Feline Stomatitis Gingivitis Complex – New approaches to an old disease I

More than just a steroid deficiency!

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Feline Stomatitis Gingivitis Complex, Feline Chronic Gingivo-stomatitis (FCGS), lymphocytic-plasmacytic gingivitis stomatitis; what every name it is given there is one thing that remains unchanged – this is a significant problem in veterinary practice. It can have a major impact on the health and welfare of affected cats and represents a major diagnostic and therapeutic challenge to the practicing veterinary surgeon. One of the biggest challenges is to define and accurately identify the problem in the first place.

A recent epidemiological study (1) used the following case definition:

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<th>1. Inflammation of the mucosa of the oral cavity, to include at least one of:</th>
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<td>• Periodontal area</td>
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<td>• Gingival mucosa</td>
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<td>• Buccal (cheek) mucosa</td>
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<td>• Fauces/palatoglossal folds</td>
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<td>• The soft palate and pharynx may sometimes be affected</td>
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<td>2. Where present, gingivitis usually extends beyond the muco-gingival margin</td>
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<td>3. The severity of the inflammation is worse than would be expected in the context of visible dental disease</td>
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<td>4. Affected areas are frequently oedematous, proliferative and/or ulcerated</td>
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<td>5. The inflamed areas are usually bright red in colour, and often bleed spontaneously or on very mild trauma (e.g. swabbing)</td>
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Points 1 and 3 were considered to be minimum inclusion criteria for cases

This study demonstrated a prevalence of 0.7%, although most veterinary surgeons would believe it to be higher, probably a consequence of the frustrations associated with its management.

AETIOPATHOGENESIS

FCGS is characterised by inflammation, and frequently proliferation and/or ulceration, of the oral mucosa particularly the caudal gingivae, caudal buccal mucosa and the glosso-palatine folds. A large number of infectious agents and non-infectious factors such as dental disease have been implicated in the aetiopathogenesis of FCGS. How these apparently unrelated conditions inter-relate and combine to produce the condition we recognise as FCGS is not obvious and the precise aetiology of this syndrome remains unproven.

Recent research has proposed (2,3) that the pivotal event in this condition is an aberrant immune response to oral antigens. In normal cats this is recognised as involving predominantly type 1 T-helper cells (Th1) associated with cell mediated immunity. In
cats suffering from FCGS the profile of inflammatory cytokines was established to be consistent with a mixed Th-1 and Th-2 (antibody mediated immunity) response\(^2\). It was also demonstrated that cat affected cats have a polyclonal hyperglobulinaemia and increased salivary IgG and IgM but reduced salivary IgA levels\(^3\). This deficiency in salivary IgA may play a role compromising the effectiveness of local oral defence mechanisms.

These individual immunological factors may explain why some cats will develop FCGS and others do not despite similar oral factors being present (Figure 1).

**PLAQUE AND PRIMARY DENTAL DISEASE**

Plaque is a soft, sticky, tooth-coloured paste-like mixture of salivary glycoproteins, sloughed epithelial cells, white blood cells and bacteria. It is therefore one of the greatest sources of oral antigens. Within hours of eating dental surfaces become coated in a layer of glycoproteins called the acquired pellicle which is then colonised by bacteria. As this immature plaque matures it becomes more organised and firmly adhered to the teeth. Mature plaque consists of 75% structural matrix and 25% bacteria\(^4\). Plaque is the cause of gingivitis and periodontal disease.

Primary dental disease is one of the most common conditions affecting cats. It has been reported that the prevalence of periodontal disease in cats is as high as 70% by the age of three years.

In addition teeth affected by periodontal disease and/or feline odontoclastic resorptive lesions (FORLs a.k.a. neck lesions) will be associated with higher levels of plaque than healthy teeth especially sub-gingivally where there is greater opportunity for
host-antigen interaction. It can often be difficult to diagnose the presence and extent of these problems without the aid of intra-oral radiography.

