

Feline Adult Onset Gingivostomatitis

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1) Categories of Oral Inflammatory Disease

There are several categories of similar oral inflammatory disease in cats. They are often broken down like this:

A) Juvenile Hyperplastic Gingivitis. Fleshy gums grow over the teeth around time of eruption, lack of calculus, bleed easily, usually 3.5 to 7 months of age or so. Mostly hyperplasia, not really destructive or painful.

B) Juvenile Periodontitis. Plaque, calculus, and disproportionately severe host inflammatory response leading to loss of supporting bone, tooth resorption starting under 9 months of age. “Sickly” kittens, small stature, chronic URI’s. Often Siamese, Maine Coon, DSH breeds.

C) Adult Gingivitis. Inflammation confined to gingiva. Plaque, calculus, but no bone loss. Adult healthy cats, onset 8 to 16 months of age, often Persian, Siamese, Abyssinian breeds.

D) Adult Periodontitis. Plaque, calculus, and disproportionately severe host inflammatory response leading to loss of supporting bone. Chronic disease in adult cats.

- **E) Adult Onset Gingivostomatitis.** This is the one we’re talking about. Plaque, calculus, and disproportionately severe host inflammatory response leading to loss of supporting bone, but additionally involves the buccal mucosa, pharynx, sometimes palate and tongue. Young to middle-aged cats.

F) Acute Necrotizing Ulcerative Gingivitis. As the name suggests, necrotic ulcerative erosive process, usually starts at the interdental papillae and progresses to exposure of crestal bone.

G) Miscellaneous Others. Other conditions that may mimic these types of oral inflammatory disease include pemphigus, uremic stomatitis, eosinophilic granuloma complex, lymphoma, fibrosarcoma, squamous cell carcinoma and others.

2) Clinical Signs of feline adult onset gingivostomatitis.

In addition to periodontal disease as mentioned above, there is bright red “acid burn” looking proliferative friable gingiva, buccal mucosal “kissing lesions” and pharyngeal mucosa that bleeds easily. There is marked plaque, tartar, often tooth resorption and missing teeth, ptyalism and halitosis. Affected cats have severe oral pain, leading to dysphagia, anorexia, dehydration,

weight loss and unkempt coat appearance. They may resist examination, open their mouths wide, paw at their mouths, run around and vocalize to the point that their owners think there must be a TMJ problem.

3) What Causes It?

Cause is not known, but there is clearly an inappropriate and overly severe host inflammatory reaction to plaque. It is thought that destruction of gingival and mucosal epithelium results in modified self antigens which then in turn are recognized by the immune system as foreign antigens. Then immune globulins are produced against not just the plaque bacteria, but also against the host epithelium, leading to further destruction. Histology shows predominantly lymphoplasmacytic inflammation with relatively fewer neutrophils and scar tissue formation if chronic. Oral infection by calicivirus may be the beginning of this chain of events, but it is thought that only a genetically susceptible individual will proceed to gingivostomatitis.

It is particularly interesting to note that this disease cannot be transmitted from one individual to another by any known microorganism, but it can be transmitted by a tissue homogenate containing modified epithelial antigens. Several individuals in a household may be affected. Sterilize your dental and oral surgery instruments between patients!

4) An Orderly Approach to Management

These cats are often quite challenging and frustrating for DVMs and clients both. It's important to inform the client that you may not be able to cure the condition, and to spell out an orderly investigational approach early on so they understand their medical and surgical management options, and how much client participation will be involved in whichever approach they choose.

A) How Sick Is This Cat? Does this patient need nutritional support? IV fluids? Serious pain control? In some severe cases syringe feeding and oral meds by the owner at home may be inadequate. You may need to recommend hospitalization, maybe a fentanyl patch and a feeding tube while you get things started. We currently like buprenorphine transdermal gel as an alternative to oral pain meds.

B) Rule Out Systemic Disease. CBC, chem panel, U/A, FeLV, FIV, thyroid testing are a good foundation. Expect high lymphocytes and globulins. Depending upon the case and you and your client's curiosities, additional testing could include evaluation for allergies to foods, pollens, dust and fleas, tests for giardia, toxo, calici, herpes, bartonella, maldigestion/malabsorption, bacterial cultures, fungal titers, immune panel, and serum protein electrophoresis.

C) Initial Dental Care.

i) General anesthesia, 2-3 biopsies, complete perio cleaning, Ora Vet gel if the client will use it. Probe and chart, full mouth series of dental x-rays to find and extract all teeth with bone loss,

tooth resorption, fractures, and hidden root fragments. Retained root segments are very common and may be challenging.

ii) Excellent homecare such as daily brushing with CHX gel, oral antibiotics, corticosteroids and pain control meds if severity warrants. Excellent flea control so they are not continually rubbing flea dirt antigens on their oral mucosa.

iii) Reevaluate every 3-4 weeks, oral exam and weigh-in. Some cats will do pretty well by repeating these steps. You may need to perform perio cleaning and diseased tooth search every 3, 6 or 12 months depending on your assessment at each recheck interval.

