RESEARCH ON IDIOPATHIC STOMATITIS IN CATS

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Abstract

Introduction.
In the last decade there have been great advances in the study of chronic oral inflammation impacts on the health of the whole animal body.
Materials and methods.
Clinical investigations were performed in the Surgical Clinic of FMV Iaşi, on 32 patients presenting characteristic symptoms.
Results and conclusions.
Secondary clinical outcomes of the research are those that revolutionizes diagnosis and treatment plan, or need treatment more than the technology itself. Gingivostomatitis have long been known, studied and systematized, but some of them have not yet been fully described and investigated as cat idiopathic gingivostomatitis. Failure diagnosis and treatment of this disease and others that are localized specifically in the oral cavity is due to the fact that dentistry is basically a new branch, implemented shortly in veterinary medicine and began to grow in treatment of pets. Treatment of this disease should be seen as a product of comparative analysis of the advantages and disadvantages involved in each case. In addition to restoring the functions of the stomatognathic apparatus, treatment raises a number of shortcomings related to threatening the integrity of various organs of the animal, to facilitate the emergence of other diseases, the sometimes high cost of treatment and, not least, the risk of its failure.

Keyword: cat idiopathic gingivostomatitis,cat stomatitis.

INTRODUCTION

Our research objectives were to diagnose and treat idiopathic stomatitis in cats called limfocytic plasmocytic stomatitis and its complications by clinical and laboratory examinations. We also aimed to implement a clear diagnostic techniques.
A key objective was to make differential diagnosis between: gingivostomatitis feline eosinophilic granuloma and some viral diseases such as feline leukemia virus (FeLV) and Feline immunodeficiency virus (FIV), feline calicivirus (FCV), which interference with the disease and often produce lesions in the oral cavity.

We seek to restore the integrity of the oral mucosa with effective treatment regimens of patients diagnosed.

MATERIALS AND METHODS

The research was carried out during 2008 - May 2012, a total of 184 cases examined cases.
Subjects included in the study were presented by their owners at clinics of the Faculty of Veterinary Medicine of Iasi, dental clinic.
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The diagnosis was established by clinical and laboratory examinations.
The study was conducted on 32 patients of the total cases of which 17 cases were diagnosed with gingivostomatitis lymphocytes plasmocytes syndrome, 7 cases of ulcerative and necrotic gingivostomatitis**,** 4 cases with hyperplasia stomatitis and other 4 cases with other reasons.
Pet owners have been properly informed and help in decision making for a feature aimed at prolonging life and patient comfort habitat creation owners.
Clinical diagnostic elements and laboratory
Dental interventions were preceded by clinical examination of the oral cavity examination respecting semiological methods.
History, reason for the request, the conditions of feeding, maintenance, previous diseases, physiological status, and hereditary predisposition to previously used medications are important in diagnosis.
Opening the oral cavity (manual or appropriate speculum) and was one of its inspection examinations helpful in the diagnosis.
If necessary we performed a radiological examination. In case tissue proliferation is performed histopathological examinations, hematological and biochemical tests for feline leukemia virus (FeLV), feline immunodeficiency virus (FIV) and Feline calicivirus (FCV).
All these steps were necessary elements to develop a diagnostic certainty which allowed us to implement a correct therapeutic behavior.

Inflammatory lesions, stomatitis

Stomatitis is inflammation of the mouth, regardless of origin, location and evolution, in which the lesion extends over large portions of the mucosa.
If the inflammatory process is limited, we distinguish gingivitis, glossitis, oropharynx and palate.
Stomatitis involved in the etiology of a number of local and general factors.

Among the general factors that favor the development of stomatitis include: metabolic disorders, vitamin deficiencies, endogenous intoxications (uraemia, hepatic failure, diabetes) or exogenous (medicinal) endocrinopathies, infectious, allergic or immune-mediated diseases.

Local factors are the most frequent saprophytic germs of mouth, which become pathogenic under certain conditions trophic private. Depending on the evolution of the inflammatory process, the multitude of causes that generate them and pathological changes, stomatitis can be acute and chronic. Both forms of evolution, depending on the etiology, may be nonspecific and specific.

