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# An update on epidemiological features, etiopathogenesis and therapeutic approaches of feline chronic gingivostomatitis

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## ABSTRACT

Feline chronic gingivostomatitis (FCGS) is a severe, immune-mediated, inflammatory disease affecting the oral mucosal of cats. It is characterized by ulcerative and/or proliferative lesions, most commonly located lateral to the palatoglossal folds. Clinically, FCGS can lead to severe malnutrition and dehydration in critical cases. The pathogenesis of FCGS is poorly understood but it is considered a multifactorial disease, likely involving infectious agents and other parameters. FCGS seems to be a manifestation of an aberrant immune response to chronic antigenic stimulation. Disturbance and im balance of the oral microbiota also may play a role in the development of FCGS. Because of its unknown pathogenesis and long disease course, it is difficult to treat and has a high recurrence rate. The current standard of care involves dental extractions of at least all premolar and molar teeth, often in combination medical therapy. Standalone medical management has shown limited long-term efficacy. Emerging regenerative therapies, such as mesenchymal stem cell treatment, offer promising alternatives for management of FCGS.

## Keywords

*FCGS, clinical features, epidemyological features, treatment, oral inflammation.*

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## Abbreviations

*FCV : Feline Calicivirus*
*FeLV : Feline Leukaemia Virus*
*FHV-1 : Feline Herpesvirus Type 1*
*FIV : Feline Immunodeficiency Virus*
*CPV : Canine Parvovirus Virus*
*FME full-mouth extractions*
*IFNs : Interferons*
*MSCs : Mesenchymal stem cells*



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## Introduction

FCGS is a chronic inflammatory disease marked by ulcerative and/or proliferative lesions effecting the gingiva and oral mucosa, particularly the palatoglossal folds [1, 2]. It may be referred to by other names, such as plasma cell stomatitis-pharyngitis, chronic faucitis, lymphocytic plasmacytic gingivitis-stomatitis and others [3]. FCGS is a painful and debilitating feline oral disease characterized by chronic severe bilateral inflammation of the gingiva, alveolar mucosa, labiobuccal mucosa, and caudal oral mucosa [4, 5]. Ulcerative or ulceroproliferative lesions are often observed in FCGS cases. In addition, FCGS has been shown to be associated with more widely distributed and severe periodontitis, as well as higher prevalence of external inflammatory root resorption and retained roots compared to other oral diseases [6]. This article reviews the current knowledge on the etiopathogenesis and epidemio-clinical features of FCGS and describes the leading treatment modalities. Cats affected by FCGS are often presented with dysorexia/anorexia, oral pain, weight loss, ptyalism, halitosis, and lack of grooming [7,8]. Although FCGS is a familiar condition encountered in veterinary practice [13,14], there is much confusion regarding the cause and subsequent treatment of the disease [14,15]. This article reviews the current knowledge on the etiopathogenesis and epidemio-clinical features of FCGS and descibes the leading treatment modalities.

## *Clinical features*

### Etiology

The etiology of FCGS is currently unknown [16], probably multiple etiologies may exist that, either alone or combined, can contribute to the presence of the inflammation [9]. Possible causative factors include viral infections particularly feline upper respiratory viruses such as FCV, FHV-1, bacterial infection like *Bartonella henselae* and altered immune status associated with FIV, FeLV [10, 11,12], as well as non infectious factors such as dental disease, environmental stress, and hypersensitivity [18, 19]. It has been proposed that the disease is an immune reaction to plaque and the tooth structure itself or the periodontal tissues [3]. According to Thomas et al. [16] FCGS is initiated from gingival inflammation and is perpetuated to the mucosa of oral cavity (**Table 1**).

