

An updated approach to chronic feline gingivitis stomatitis syndrome



Figure 1.

THE condition currently, and most commonly, known as Feline Chronic Gingivitis-Stomatitis (FCGS) is a relatively common and frustrating problem to the small animal practitioner. In a study of nearly 5,000 cats by 12 practices, the prevalence of FCGS was 0.7% (Healey, 2007).

Many cases prove to be extremely frustrating with a confusing number of

different treatments in current use. The purpose of this article is to bring together a logical method of investigation and treatment for these cases based on the known evidence base where possible.

The syndrome is characterised by persistent and severe inflammation and ulceration of the oral soft tissues. Many times this includes the tissues lateral to the palatoglossal folds (Figure 1) in addition to the gingiva, pharyngeal and lingual mucosa.

Stomatitis is defined by the American Veterinary Dental College

(www.avdc.org) as: "Inflammation of the mucous lining of any of the structures in the mouth; in clinical use the term should be reserved to describe widespread oral inflammation (beyond gingivitis and periodontitis) that may also extend into sub-mucosal tissues (e.g. marked caudal mucositis extending into sub-mucosal tissues may be termed caudal stomatitis)."

Paradoxically, the condition is often present in the absence of significant accumulation of calculus on the teeth.

The syndrome can be seen at three distinct times in a cat's life. Firstly, around the time of kitten vaccinations, oral inflammation can occasionally be seen. Whether this is an immune response to vaccinal elements or the eruption of deciduous dentition and consequential increased levels in dental plaque is not known.

A second period to see an increase in oral inflammation levels is when the

tooth surface and elsewhere in the mouth.

Not all cases of FCGS present in the same manner. A wide range of severity of initial signs does commonly occur. Some cats respond well to routine periodontal therapy and improved hygiene alone while others will respond poorly to any treatment.

The implication is that some cats have a very low threshold to the trigger factors(s) whilst others have a higher threshold approaching the level for normal cats. Most intractable cases (87%) improve with elective tooth extraction and a few cases (13%) do not respond to any treatment (Hennet, 1997; Girard and Hennet, 2005; Hennet, 2010).

Clinical signs

Many, but not all, cats present with dysphagia and pain due to extensive oral inflammation and ulceration of soft tissues. In some cases it can be hard to understand how the individual eats or functions normally with such extensive oral inflammation.

Inflammatory lesions may involve some or all of the oral soft tissues. Most cases present with inflammation and ulceration of the tissues lateral to the palatoglossal folds in addition to the gingiva and mucosa overlying the cheek teeth. Other oral tissues such as the pharynx, tongue and the mandibular molar salivary glands can also be affected in severe cases.

Commonly reported signs include:

- anorexia and/or dysphagia;
- severe halitosis;
- weight loss – chronic or acute;
- lack of (or an inability to) groom;
- a reluctance to eat hard food;
- submandibular lymphadenomegaly – nodes often dramatically increased in size and painful when palpated;
- variable, sometimes minimal, accumulation of plaque and calculus;
- teeth may be missing, affected by TRs or suffering from furcation exposure and excessive mobility after recession of the periodontal tissues.

Aetiology

This is a complex multifactorial condition and there is no simple aetiological agent for the syndrome. Certain factors are thought to have an effect but the most commonly held view is that these cats suffer from an immunological over-reaction to low levels of oral antigens – dental plaque mainly. Factors involved are:

- **Breed.** Mixed breed cats make up the majority of cases seen in the author's

practice by a factor of 2:1. In the same case survey, the Maine Coon and Siamese breeds do appear to have more affected individuals than any other.

In a survey of nearly 5,000 cats in 12 practices over a 12 week period, 34 cases of chronic FCGS were identified and no breed bias was identified (Healey, 2007).

• **Environmental factors.** Colony cats or those in multi-cat households appear to be more commonly affected early in life. Increased stress levels plus the close proximity of other cats allowing transmission of viruses and other micro-organisms are held to be significant factors.

• **Plaque bacteria.** The oral bacteria present in dental plaque matrix drives the inflammatory response in normal individuals. Cats that appear to be plaque intolerant show a variable threshold to the bacterial load of the mouth displaying an abnormal and non-specific level of inflammation.