I met the nonspecific stomatitis inflammation of the oral mucosa, which we grouped by morphological changes in exudative stomatitis, stomatitis hyperplastic, necrotic stomatitis.

Hyperplastic proliferative stomatitis are inflammatory lesions that occur predominantly in the gums. Based on a hormonal disorder, drug or combination of other factors.

I found a histopathologic epithelial cell proliferation, as proving the presence of mitoses hyperplasia. In chorion found intraepithelial haemorrhage, infiltration lymphocytic and hstiocytic**,** histiocytic differentiation of and synthesis of collagen fibers (Figure 1).

Ulcerative and necrotic gingivostomatitis are the predominant inflammatory type alterative necrotic changes in gingival and buccal mucosa.

This is where a variety of biotic agents, whose aggression is favored by general and local factors.

Exacerbation of virulence factors that favor local saprophytic flora are chronic inflammatory processes: shallow marginal periodontitis and factors that maintain chronic irritation. These factors influence microbial germs multiplying - streptococci, aerobic, followed by increasing metabolism with high consumption of oxygen, which creates anaerobes development.

Macroscopic inflammatory process starts with congestion, mucosal edema, which appears purplish-red, swollen and bleed easily. Subsequently appear initially localized ulcers on the gums, the top inter-dental papilla, extending the oral mucosa, tongue, pharynx. Ulcers have irregular borders surrounded by hyperemia, have a tendency to confluence and ashes covered by false membrane. Ulcers can be shallow or deep. Evolving disease with enlarged jaw.

Microscopically, the absence of squamous epithelium limited to the presence of necrotic debris, fibrin, microbial germs and polymorphonuclear

neutrophil. In chorion appear hyperemia, edema and diffuse polymorphonuclear infiltration (Figure 2).
Evolution is generally good. By necrosis of interdental papillae amputation creates job retention, however, favors the formation of tartar.

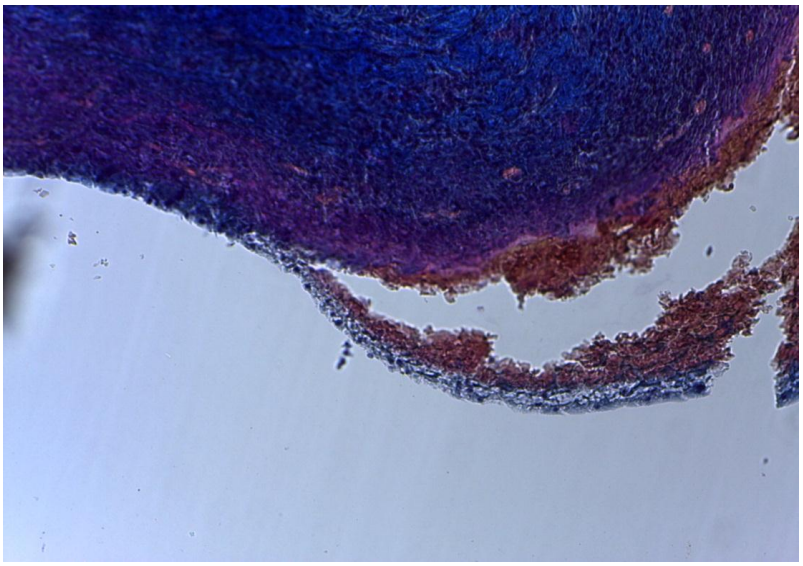

Figure 1. Gingivostomatitis hyperplasia. Col. HEA x 200.