### Abbreviations-Cont'd

*OR : Odds ratio*
*PME : partial-mouth extractions*
*RFeIFN - ω : Recombinant feline interferon omega*
*LPS : lipopolysaccharide*
*MHC- II : major histocompatibility complex class II*

### Clinical signs

FCGS is a severe inflammatory syndrome involving the immune system that affects [17]. The disease varies in severity and may include faucitis, pharyngitis, or palatitis [3]. Clinical signs include severe oral pain,ptyalism, mandibular lymphadenopathy [10], poor grooming habits and unkempt appearance [7, 8], dysphagia, pawing at the mouth, anorexia, crying out in pain when eating or yawning[24, 25] halitosis, loss of appetite, depression, weight loss [2] and (in severe cases) even dehydration [26]. Affected cats can become severely debilitated and because of the unclear pathogenesis and relapsing course of the disease, FCGS remains one of the most challenging oral conditions to manage in feline practice [2]. Therefore, euthanasia may be considered as last resort, when quality of life is significantly declined [27].

The hallmark lesions include caudal stomatitis and alveolar mucositis, both of which are commonly assessed using a standardized five- grade scoring system [27].

-Grade 0 : No visible lesion.

-Grade 1 : Mild, non-ulcerative, non-proliferative inflammation. Lesions do not bleed spontaneously or under slight pressure.

-Grade 2 : Moderate, non-ulcerative inflammation with mild proliferative changes. Lesions do not have spontaneous bleeding even with slight pressure.

-Grade 3 : Moderate, ulcerative or ulceroproliferative inflammation, without spontaneous bleeding, but with bleeding when slight pressure is applied.

-Grade 4 : Severe, ulcerative or ulceroproliferative inflammation with spontaneous bleeding.

In addition to clinical grading, histological examination of the the oral mucosa tissues affected by FCGS, shows a diffuse and dense cell infiltration, containing lymphocytes and plasma cells which are predominantly observed. In contrast, relatively few neutrophils, mast cells have been observed, thus showing the characteristics of chronic inflammation [28, 29]. These histological features are the basis for the disease's alternative nomenclature, such as plasma cell gingivitis (-stomatitis)-pharyngitis or lymphoplasmacytic gingivitis [30]. Figure 1. shows Ulcero-proliferative lesions of FCGS in tissues lateral to palatoglossal folds plus maxillary gingivitis and alveolar mucositis both sides.

### Diagnostic

Diagnosis of FCGS is primarily clinical and relies on the identification of characteristics oral lesion [3, 27] through thorough visual inspection of the oral cavity [10, 31]. FCGS is mostly characterised by bilateral inflammation of the mucosa in the caudal oral cavity, a hallmark feature that helps distinguish FCGS

**Table 1.**
Microorganismes (bacteria and virus) associated with FCGS recorded in some studies.

| References | Frequency of cats with FCGS | Bacteria or virus explored | Microorganisms associated with FCGS (P value when reported) |
|---|---|---|---|
| Thompson et al. [31] | 50% (10/20) | FCV (by culture) ; FeLV (by immunochromatography) | None reported |
| Lommer and Verstraete [12] | 51% (25/49) | FCV (by culture) ; FHV-1 (by culture) | FCV ; FHV-1 |
| Dowers et al. [11] | 53.4% (70/131) | Bartonella (by ELISA, culture and PCR); FCV (by PCR); FHV-1 (by PCR) | FCV ($p = 0.0006$) |
| Sykes et al. [32] | 3% (9/298) | Bartonella (by culture and immunofluorescence) | Bartonella isolation ($p = 0.001$) |
| Dolieslager et al. [33] | 62.5% (5/8) | Bacterial flora (by culture and PCR) | Pasteurella multocida subspecies multocida |
| Kornya et al. [34] | 3.9% (203/5179) | FeLV (by ELISA) ; FIV (by ELISA) | FIV |
| Fernández et al. [35] | 43% (154/358) | FHV 1 (by PCR); FCV (by PCR); Chlamydophila felis (by PCR); Mycoplasma felis (by PCR) | FCV ($p$ <0.001) ; C felis ($p$ = 0.025); M felis ($p = 0.003$) |
| Rolim et al. [36] | 60.5% (26/43) | FCV (by immunohistochemistry); FeLV (by PCR); FIV (by PCR) | None |
| Thomas et al. [20] | 27.7% (25/90) | FCV (by culture) | FCV ($p = 0.010$) |
| Whyte et al. [37] | 11.8% (4/34) | FCV (by immunofluorescence); microbacteriome (by phenotype and conventional biochemical methods) | None |
| Nakanishi et al. [38] | 30.7% (32/104) | FHV-1 (by PCR); Chlamydia felis (by PCR); M felis (by PCR); Bordetella bronchiseptica (by PCR) | FCV ($p = 0.018$) |
| Fried et al. [39] | 54.7% (23/42) | FCV (by genomic sequencing) | FCV ($p = 6.0\times10–42$) |
| Krumbeck et al. [40]. | 50% (14/28) | Bacteriome and mycobiome (by DNA sequencing) | None |