Specific bacteria, as seen in periodontal disease, have been reported in these cats and *Pasteurella* and *Prevotella* species are more highly represented than others. Around half the cats seen by the author harvested pure cultures of *Pasteurella multocida* (Dolieslager, 2011).

Viruses

• **Feline Calici Virus.** Many papers report a level above 70% of chronically affected individuals with signs for over six months showing positive testing to virus isolation following oropharyngeal swabbing for Feline Calici Virus (Knowles, 1989; Thomson, 1984; Harbour, 1991). The significance of this within the syndrome is not known.

It is possible that the virus damages cell membranes allowing easier antigenic penetration by other agents. However, other co-factors are required before this virus can cause disease as FCV carriage in the cat population is around 20-30% (Knowles, 1989; Zicola, 2009).

One research study (Hennet and Boucraut-Baralon, 2005) considered that chronic palatoglossitis lesions, as opposed to buccostomatitis lesions, to be more specifically associated with calicivirus carriage.

• **Feline Immunodeficiency Virus (FIV)** infection may have a role in producing oral lesions by predisposing the cat to secondary infections. Both FIV and FeLV may contribute to an aberrant immune response to oral antigens but

NORMAN JOHNSTON outlines a logical method of investigation and treatment of this relatively common and frequently frustrating problem - based on the known evidence base



permanent teeth erupt. Lastly, and most commonly, FCGS is seen later in life with a mean age of seven years.

This syndrome must be considered as part of a full oral cavity examination. The presence of other problems concurrently, such as tooth resorption lesions (TRs), frequently adds to and confuses the picture.

It has been known for some time that carriage of Feline Calici Virus (FCV) is a co-factor in the induction or progression of the complex. The relationship between calici infection and FCGS appears strong with 70-90% of chronic stomatitis cats testing positive compared with 20% of general population cats (Knowles, 1989; Harbour, 1991).

It has also been reported that the relationship between FCV and FIV appears strong but the association between the two has never been established for FCGS cats (Knowles, 1989 and 1991).

Many cats also present with a hypergammaglobulinaemia (Harley, 1999; Harley, 2003). This implies B lymphocyte proliferation and therefore no humoral immune response depression. It is probable that affected cats are intolerant to even small quantities of bacterial plaque on the

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results with virus isolation techniques to be considered truly negative.

• **Initial intralesional use.** The consensus statement from 2010 and a subsequent study (Hennet, 2011) indicated that intralesional treatment is not necessary to initiate therapy.

• **Subcutaneous injections.** This method of administration has been described previously (Southerden, 2006) but according to subsequent research it appears to be substantially less effective, and markedly more expensive, than transmucosal administration for treatment of FCGS.

Corticosteroids

These drugs are used, by some practitioners, principally to control inflammation in refractive cases which have had elective cheek teeth extraction and are not sufficiently controlled by feline recombinant interferon.

If their use is justified on welfare grounds (often described as rescue therapy), the overriding principle must always be to use the minimum effective dose rate.

This means using a short-acting molecule (prednisolone) at the lowest effective dose rate such as 5mg twice weekly or 2mg every other day tapering downwards. They can be used in conjunction with feline recombinant interferon omega.

NSAIDs

If used, the first choice option appears to be meloxicam. Any NSAID needs to be prescribed with due regard to the appropriate guidelines for use of long-term NSAIDs in cats (Sparkes, 2010). Some new molecules such as robenacoxib may show promise.

Cyclosporine

There is currently insufficient data from published papers to recommend the use of cyclosporine in the management of FCGS syndrome.

Some data have been published on this molecule, as part of a dermatology study, suggesting four out of eight cats treated responded and could be maintained on every second day dosing. However, other studies have been equivocal about the benefits and a placebo-controlled trial in a small number of cats did not show a significant difference from placebo only.

Some suggestions for use have been provided but monitoring of blood levels to avoid toxicity is deemed essential due to erratic absorption differences. In general, the currently available data are not sufficient to support a recommendation to use this drug, according to the consensus study group.

Azathioprine/chlorambucil/low dose doxycycline/gold salts

There are insufficient data to

recommend the use of any of these agents in the management of FCGS syndrome. The potential side effects can be significant and excessive use of immunosuppressive doses can be a problem long term.