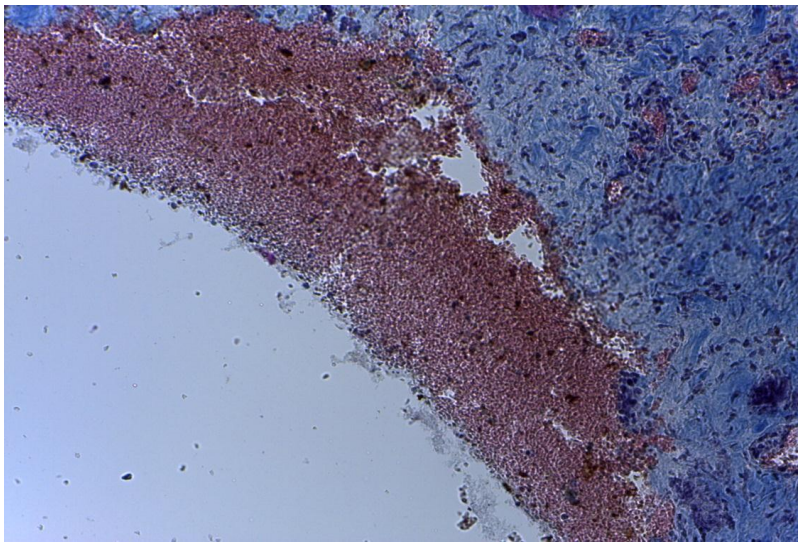

Figure 2. Active ulcer in the lining of the gingival Col. HEA x 200

Inflammatory hyperplastic lesions (pseudo lesions). Inflammatory hyperplasia is the term used to describe numerous nodular neoformaţii the mouth, which histologically resemble inflammatory granulation tissue.
This similarity is based on the degree of development of inflammatory action and reaction components in cicatricial.
Thus, sometimes the look is predominantly epithelial hyperplasia with reduced conjunctive reaction.
In other cases the lesion is fibroma, including angiomatosis changes or collagen sclerosis, with minimal epithelial component.
Depending on the degree of maturation of granulation tissue may be young, exuberant, rich neoformation vessels. Sometimes, reducing cell population and building a neovasculature sclerosis causes collagen scar.
This variation is reflected in the variability of histological aspects of clinical issues that may have inflammatory hyperplasia.
Terms like "fibroids" or "papilloma" are used to describe these lesions, even if there are no signs pointing to a neoplastic etiology.
The main etiologic factor of these injuries is chronic trauma (dental fractures etc). In many cases, chronic irritants can be clearly demonstrated (eg palatal hyperplasia).
Most lesions are localized on the surface of the mouth, irritation exposed location. Are classified as inflammatory hyperplasia two deeper lesions such as granuloma with giant cells of bone repair, thanks histological and clinical course.
Eliminating chronic irritant and inflammatory hyperplasia lesion, they will relapse, confirming their benign nature and etiology of chronic irritant.
Inflammatory fibrous hyperplasia may locate at any point in the mouth as a neoformation pedicle and sessile. Hyperplasia is often identified with papillomas, if the lesion is pedunculated and keratinized, and fibroids, if the lesion is sessile, tough and covered by a thin squamous epithelium. The gum, an injury of this type is often confused with epulides. Histopathological examination found neoformation vessels and fibroblast hyperplasia (Figure 3).
Most inflammatory fibrous hyperplasia remain small. Lesions with a diameter greater than 1 cm are rare on the tongue and floor of mouth, probably because it limits the size of masticatory trauma.
Biting pressure usually causes lesions keratinization.
In the differential diagnosis of inflammatory hyperplasia should be considered that the lesion may be true or injury papilomatoasă papilloma viral origin. Nodular formations on the surface of the tongue can be

neurofibromas, lymphoid nodules or cystic dilatation of mucous gland ducts.
Recurrences after removing inflammatory fibrous hyperplasia, can be interpreted as a persistence of chronic irritation factor action.
Inflammatory fibrous hyperplasia have malignant potential, however they must be removed surgically. Their appearance brittle, and often ulcerated bleeding arising from their histological structure.
Microscope is composed of a richly vascularized granulation tissue with minimal collagen support. Are present abundant polymorphonuclear cells and chronic inflammatory cells (Figure 4, Figure 5).
Tissue is swollen and the presence of microabscesses. The existence of these lesions indicates the need for periodontal inspections, appropriate treatment should eliminate the irritating factors scaling and gum pockets.
All these pathological conditions described above are irritants to the lining of the oral cavity, the premise is easily prone to a chronic inflammatory reaction that ultimately interested in the free edge of the gums, sublingual mucosa receses, soft palate and glosopalatal folds.
From this moment the actual installation of the syndrome of chronic stomatitis, things evolve in a poor outcome until it reaches the visible expression of clinical signs. They consist of difficulty in mastication and prehension, reaching a stage when sick cat can not chew or dealing with a high difficulty. In many cases animals unable to reach could feed, but usually refuse liquids, such stomatognathic apparatus is unable to work. Disease can be identified in the early stages of the veterinarian advised that the examination of the oral cavity will notify the changes. Early detection of disease or predisposing factors that are precursors worthy of attention for establishing a diagnosis and proper treatment to prevent disease progress will bring a sick animal disease control stage.
From research conducted both by us and by other practitioners symptoms do not resolve itself only in a very small percentage, which is considerably increased by the application of appropriate treatment regimens.
Association with immunodeficiency, feline leukemia feline coronavirosis is demonstrated in many cases of gingivostomatitis syndrome. For confirmation usually use standardized tests.
In terms outlined histopathological infiltration by lymphocytes, monocytes and plasma cells. These can generate advanced chronic stage appearance of plasmacytoma.