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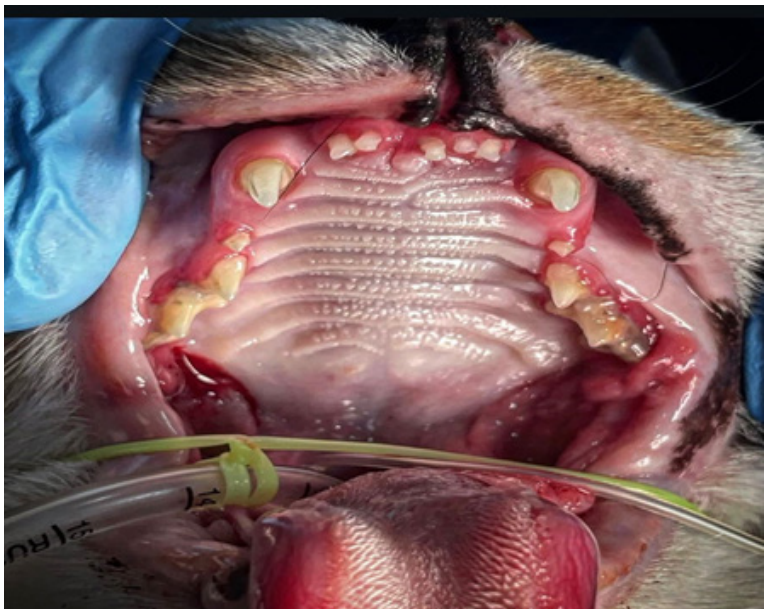

**Figure 1.**
Ulcero-proliferative lesions of FCGS in tissues lateral to palatoglossal folds plus maxillary gingivitis and alveolar mucositis both sides.

from other oral diseases [4]. The affected gingiva and oral mucosa in FCGS exibit variable degrees of inflammation, proliferation, and ulceration [31]. The mucosal surfaces is typically appear bright red, with friable tissues that bleed easily [24]. Additional diagnostic tests are essential to fully evaluate the patient health, these include [31] dental radiographs, complete blood count and serum biochemical profile and evaluation of FeLV/FIV status. In cases inflammation is asymmetrical, appears atypical, or radiographic findings raise suspicion for neoplasia, a biopsy should be submitted for histopathological evaluation [32]. As previously mentioned, FCGS lesions may occur in multiple areas, from the gums in the oral cavity to the pharynx [27]. The inflammatory process often extends beyond the mucogingival junction, encompassing the alveolar mucosa

and other soft tissues including the lingual mucosa, glossopalatine folds, caudal oral mucosa and, in some cases, the fauces [33]. According to Healey et al. [13], the most frequently affected sites include gingival mucosa (ie, visible gingiva extending from the teeth to the mucogingival junction), the periodontal area (ie, the part of the visible marginal gingiva immediately adjacent to the teeth), and the glossopalatine folds, commonly referred to as the fauces..