**FELINE CALICIVIRUS**

The exact role of Feline Calicivirus (FCV) in the development of FCGS has not been established and is subject to debate. There would appear to be compelling evidence for it as an aetiological agent as it has been isolated from 85 to 100% of cases of FCGS and which is further supported by recent case reports where clinical resolution was associated with cessation of FCV shedding. However, FCV has also been isolated from between 20% and 25% of healthy cats and 21% of cats with classical periodontal disease. Experimental attempts to induce FCGS using FCV isolated from clinical cases have also been unsuccessful.

It has been proposed that it is not one specific antigen that is responsible for the development of FCGS but the cumulative effect of all the different antigens within the oral cavity. Each cat may have its own individual threshold of antigenic stimulation that needs to be exceeded in order to precipitate the condition and FCV may be one of the many significant sources of antigen contributing to the total.

**DIET**

In one case report it was suggested that there might have been an association between a change in diet and recovery from FCGS. Recent research in North America has also proposed that in the later stages of the disease the disruption to the integrity of the oral mucosa allows food antigens (primarily proteins) to be presented via an abnormal route. This in turn causes a tissue reaction which perpetuates the pathological processes already occurring.

**FIV/FeLV**

There has been considerable interest in the role of retroviruses in the development of FCGS. Both FIV and FeLV have been isolated from FCGS cases (FIV 10-81% and FeLV 0-17%). Of the two FIV is considered to be the more significant primarily through predisposing the host to secondary infections although it may also have an immunomodulatory effect on the host response to oral antigens.

**DIAGNOSIS**

One of the difficulties with FCGS is that it irrespective of the name the condition is given they are all descriptive terms rather than a specific diagnosis. In general practice FCGS is frequently diagnosed on the basis of the following criteria:

- A history of longstanding oral inflammation
- Failure to respond to ‘scale and polish’
- (Inflammation involving tissues other than the gingiva, e.g. stomatitis, “faucitis”)
- Biopsy result demonstrating lymphocytic and plasmacytic infiltrates
- Positive swab for feline calicivirus virus on isolation
Many of the major differential diagnoses of FCGS (Figure 2) could have a similar clinical presentation, histology and viral status. Consequently a true diagnosis of FCGS is made by the elimination of the differentials listed.

![Figure 2: Differential Diagnosis of FCGS](image)

- Periodontal Disease
- Feline Odontoclastic Resorptive Lesions
- Eosinophilic Granuloma Complex
- Neoplasia
- Trauma
- Acute Calicivirus Infection
- Retained Roots

The FeLV/FIV status of all cats with FCGS should be established as these viruses will adversely affect the prognosis and may require specific treatment in addition to the principles of management targeting the oral cavity. They also represent a significant risk to other patients if the equipment used for dental procedures is inadequately sterilised. FCV isolation should also be included in the virology profile.

Routine biochemistry and haematology is useful to identify any underlying systemic disease especially as in one study \(^{(3)}\) 10% of cases had chronic renal failure which could significantly influence both the treatment plan and the prognosis.

Care must be taken to avoid reaching a diagnosis and formulating a treatment plan on the basis of a visual examination alone. Radiography should be regarded as an essential part of the diagnostic work-up in a case of FCGS; without it it is virtually impossible to fully assess the extent and severity of dental disease. It may be utilised during the first examination under anaesthetic or to investigate cases which have failed to respond as expected to an initial routine prophylaxis procedure (scale and polish). Radiography will also help identify any other problems such as broken root fragments, rough edges of alveolar bone or alveolar bone fragments.

There are sound reasons for including a biopsy at an early stage in the investigation of FCGS as the relationship between chronic inflammation, irritation or mechanical trauma and neoplasia has been well established in veterinary medicine. Any asymmetrical lesions should be regarded as suspicious always be biopsied. When taking a biopsy it is important to take a fairly deep section to ensure a representative sample due to superficial necrosis and inflammation.

REFERENCES