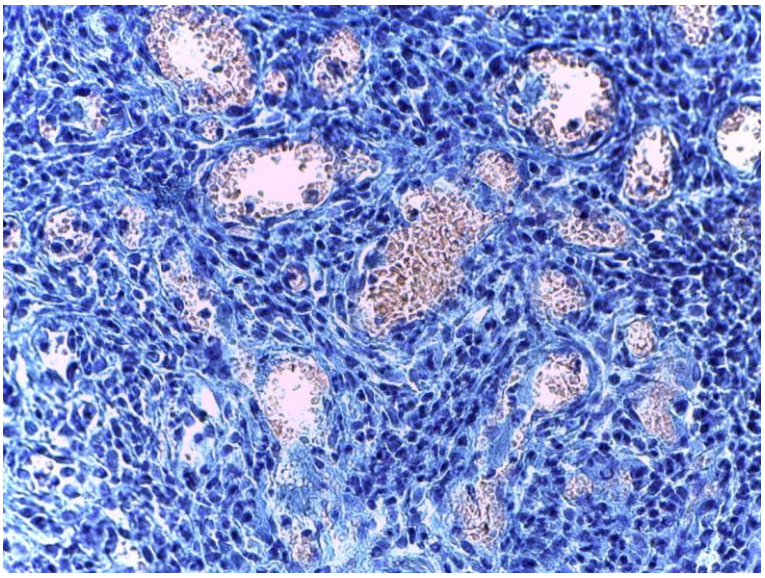

Figure 3. Fibroblast hyperplasia. Col.HEA, x 200.

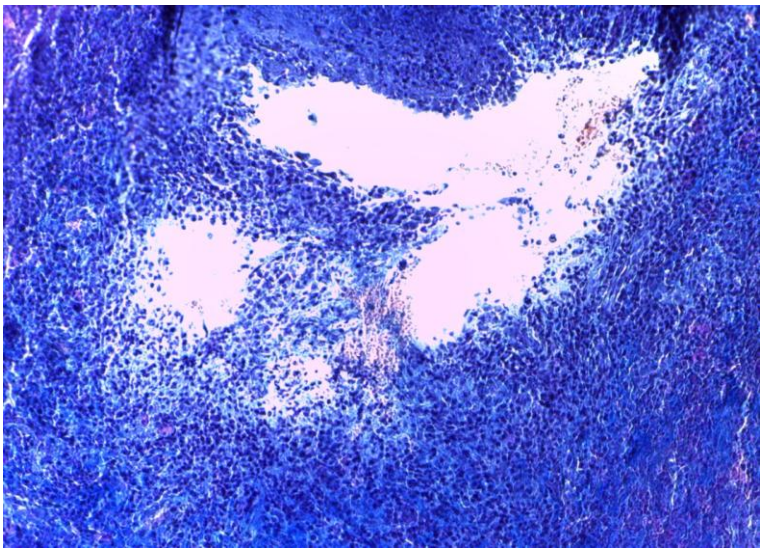

Figure 4. Focal inflammation. Col. HEA, x 200.

Following a strategy established treatment planning increases the likelihood of obtaining a positive result with satisfaction both parties. Treatment plan designed to ensure a net improvement in health status, a benefit for the patient and owner. They must understand and accept that treatment success

depends largely on their efforts sequentially adequate maintenance of oral health through the scheme imposed. Treatment planning is not an exact science, but an art supported by clinical experience, scientific knowledge filtered.