However, the clinician must be cautious in diagnosing FCGS. The presence of severe gingivitis in a patient, even in conjunction with the detection of FCV via PCR, does not automatically provide a diagnosis of FCGS [33]. The clinical sign that differentiates caudal stomatitis from periodontal disease is the presence of caudal inflammation (distal to the teeth) referred to as caudal stomatitis. This presentation was previously called faucitis, but is now known as caudal mucositis contrast, in cases of typical periodontal disease, inflammation is associated with the gingiva tissues adjacent to the teeth, and rarely extends into the caudal oral mucosa [1]. Also, many cases of juvenile gingivitis may be mistaken for FCGS and if inflammation is restricted to gingival tissues, a diagnosis of FCGS should not be made [33].

### *Epidemiological features*

FCGS is considered multifactorial [13]. Some studies suggest that nutritional factors, physiological or environmental stresses, dental disease and genetic predisposition may be the cause of FGS [10]. Viral infections, including FeLV, FCV, FIV, FLV and FHV-1, might be implicated in the development of FCGS [22]. However, these infectious agents have been isolated not only from affected cats, but also from control animals [34], making it difficult to establish a definitive causal relationship in each individual case of FCGS [35]. In addition to viral pathogens, certain anaerobic bacterial species have also been proposed as potential contributors [36]. Immunological studies have found alterations in cytokine expression patterns and immunoglobulin profiles in FCGS-affected cases compared to controls [37]. Furthermore, it has been suggested that immunosuppression due to an unrelated health conditions may play a role [38].

### *Prevalence*

The reported FCGS varies considerably across studies, ranging from 0.7% to as high as 45.76% [17, 27,41]. In 2004, Verhaert and Van Wetter [39] reported a prevalence rate of 12%. Later, in 2007, Healey et al. [13] targeted domestic cats that visited a primary hospital, in his study, and reported a significantly lower prevalence of 0.7%. In 2009, Girard et al [14] studied colony cats that had no contact with the external environment and recorded a prevalence of 5.5%. More recently, in 2024, Dai et al. [2] reported a prevalence of 1.96% in cats admitted to three animal hospitals in Xi'an, China. On the other hand, high prevalences have been found by Da Silva et al. [40] who recorded a prevalence of 34.88% of stomatitis and Öztürk Gürgen et al. [41] who recorded 45.76%.

### *Potential viral causes*

Several viruses with global distribution have been associated with the pathogenesis of FCGS, including FCV [13, 42], FHV [22], FeLV and FIV [43]. Many of the epidemiological and clinical features of these pathogens have been documented (17, 18, 44). Among these, FCV seems to has the most consistent evidence of being associated with FCGS [11, 22, 29, 45]. Nakanishi et al. [46], by using PCR assay, reported that 63% of cats diagnosed with FCGS tested positive for FCV, compared to 36% in the control group. Their findings also suggested that the microflora of the oral cavity of cats with FCGS might be disrupted. In contrast, no statistically significant difference was found in the prevalence of FHV-1 between affected and control groups. Supporting this, Martijn [30], detected FCV in 95.5% of cats with FCGS, while only 4.1% of control cats tested positive. Also FHV was detected in 2.3% in FCGS cases and and was absent in controls . Similarly, Thomas et al. [17] found the incidence of FCV to be significantly higher in cats with FCGS (60%) compared with control cats (24%). However, not all studies have been able to consistently prove that chronic infection by FCV is directly implicated in the pathogenesis of FCGS [28, 43, 47]. Also, the association of FIV and FeLV with FCGS is still not completely elucidated [47, 48, 49].

Regardless of the precise role of individual pathogens, well-known risk factors for these viruses include free-roaming behavior and residence in multi-cat environments such as shelters, shared households, and breeding catteries [17]. Notably, some studies showed that the prevalence of FCV, FeLV and FHV is higher in multi-cat environments [44, 50]. Moreover Radford et al. [51] noted that the prevalence of FCV infection is proportional to the number of cohabiting cats. There is consistent evidence that FCV is associated with the disease, and an etiologic role is suspected [11, 42]. Free-roaming behavior is a known co-factor for FeLV, FIV, FHV and FCV infection [50, 52]. Thus, it could be suggested that infection alone is not sufficient to initiate FCGS and that additional conditions related to environments may also play a critical role. Multi-cat conditions also facilitate permanent exposure to viral particles shed by chronic carriers, favor high rates of

viral evolution and facilitate cyclic reinfection of susceptible animals [53].