D) Medical Options. If not doing well, there are lots of oral meds to try. Unfortunately, oral meds can be difficult or impossible for clients to administer to these cats with severe mouth pain, and will likely be needed for the life of the cat or until you extract all the teeth.

i) Corticosteroids. Probably the most effective medical option. Injectables are more effective but not as safe as oral forms. Some say prednisolone is preferable in cats to prednisone. Prednisolone at 2 mg/kg BID x 14d, then tapering over 2 months or so down to the lowest effective alternate day dose (perhaps 1-2 mg/kg q 48hrs) would be a good choice. Methyl pred (good safe choice) 4 mg = prednisolone/prednisone 5 mg = triamcinolone 1.5 mg (effective but less safe) = dexamethasone 0.5 mg (ditto). Topical oral Lidex gel (fluocinonide 0.05%) is another option to try to get high local corticosteroid levels but lower systemic levels.

ii) Antibiotics. Will help a lot, but will stop working when your client stops giving them. You can use them for 7-10d at a time as needed for flare-ups in your patients that are doing pretty well on maintenance corticosteroids. Clavamox, clindamycin and metronidazole are good choices. Azithromycin 10 mg/kg q 24hr x 10d is recommended by those who believe that Bartonella is important. Azithromycin, metronidazole, tetracycline and doxycycline have beneficial antiinflammatory effects in addition to their antibiotic properties. Inj long-acting cephalosporins are convenient but not ideal in spectrum and distribution.

iii) Cyclosporine. Atopica can be used for suppression of the lymphoplasmacytic inflammation starting at 5 mg/kg q 24 hrs. Neoral is a different and older formulation that is intended to improve absorption in the GI tract, so starting doses are lower: 1-5 mg/kg divided BID. Loses efficacy if compounded with a palatable liquid, but can be mixed with an equal volume of clam juice or VAL syrup or whatever immediately before giving to the patient. Should check blood levels after 2 weeks, because absorption is unpredictable. Seems to be safe and effective. Pretty expensive.

Here's one reasonable combination for a 10 lb cat:

Neoral 20 mg BID x 2 weeks, then check blood level (whole blood HPLC 500ng/ml to start, then 250 ng/ml maint. \$122 our cost) and adjust PLUS...

Prednisolone 10 mg BID x 2-3 weeks until remission, then 10 mg q 24 hrs 2-3 weeks, then 10 mg q 48 hrs long term (eventually 5mg q 48 hrs if effective) PLUS...

Clindamycin 25 mg BID x 7d to start, and then repeated if needed now and then (perhaps once every month or two.)

Here are some other medications that don't seem to help much:

iv) Chlorambucil alkylating agent to kill the small mononuclears involved in the inappropriate inflammatory response, often started along with pred. Chlorambucil 0.2 mg/kg/day, comes in 2 mg tabs that shouldn't be divided, so give a whole tab q 48 to 72 hrs depending on body weight for 4-8 weeks, then taper down alternately with pred.

v) Azathioprine antimetabolite, modulates cell mediated immunity and T-lymphocyte dependent antibody synthesis. Toxic in cats, not currently recommended.

vi) Gold salts used for various autoimmune diseases. Injectable only, 1 mg/kg once a week 6 to 8 week, then back down to once a month. Can be toxic in cats.

vii) Lactoferrin purported to be an immunomodulator. 350 mg capsules, mix 1/2 with 1 teaspoon milk, swab it on the gums or have cat drink it BID.

viii) Coenzyme Q-10 antioxidant.

ix) Stabilized chlorine dioxide used to reduce volatile sulfur compounds associated with halitosis, gingivitis, and maybe periodontitis.

x) Lysine intended to treat Herpes infection, 250 mg BID

xi) Interferon-alpha originally thought to be an antiviral protein, has been shown to be an immunomodulator. Add 3 million units to 1 liter sterile saline, divide into aliquots and freeze, thaw and dilute when needed to produce 30 U/ml dispensing solution, 3 U/cat PO q 24 hrs on alternating weeks.

E. Surgical Options

i) Laser treatments. Plaque and diseased epithelium are causing the inflammatory disease. You can remove the plaque during your periodontal cleaning, and you can remove the diseased gingiva and epithelium with your CO2 laser. Not a cure, but an option to get a remission. Often needs to be repeated several times and followed up with medical treatment. Most veterinary dentists have not had much success with this modality, but there are a few who have.

ii) Extractions of all cheek teeth. To start with, you may choose to leave the incisors and canines but only if the surrounding periodontium is PERFECTLY healthy. Dental x-rays are a must. You must get all root fragments-- no burring out the roots or crown amputations. It is best to flap entire quadrants and perform open debridement of the alveolar bone, remove diseased gingiva, flush with lots of warm sterile saline until there is only clean healthy bleeding bone, then suture closed. Some advocate the use of CO2 laser here. I would be concerned about leaving char and non-vital bone behind that would continue to elicit inflammation. Don't forget about nutritional support and pain control here.

iii) Full Mouth Extractions. Sometimes the plaque on the incisors and canines is enough to keep the inflammatory disease going, so you have to get these teeth out in a similar fashion. Two published studies reported complete resolution in 60% and marked improvement in 20% more. Another study reported 7% didn't improve at all.

F) What if all the teeth are out but inflammation persists...

Here's what we are currently trying (with admittedly limited success):

If proliferative tissues persist consider CO2 laser.

Check a FURD PCR panel. If herpes positive try famcyclovir 62.5 mg (1/2 tab) PO BID x 14d.

If calici positive try Virbagenomega, marketed as feline interferon but not yet in the USA. Initially was intended as intralesional inj's, now mostly used PO. One 10 MU vial is diluted in 100 ml saline and then stored frozen in 10 x 10 ml vials. Use them one by one by keeping in fridge for up to 21 d. Give 1 ml PO q 24 hrs for 100 d. Alternate sides of mouth. It's about \$110 our cost for each vial, and it must be imported and kept frozen at every moment.

If all else fails or is declined, triamcinolone oral suspn 1.5 mg PO once a day to start, then taper down to once or twice a week. Not ideal, but with informed consent may be the best you can do.