Treatment in controlling the inflammatory response consisted in prednisone administration at a dose of 2-4 mg/kg/day and if the patient responds well to treatment, the dose will be reduced gradually. Simultaneously applied vitaminotherapy A, and zinc mineral supplements based on soft tissue maintenance and insisted on local professional prophylactic treatment consisting of scaling and brushing and administration of chlorhexidine for oral hygiene maintenance.

Inflammation of the gums and mouth can come from a variety of local and systemic causes. The most common cause of gingivitis is dental plaque and tartar (Figure 6, Figure 7, Figure 8, Figure 9).

Calicivirus and herpes virus can cause ulcerative stomatitis. Cats affected by this virus presents as clinical signs: sneezing, runny purulent ocular and nasal, oral mucosal ulceration apathy and especially language and palate.

Chronic gingivitis, tartar unless it is associated with infection with feline immunodeficiency virus (FIV).

Success in the treatment of gingival stomatitis attention involved a primary cause of the disease if it can be identified.

Clinical studies have suggested a possible involvement of different viral agents, particularly herpes virus and species of Gram-positive bacteria anaerbobe. However, attempts to reproduce the disease using these presumed infectious etiological agents were unsuccessful.

Cats with chronic stomatitis require a thorough investigation before any treatment. Its aim is to reach a diagnosis in itself, but mostly an attempt to identify root causes. Such analysis includes testing for FIV and FeLV, haematological and biochemical blood tests and sometimes routine biopsy and microscopic examination of tissues affected. A thorough oral examination and dental, including radiographs of the entire oral cavity for the presence of periodontitis, resorptive lesions, root debris and other injuries is mandatory. Systemic diseases such as chronic renal failure and diabetes, which may predispose to the development of severe gingival inflammation in the presence of the plate, should also be excluded before starting any treatment.

Chronic gingivostomatitis (CGS) describes a clinical syndrome characterized by focal or diffuse inflammation of the gums and oral mucosa (Figure 10). The most common laboratory results described in cats with

CGS include serum globulins, predominantly hypergammaglobulinemia and submucosa inflammatory infiltrate consisting of plasma cells, lymphocytes, macrophages and neutrophils. Serum globulins in cats affected and inflammatory nature of the submucosa led some authors to suggest that there could be an immunological basis for disease. So far, no intrinsic immunological abnormality in cats affected with CGS was not identified, however, is immune mediated disease.

Another treatment consisted of up to 10mg Triamcinolone acetate/administration in combination with metronidazole 30-60mg/kg/day in two divided doses for 10-15 days. Vitaminotherapy complex A, B, C and E, and for FeLV and FIV viral disease was used specific treatment. In this case a possible cause was found essential for inflammation in the oral mucous membranes namely FIV infection.

Another general treatment consisted of administration of immunomodulatory effect of levamisole for dose 2-5mg/kg/day or 30mg/ animal three times a week plus clindamycin 5mg/kg corp/12 hours for 3-4 weeks. There were 3 doses of interferon within 7 days and vitamins therapy complex, A, B, C and E.

In this case the cat was re-examined at 6, 10, 14 weeks and oral samples were taken for histo-pathological examination. Notice a significant improvement in oral inflammation.

This disease is considered to be an inappropriate response to oral antigens, ie plaque present on the surface of teeth.

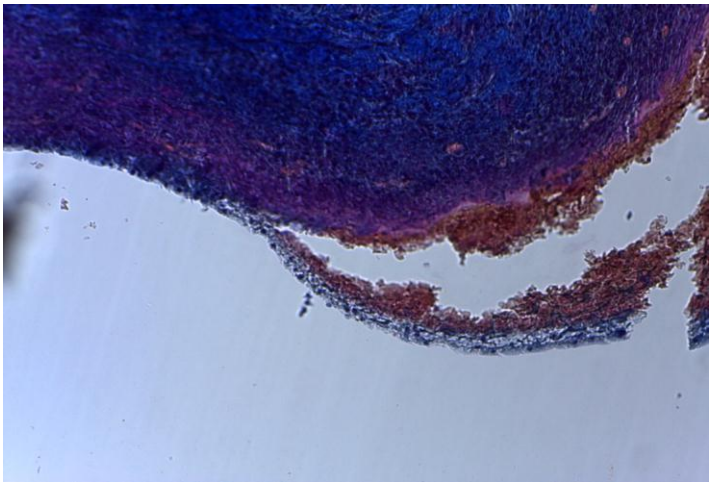

Figure 5. Gingivostomatitis hyperplasia. Col. HEA x 200.