Although there is strong evidence supporting the involvement of FCV in FCGS, the inability to recreate the disease in naïve population and the effectiveness of treatments such as full-mouth dental extractions in many cases, have cast doubts on a singular role for FCV and raised suggestions that this disease may be influenced by the nature of the host's immune response and derangements (dysbiosis) of the oral microbiological flora [54].

### *Bacterial burden in FCGS*

In addition to viral and host immune factors, bacterial organisms are thought to play a role in the pathogenesis of FCGS [17]. Some studies reported that bacteria, especially gram negative anaerobe bacteria, play a certain role in the pathogenesis of FCGS. Especially gram negative anaerobe bacteria [36]. In relevant studies on the oral bacteria associated with FCGS, different experimental results have been reported. One study reported that the oral microbiota diversity of cats with FCGSs was greater compared to healthy controls [55]. Notably, some studies have also reported that the detection rate of anaerobic bacteria in the oral microbiota of cats with FCGSs was significantly greater than that of healthy controls [2,46]. According to Rodrigues et al. [55] higher abundance of gram-negative and anaerobic bacteria was found in FCGS and periodontitis, suggesting a possible role of bacterial biofilms in the pathophysiology of both diseases. Among the bacteria most commonly identified in FCGS-affected cats are Porphyromonas app., Treponemas app., and Fusobacterium app., [2]. The cell membrane of gram negative anaerobe bacteria contains LPS and this component plays an important role in the initiation of the infecetion [56]. Moreover, these bacteria are also an important aetiopathologic factor in oral infections in humans [36]. The success of full mouth extractions can lighten and even remove the inflammation [57]. This suggests that dental plaque and calculus with all their residential bacteria play an important role in maintaining the inflammatory oral condition [30].

### *External environmemt and lifestyle*

Factors relating to multicat environments as well as the stress of living in such environments may be necessary in addition to an infectious cause to trigger the development of FCGS [58]. A recent studies investigated the association of multicat environments and outdoor access with the prevalence of FCGS and showed that the prevalence of FCGS was higher in multicat than single-cat households, and that each additional cat in the household increased the odds of FCGS by more than 70% [58].

**Age**

FCGS can occurs in cats of all ages, after tooth replacement [14]. Although based on multiple studies, the condition is most frequently diagnosed in adult cats [13, 41] and the mean age for cats with FCGS was found to be between 5 and 8 years [13, 30, 41, 61]. Nakanishi et al. [46] showed that cats may be affected at an early age.

**Sex**

Many studies showed that there was no significat correlation between FCGS and sex [2,13, 27, 41]. However, Martijn [30] reported a significant positive associated between male sex and FCGS, noting that male cats were four time more infected than female (odds ratio : OR=4.1). Similarly, some other studies found high rates of FCGS in neutered males [13, 39]. A higher prevalence of FCGS was also identified in males than in females in a study done by Kim et al. [27], though the observed results were not statistically significant. Perhaps male cats, particularly those with outdoor access, are more exposed to infectious diseases, which might play a role in developing FCGS, because in general male cats have a greater territory outside and are more aggressive towards other males [30].

**Breed**

According to the breed, studies showed differents results Healey et al. [13] and Dai et al. [2] found that there is no significants correlation between breed and FCGS. However in other studies, some breeds like ; Siamese, Abyssinian, Persian, Himalayan and Burmese breeds, which have all been cited in the literature as potentially predisposed [59, 60]. Martijn [30] revealed that 47.7% of the FCGS cats were purebreds, while 4.5% were crossbreds and that the purebreds significantly associated with FCGS (OR=25.2). This study also found that 61.9% of purebreds were Main-Coons. Conversely, others studies noted that mixed breeds were more predisposed to FCGS. For example, in a study of Hennet [61], in a case series involving 30 cases of FCGS-effected cats, the majority were mixed breed, with only three Siamese, three Persian and one Foreign breed represented. Similarly, Healey et al. [13] found that 91% of cats with FCGS were mixed breed ; with only 2 purebreds (1 Persian and 1 Siamese), and 1 unclassified individual. In general, some authors noted that Purebreds cat may be predisposed in developing oral diseases, but in the case of FCGS specifically, a percentage of purebreds mostly ranges from 10% to 25% [13, 61].

