


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