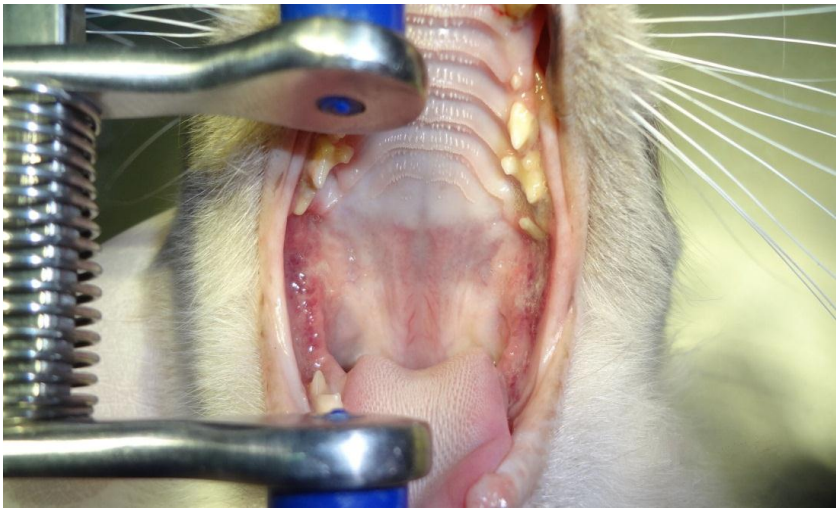

Figure 6. Gingivostomatitis on the soft palate and its folds.