### *Leading treatment modalities*

In general, there are 2 approaches to the treatment of FCGS : surgical and medical, often combined. However, on its own, medical treatment typically does not have favorable long-term outcomes [17] and has been shown to only provide temporary improvement [5, 8]. Surgical treatment, particularly FME or PME involving the premolar and molar teeth, has demonstrated the best long-term outcome [23, 47]. Clinical studies report that approximately 80% of the cats submitted to dental extractions, FME or PME, obtained significant improvement, with some achieving complete remission of the clinical signs, with or without the need for combined medical treatment [8, 61] (Fig.2).

### *Surgical treatment*

Extraction therapy is the preferred treatment for FCGS and should be performed as soon as possible [1]. Bellei et al. [7] showed that the extraction of teeth has shown better results compared to drug therapy, with clinical cure achieved in up to 57% of treated cases. According Hennet [61] approximately 60% of cats had significant improvement following dental extractions, while 20% had partial improvement, and the remaining 20% had little or no improvement. Based on the findings of Druet and Hennet [23] PME (along with other teeth that independently have indications for extraction, such as severe periodontitis, retained roots, or resorptive lesions) as the first stage of treat-

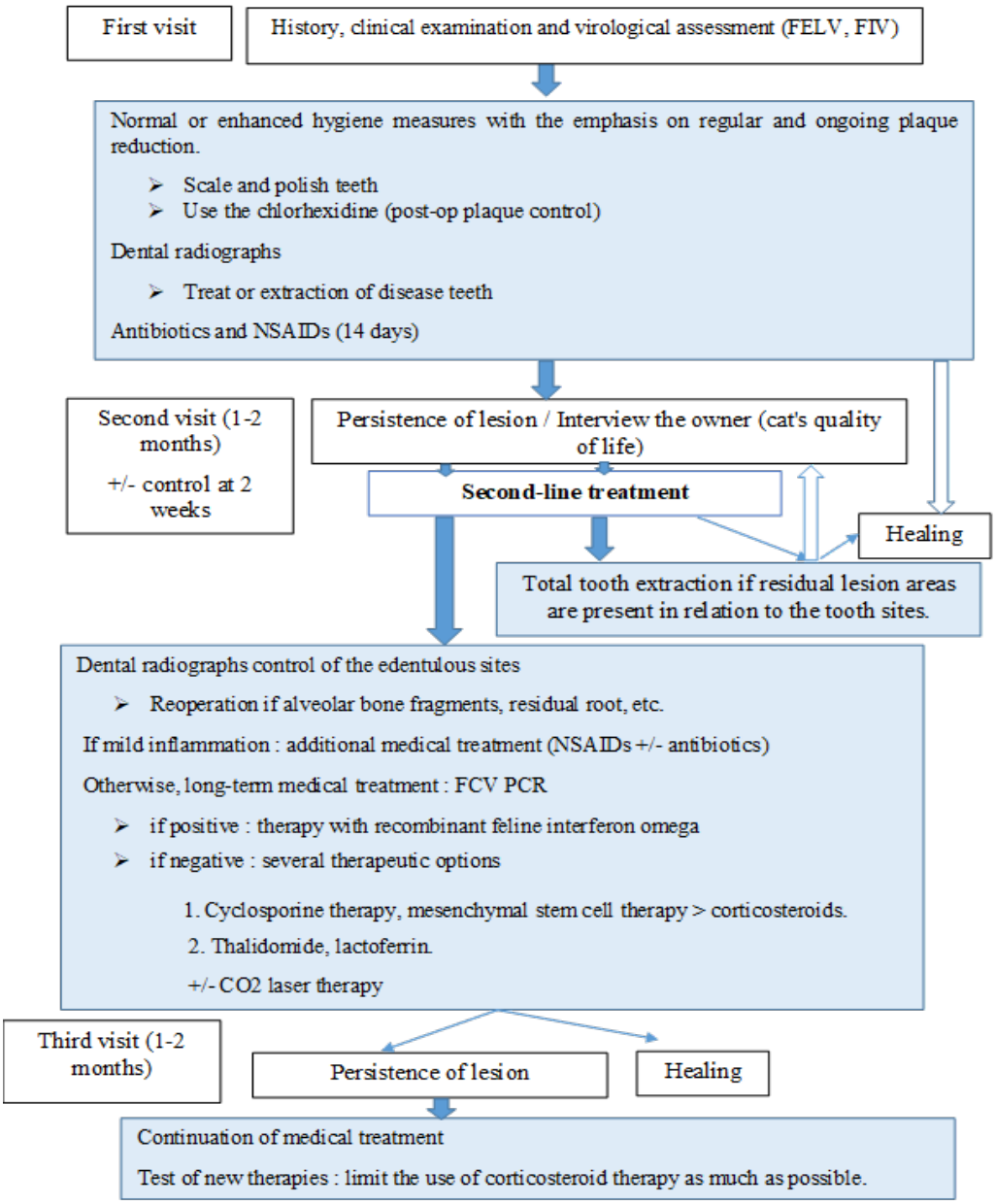


**Figure 2.**
Proposed therapeutic approach for a cat with FCGS.

ment is the highest evidence-based recommendation. PME also has other advantages such as reduced anesthetic time, less surgeon fatigue, and minimized surgical trauma. If there is no positive response within 1 to 4 months, FME may be pursued as the second stage of treatment. The most successful long-term treatment for FCGS is extraction of all premolars and molars, along with careful smoothing of the alveolar bone [10, 31]. Extraction of the rostral teeth is indicated when inflammation involves their gingiva [32]. While some practitioners perform FME when significant oral inflammation is present [1], others prefer to leave the canines and incisors intact, if possible [25, 32]. The vast majority of cats have an excellent response to extractions , requiring no additional therapy [4, 31, 32]. If extraction therapy is not effective, it is usually due to the presence of retained roots [31, 32]. For this reason, postoperative dental radiographs must be exposed to document complete extraction of all tooth roots [31, 32, 62].

