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The inflammatory diseases of the feline oral cavity are common in the daily clinical practice and many times, constitute therapeutic challenges to the veterinarian. (Frost & Williams, 1986; Gruffydd-Jones, 1991; Knowles et al., 1989). According to the classification proposed by Waters et al. (1993), the feline gingivitis can be classified in four degrees, according to the intensity and characteristics of the oral lesions. The degree zero (0) corresponds to the absence of any gingivitis sign; one (1) to a mild gingivitis, in which a discrete hyperemia in the gingival margin is seen, without signs of tissue hyperplasia; two (2) moderate gingivitis, where the cat presents an evident gingival hyperemia but without signs of hyperplasia and/or gingival ulceration; three (3) severe gingivitis, with evident hyperemia of the gingival margins, areas of hyperplasia and/or ulceration of the gingival tissue, signs of periodontal disease such as formation of periodontal pockets, atrophy of the dental alveoli and tooth loss; four (4) severe gingivitis, where the cat presents a very evident gingival hyperemia, hyperplasia and/or generalized gingival ulcerations and bleeding of ulcers in the palate, faucitis, glossitis, loss of teeth and friability of the gingival margins.

This oral inflammatory disease in cats primarily leads to a simple gingivitis, in which only gingival tissue is affected, presenting hyperemia, edema and mild friability. As the condition degenerates, stomatitis takes place and the inflammation extends beyond the gingival junction, affecting all oral cavity. Cats with this chronic status present intense inflammation of oral and gingival mucous membranes, frequently with ulceration and extensive proliferation of a granulation tissue, with lesions that may extend to the regions of the palate, tongue and faucies. (Deihl & Rosychuck, 1993; Pedersen, 1992).

Other clinical signs very common in these oral affections are intense halitosis, dental friability and dysphagia. In extreme cases it is very painful and the cat becomes anorectic. (Gruffydd-Jones, 1991; Harvey, 1994).

In general, factors that may contribute to the development of gingivitis and stomatitis are: diet, oral conformation, oral hygiene performed by the owner, specific genetic characteristics (juvenile gingivitis of the Abissinian, Persian and Maine Coons), immune-mediated diseases and systemic diseases like infections by the feline immunodeficiency virus (FIV), feline leukemia virus (FeLV) and herpesvirus and/or calicivirus. (Deihl & Rosychuck, 1993; Tenorio et al., 1991).

The calicivirus (FCV) belongs to the Caliciviridae family and it is a small virus, of spherical capsid, non-enveloped, of RNA simple chain and genome of approximately 7.7kb (Hurley & Sykes, 2003; Thiel & König, 1999). It is the responsible pathogen for great part of the infections of the superior respiratory tract of domestic cats (Hurley & Sykes, 2003; Lommer & Verstraete, 2003; Knowles et al., 1991; Radford et al, 2000; Thiel & König, 1999). It has been described the involvement of FCV in chronic stomatitis, as well as in the ulcerative e ulceroproliferative glossitis refractory to the treatment (Diehl & Rosychuck, 1993; Hurley & Sykes, 2003). The incidence of the infection for FCV in cats with chronic gingivitis-stomatitis-pharyngitis, reported in several studies, varies from 20 to 90%, however, it is not known for sure which the involvement of FCV as primary etiological agent is. (Addie et al, 2003; Diehl & Rosychuck, 1993; Lommer & Verstraete, 2003).

The cats that recover of superior respiratory tract disease can develop persistent infection in the oropharynx, called state of carrier of FCV. The virus is eliminated continually, although the magnitude of the elimination varies with the time and among individuals. Those cats can serve as infection source for other susceptible cats. In many animals, the elimination of the virus finishes weeks to months after the infection, but in a small number of cats, the elimination lasts to the whole life (Hurley & Sykes, 2003). Because of the state of chronic carrier, the prevalence of the infection for FCV in healthy cats is high, reaching from 8 to 24% of the domestic cats (Hurley & Sykes, 2003; Thiel & König, 1999). As approximately 85% of the cats with chronic stomatitis seem to be chronic carriers, this fact can be a prerequisite for the induction of the chronic stomatitis in those individuals (Knowles et al, 1991).

Poulet et al. (2000) demonstrated that the FCV associated to the pictures of chronic stomatitis-gingivitis is antigenically different from the ones that cause disease of the respiratory tract, being these probably result of the chronicity of the infection. It is believed that there are no specific biotypes for different manifestations of diseases, but antigenic variation, with the presence of hypervariable areas of the proteic capsid typical of the Caliciviridae family. In a study accomplished by Lommer & Verstraete (2003), it was demonstrated that 88% of the cats studied that presented chronic gingivitis-stomatitis had infection for FCV and feline herpesvirus type 1 (FHV-1) concomitantly. The authors believe that the infection for FCV and FHV-1 combined with bacterial plaque stimulates the increase of lymphocytes infiltration in the oral mucosa. Anyway, the causal relationship could not be demonstrated.

Materials and Methods

78 cats from different ages, sex and breeds were divided into two groups of 39 animals each. The group I is composed by cats with gingivitis (grades 2 to 4), according to the system proposed by Waters et al. (1993) and the group II is composed by cats without gingivitis, constituting a control group. From each animal, one sample of the oral mucosa were collected with a swab and analyzed through polymerase chain reaction (PCR), to search for calicivirus. The PCR was performed as previously described by Sykes et al. (2001).

Results

Seventy eight cats was submitted to oral PCR test for FCV. For the 39 cats of group I, 20 cats (51.28%) were positive for the presence of FCV in gingival tissue. For 39 cats of group II, 8 (20.51%) were positive for the presence of FCV. It was used the Fischer’s exact test, and a p value < 0.05 was considered significant. A significant difference between the groups (p value = 0.0088) was obtained.

Discussion and Conclusion. The experiment demonstrates a significant high frequency of FCV in cats with chronic gingivitis. The high occurrence of infection by FCV among cats with chronic gingivitis may suggest a role of this virus in the onset and progression of gingival disease. In fact, several studies demonstrate that cats infected by the FCV have a greater predisposition to chronic gingivitis, associated or not to clinical manifestations of this disease (Hurley & Sykes, 2003; Knowles et al, 1991; Lommer & Verstraete, 2003; Thiel & König, 1999).

The correlation between FCV infection and development of chronic gingivitis is still speculative; the FCV could be an opportunistic agent, or...
have some influence in the onset of progression of gingivitis. The difference between the prevalence of infection for calicivirus in animals with and without gingivitis found in this study reinforce others reports and suggests participation in the pathogenicity or even in the perpetuation of the gingival disease. However, this association still is a speculation and further researches are needed.

References

**Keywords:** chronic, gingivitis, cats, calicivirus