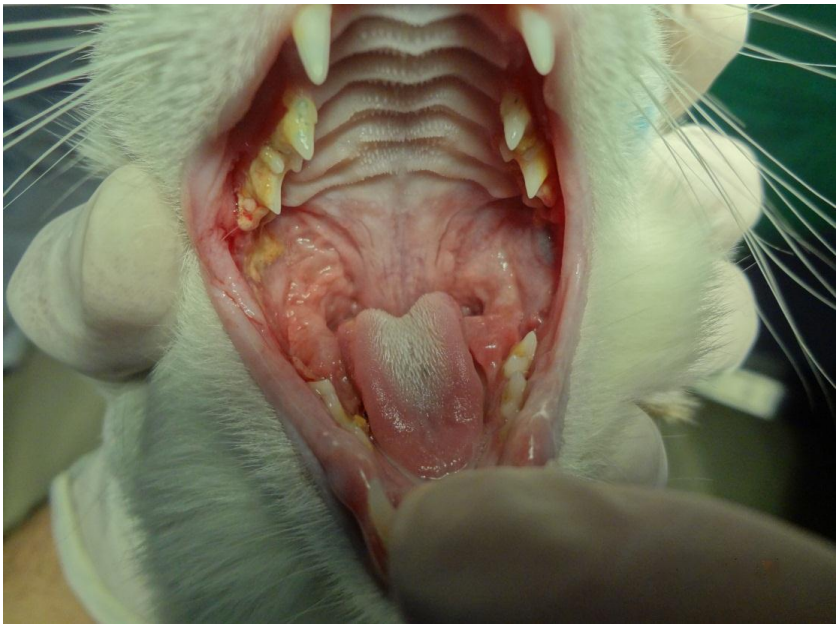

Figure 7. Glosopalatine mucosal hyperplasia

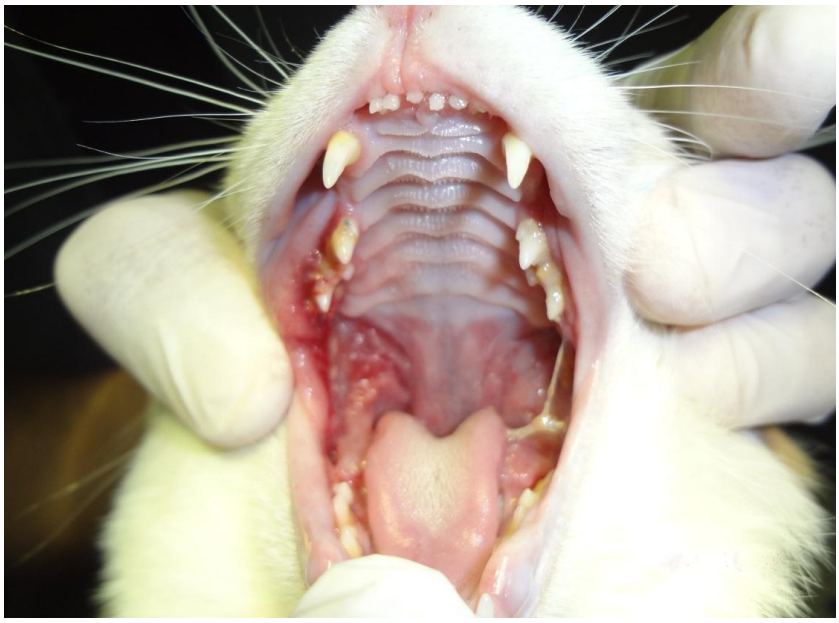

Figure 8. Location on molar gingival mucosa.

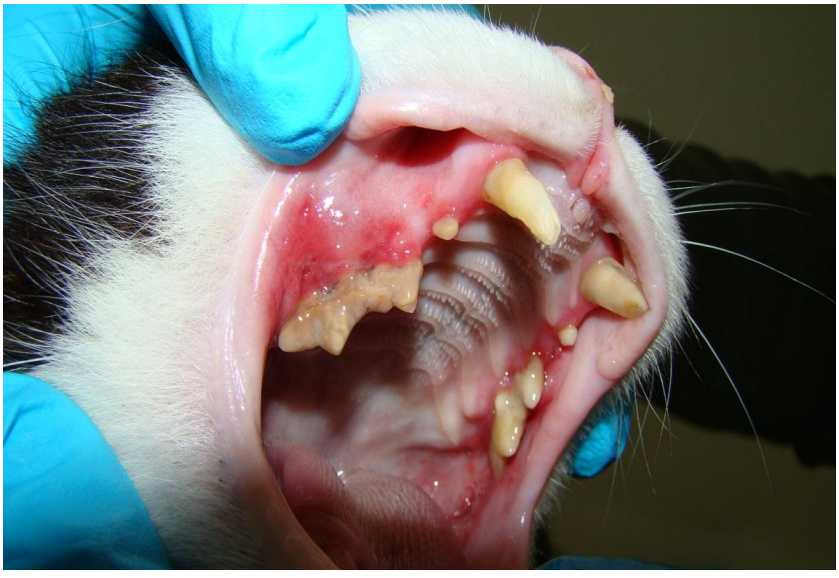

Figure 9. Lateral aspect of gingival inflammation and oral mucosa.

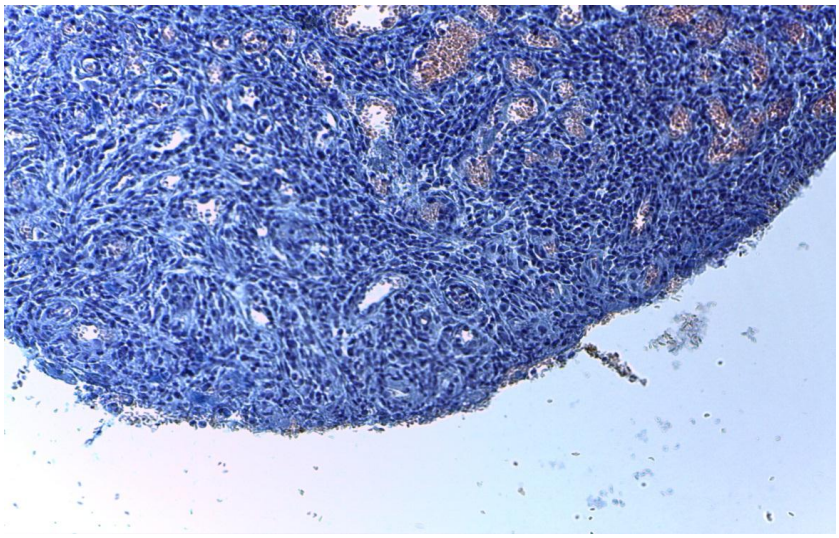

Figure 10. Fibrous chronic gingivostomatitis. Col. HEA, x 200.