### *Medical management*

When owners are reluctant to have multiple extractions performed, medical management may be attempted as an alternative, however this approach has several disadvantages :

- Many products used are oral medications, which require once or twice daily administration.
- Medical therapy is almost invariably a life-long process, and many products have significant side effects.
- No medical protocol has shown to be completely effective ; usually they only reduce the clinical signs temporarily [25, 62].

Medical management consists of palliative measures, including systemic analgesics to treat associated pain, anti-inflammatories to treat the oral inflammation, and antibiotics to treat secondary infections [58]. Other available treatments are described mostly for cases that fail to respond to surgical intervention and offer variable response rates. These include: systemic ciclosporine [63], topical or systemic rFeIFN-ω [4] and MSCs therapy [20, 64].

#### Antibiotics

Systemic antibiotics may decrease some oral inflammation. However, this effect is generally temporary at best, and most cats will experience relapse, often even during the course of antibiotic therapy [32, 62].

#### Pain management

Regardless of modality, all treatment options require adequate pain management. Appropriate therapy depends on factors including comorbidities (eg, renal or hepatic insufficiency), concurrent medications being administered (eg, corticosteroids), patient compliance, and the owner's ability to assess and manage oral pain. Typically, long-term pain management includes administration of opioids (eg, buprenorphine) in combination with gabapentin. A recent randomized, prospective, blinded, controlled, crossover study showed that buccal administration of buprenorphine had a significant effect on reductions in pain scores, while maintaining low interindividual variations in plasma drug concentration in cats with FCGS [65].