CO₂ laser surgery

There are insufficient data to recommend the use of CO₂ laser use routinely in the management of FCGS syndrome. The consensus group felt it may have a possible role with adjunctive pain control.

One single cat case study concluded that the use of a CO₂ laser assisted recovery of soft tissues after extraction therapy but would not have been as useful as a monotherapy (Lewis, 2007).

Additive-free foods and additional nutritional support

It is necessary to ensure good quality nutritional support to encourage an effective immunological response and post-extraction healing process.

Various diets and supplements have been suggested, including vitamin preparations and omega-3 EFAs, but there is no study which has data to prove a recommendation for any specific product. There is anecdotal evidence that use of diets or supplements high in omega 3 EFAs affects platelet function and can result in excessive haemorrhage during extraction surgery.

The beneficial effect of a recovery food post-surgery has been demonstrated in cats with FCGS syndrome (Thyse, 2003).

Additive-free and hypoallergenic foods have also been suggested but the results are anecdotal at best with no known study proving efficacy.

Summary

Feline Chronic Gingivitis Stomatitis Syndrome is a poorly defined syndrome of unknown aetiology characterised by focal or diffuse chronic inflammatory response involving the gingiva, oral mucosa, and often the pharynx, tongue and other oral soft tissues.

Commonly described clinical findings include elevated serum globulins and a sub-mucosal infiltrate of plasma cells, lymphocytes, neutrophils, and macrophages. Potentially various viral agents and bacterial species are involved.

It is considered that atypical hyperimmune responses are the basis of the syndrome. Multiple mechanisms appear to be acting concurrently.

Successful management of this complex requires a logical diagnostic approach. The need for first-line information gathered before treatment alters the host response cannot be over-stated.

Definition of oral and oropharyngeal Inflammation

Source: American Veterinary Dental College Nomenclature Committee (www.avdc.org/nomenclature.html)

Oral and oropharyngeal inflammation is classified by location as:

Gingivitis: inflammation of gingiva

Periodontitis: inflammation of non-gingival periodontal tissues (i.e. the periodontal ligament and alveolar bone)

Alveolar mucositis: inflammation of alveolar mucosa (i.e. mucosa overlying the alveolar process and extending from the mucogingival junction without obvious demarcation to the vestibular sulcus and to the floor of the mouth)

Sublingual mucositis: inflammation of mucosa on the floor of the mouth

Labial/buccal mucositis: inflammation of lip/cheek mucosa

Caudal mucositis: inflammation of mucosa of the caudal oral cavity, bordered medially by the palatoglossal folds and fauces, dorsally by the hard and soft palate, and rostrally by alveolar and buccal mucosa

Contact mucositis and contact mucosal ulceration: lesions in susceptible individuals that are secondary to mucosal contact with a tooth surface bearing the responsible irritant, allergen, or antigen. They have also been called "contact ulcers" and "kissing ulcers"

Palatitis: inflammation of mucosa covering the hard and/or soft palate

Glossitis: inflammation of mucosa of the dorsal and/or ventral tongue surface

Cheilitis: inflammation of the lip (including the mucocutaneous junction area and skin of the lip)

Osteomyelitis: inflammation of the bone and bone marrow

Stomatitis: inflammation of the mucous lining of any of the structures in the mouth; in clinical use the term should be reserved to describe wide-spread oral inflammation (beyond gingivitis and periodontitis) that may also extend into submucosal tissues (e.g. marked caudal mucositis extending into submucosal tissues may be termed caudal stomatitis)

Tonsillitis: inflammation of the palatine tonsil

Pharyngitis: inflammation of the pharynx

Once this information is available, a treatment plan and prognosis can be considered.

The role of bacterial plaque is crucial, whatever the state of the host immune response. Diligent professional scaling, polishing and subgingival debridement and attention to existing dental disease underpins any treatment in tandem with aggressive home-care by the owner.

Cases failing to respond to simple plaque control should be considered for elective cheek teeth extraction and adjunctive treatments at an early date. Those cases still non-responsive but FCV positive may be helped by interferon therapy.

It is important that the owner is involved at an early stage with discussions as to aetiology, treatment plans and help with home-care. A highly-motivated owner is a strong ally in the provision of successful treatment.

Selected references and further reading

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