CONCLUSIONS

Lymphocytic plasmocytic chronic stomatitis is a disease whose cause is somewhat unknown. Has several names including: chronic feline gingivo-stostomatitis, gingivo-pharyngitis plasma cell and idiopathic gingivostomatitis.

Cats of any age and breed can be affected, but young cats are prone to disease. Signs included anorexia, salivation, dysphagia and halitosis. Intermittent exacerbations and relapses are common.

Chronic gingivostomatitis describes a clinical syndrome characterized by focal or diffuse inflammation of the gums and oral mucosa. The most common clinical results described in cats with CGS include serum globulins, predominantly hypergammaglobulinemia and submucosa inflammatory infiltrate consisting of plasma cells, lymphocytes, macrophages and neutrophils. Serum globulins in cats affected and inflammatory nature of the submucosa led some authors to suggest that there could be an immunological basis for disease. So far, no intrinsic immunological abnormality in cats affected with CGS was not identified, however, may be immune mediated disease.

Cats with chronic stomatitis require a thorough investigation before any treatment. Its aim is to reach a diagnosis in itself, but mostly an attempt to identify root causes. Such analysis includes testing for FIV, FeLV and FCV,

haematological and biochemical blood tests and sometimes routine biopsy and microscopic examination of tissues affected.
Satisfactory results were recorded in general about treatment with prednisolone at a dose of 2-4 mg/kg/day. If the patient responds well to treatment, the dose will be reduced gradually. Simultaneously applied vitamin therapy with vitamin A, C and zinc mineral supplements based on soft tissue maintenance and insisted on local professional prophylactic treatment consisting of scaling and brushing and administration of chlorhexidine for oral hygiene maintenance.
Another regimen with equally good results consisted of Levamisole for immunomodulating effect dose 2-5 mg/kg/day or 30 mg/animal three times a week plus clindamycin 5 mg/kg corp/12 hours 3-4 weeks. There were 3 doses of interferon within 7 days and vitamin therapy complex, A, B, C and E.
A thorough oral examination and dental, including radiographs of the entire oral cavity for the presence of periodontitis, odontoclastic resorption lesions, root debris and other injuries is mandatory. Systemic diseases such as chronic renal failure and diabetes, which may predispose to the development of severe gingival inflammation in the presence of the plate, should also be excluded before starting any treatment.

Guidance

Lymphocytic plasmocytic stomatitis require aggressive therapy may include teeth cleaning, debridement of necrotic tissue and extracting any teeth close. Extraction of all premolars and molars may be necessary and results in even diminish distant lesions of teeth.
Cats with chronic stomatitis require a thorough investigation before any treatment. Its aim is to reach a diagnosis in itself, but mostly an attempt to identify root causes. Such analysis includes testing for FIV, FeLV and haematological and biochemical blood tests and sometimes routine biopsy and microscopic examination of tissues affected.
A complete blood count is needed to verify cytopenias every 2 weeks during the incubation period and every month during improvement. A urine analysis should be done to verify proteinuria.
Local application of chlorhexidine oral cavity is required. Accumulation of plaque can be reduced by special diets and using a dry toothbrush.
Recommended as using only interferon therapy in cats diagnosed positive tests for FeLV, FIV and FCV, which was an improved. It also assesses intralesional injection of interferon as a possible protocol.

Current treatment recommendations for cats with CGS include a combination of periodontal treatment and home care regimen that plaque buildup is kept to a minimum. In some cats can lead to a reduction in inflammation. Unfortunately, most cats do not cooperate adequately in the provision of care at home and board passes the critical level. These cats require extraction of premolars and molars. In some cats is necessary to remove all teeth. Extraction of all premolars and molars gave the most reliable results, up to 80% of cats were clinically cured. The other 20% that are not susceptible to extraction can be treated using the schemes described above.

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