#### Corticosteroids

Corticosteroids are, by far, the most commonly used and effective drugs for immune modulation, resulting far more reliable clinical improvement than antibiotic therapy [32]. Prednisolone, a short-acting corticosteroid, is often used to reduce inflammation [17]. However, long-term use may have side effects, such as induction of diabetes mellitus and opportunistic infections [62, 63]. Chronic corticosteroid therapy should only be used as a last resort option, typically only when surgical treatment is declined [1].

### *Recombinant feline interferon omega (rFeIFN-ω)*

Feline interferon is reported to provide both antiviral and immunomodulatory effects, resulting in restoration of the normal local immune system [1]. IFNs are a group of signaling proteins that have the ability to interfere with viral replication. rFeIFN- ω is marketed for use against viruses like CPV, FeLV, FIV, FHV-1 and FCV [66].Oromucosal absorption of IFN has been shown to stimulate local immunomodulation via oropharyngeal lymphoid tissues, whereas gas trointestinal absorption leads to degradation of the glycoprotein [67]. In a controlled, randomized, double-blinded study evaluating oromucosal administration of rFeIFN- ω over 3 months in 19 cats, substantial improvement was seen in 45% of the cats, of which 10% achieved clinical remission. Another recent controlled study showed that subcutaneous administration of rFeIFN- ω may be effective for the treatment of FCGS in FCV-positive cats, as it appears to inhibit FCV replication [68]. Several studies have shown efficacy in resistant cases but, current evidence does not demonstrates its efficacy as a primary treatment [1].

### *Cyclosporine A*

Cyclosporine A provides immunosuppressive effects primarily via inhibition of T-cell activation through downregulation interleukin-2 expression, a proinflammatory cytokine involved in a positive feedback loop that increases T-cell numbers [69]. It may

also have inhibitory effects on B-cell reproduction. Cyclosporine A has been proposed as an immunosuppressive drug for cats with caudal stomatitis [32] and some have promoted it as an alternative to extractions in order to avoid glucocorticoid use. While there is no published information that supports the use of cyclosporine A prior to extractions, it has been shown to be effective in cases refractory to extraction therapy [63] and may provide an alternative to long-term steroid therapy. Therefore, Niemiec [1] noted that cyclosporine should be reserve only for use in patients in which medical management is necessary post extraction [25]. Cyclosporine A must be used with caution in cats with hepatic or renal disease, and there are reports of fatal opportunistic infections associated with its use [70]. The bioavailability of the 3 available forms of cyclosporine is quite variable, and dosing depends on which form is used [62]. A veterinary specific formulation, Atopica (Novartis), is approved for use in cats with feline atopy and may be considered a suitable option for FCGS management [1].

### *Mesenchymal stem cells (MSCs)*

MSCs are fibroblast-like, multipotent stem cells that have immunomodulatory effects through inhibition of T-cell proliferation, alteration of B-cell function, downregulation of MHC-II on antigen presenting cells, and inhibition of dendritic cell maturation [71, 72]. The efficacy of both autologous and allogeneic, fresh, adipose-derived MSCs administered intravenously has been studied in cats with refractory FCGS [20, 71]. Treatment with autologous adipose-derived MSCs in 7 cats resulted in a positive response rate of 71.4% (reflected by clinical remission in 42.8%, substantial improvement in 28.6%, and no response in 28.6% of cats), over a follow-up period of 6 to 24 months [71].

## Conclusion

The etiology of FCGS is often unknown and a multifactorial disease, with potential contributions from bacteria and viral pathogens , genetic predisposition and environmental stressors. Epidemiological studies of the disease are rare, and many features have yet to be documented. Successful managment of this complex requires a logical diagnostic approach and to understand the possible etiopathogenic mechanisms, it is essential to understand the epidemiological characteristics of the disease in order to propose available treatments and preventive approaches.

## Authors' Contributions

The author contributed alone to the realization of the work.

## Conflict of interest

The authors declare that there is no conflict of interest.

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