FELINE STOMATITIS

Rosalind Gaskell

Inflammatory conditions of the mouth are a relatively common problem in the cat and are often notoriously unresponsive to treatment. In many cases this is because the underlying causes are just not understood, and thus it is impossible to use any rational or specific treatment.

The major presenting signs of feline stomatitis are dysphagia, generalised signs of oral discomfort, halitosis, excessive salivation, sometimes bleeding, inappetance, and in some animals there may be some associated weight loss and dehydration.

First of all it is most important to establish whether or not the disease is primary, or whether the cat is suffering from some other more generalised disease in addition to the stomatitis. For example, conditions such as chronic kidney disease and, possibly, diabetes may predispose to oral ulceration and inflammation of the gums. In chronic renal disease this is thought to be due to the effect of bacterial conversion of high blood and salivary urea to ammonia, which is irritant. In diabetes the mechanism is not clear, but is thought to be due to reduced tissue resistance and repair, and secondary bacterial and fungal overgrowth because of the glucose in saliva. It has also been suggested that the presence of feline leukaemia virus infection may be associated with a chronic ulcerative stomatitis which is typically refractory to treatment, presumably as a result of accompanying immunosuppression. Therefore all cases should be tested for feline leukaemia virus status, as an aid to prognosis.

Primary feline stomatitis may be divided somewhat arbitrarily into conditions which are acute, and those which are more chronic. The acute conditions are of relatively sudden onset, and of more finite duration, and include such things as traumatic lesions due to road accidents and falls - the cat classically lands on his nose. This results in minor injuries such as broken or chipped teeth and lacerations to the tongue and lip; more severe lesions include cleft palates and mandibular and maxillary fractures. Generally such lesions heal well, although fractures must be wired together fairly quickly to avoid the necessity for more complicated surgery later on.

Foreign bodies such as bits of bone or stick are not often found in the mouth of cats, presumably because they are quite selective in their eating habits. However, sometimes fish bones may get stuck, and they do like playing with needles and threads: sometimes an attached needle will penetrate the mouth or pharynx, and must be removed after radiographic examination, under anaesthesia. In North America it has been suggested that dry cat food can cause abrasions of the hard palate (Johnson and Povey, 1982) but this would seem to be a comparatively rare event, and probably outweighed by the advantage of improved dental hygiene which follows dry cat food feeding.

Chemical irritants - such as household cleaners and disinfectants - are often blamed for mouth ulceration in cats, and in some cases almost certainly do so, but these are probably very uncommon causes compared to the feline upper respiratory viruses, that is, feline calicivirus, and

feline viral rhinotracheitis (FVR) virus. Both of these viruses may produce stomatitis, but of the two, feline calicivirus is much more likely to do so. It has been shown that while there is only one main serotype of feline calicivirus, within this serotype, and varying slightly from each other, there are many different strains of the virus which are of varying pathogenicity for cats. Some of these strains produce mouth ulceration only. These ulcers typically occur on the dorsal surface of the tongue, but they may appear elsewhere, for example on the hard palate, or around the nostrils. Other strains cause a mild upper respiratory disease syndrome with mild sneezing and discharges from the eyes and nose, which may or may not be accompanied by mouth ulceration. Yet other strains produce a more severe syndrome more similar to FVR, some strains produce an interstitial pneumonia where the only clinical sign may be difficulty in breathing or perhaps in young kittens, unexplained death, and finally some strains appear to be relatively harmless and the resulting infection is entirely subclinical. Some strains of feline calicivirus have also been identified which produce only fever, muscle soreness and limping (Studdert et al., 1970; Pederson et al., 1983).

With feline viral rhinotracheitis, however, the characteristic syndrome is a more severe upper respiratory disease, and mouth ulceration is much more of a rarity. Typical signs include fever, depression, and loss of appetite. There is a marked sneezing, nasal discharges, difficulty in breathing, and sometimes coughing, and affected cats often show severe conjunctivitis and accompanying ocular discharges. Microscopically one can also see signs of pharyngitis and tonsillitis, and more rarely small tongue ulcers may appear - all these may lead to excessive salivation, signs of oral discomfort, and difficulty in eating, typical of the cat with acute stomatitis.

Treatment of cases of viral-induced stomatitis must, at present, be limited to antibiotic therapy to control secondary bacterial infection, vitamin supplementation, and good nursing, because, as yet, there are no suitable antiviral drugs marketed to control the virus infection. Whilst lesions are present, soft liquid foods should be given, and if dehydration is a problem, subcutaneous or intravenous fluids may be indicated. In severe prolonged cases, a pharyngostomy tube may be helpful (Lane, 1977).

The more chronic forms of feline stomatitis include conditions such as eosinophilic granuloma, neoplasia, dental disease, and non-specific inflammatory conditions. Eosinophilic granuloma, or labial or so-called 'rodent' ulcer, is a non-neoplastic, chronic, localised and ulcerative lesion, sometimes with raised proliferative edges, which typically occurs at the mucocutaneous junction of the anterior part of the upper lip on either side of the midline. Occasionally, there are other lesions present in the mouth, such as on the tongue, and sometimes lesions occur elsewhere on the body, for example on the skin of the abdomen, legs and feet. The condition is called eosinophilic granuloma because the lesion is classically, though not always, infiltrated with eosinophils, but eosinophilia is not a common finding. The cause of the condition is unknown, although some people have suggested self-trauma from licking, or a low grade bacterial infection. Misalignment of the canine teeth has also been suggested in one or two cases but the condition is probably multifactorial. More recently poxvirus infection has also been associated with the presence of similar lesions, but it is not known if the infection was primary or secondary (Gaskell et al, 1983; Martland et al., 1983). It

has been suggested that eosinophilic granuloma is more common in adults, and females. The major differential diagnosis of the condition is squamous cell carcinoma, or more rarely, other tumours such as fibrosarcomas, and if there is any doubt then the lesion should be biopsied.

Conventional treatment of this condition has included the topical application of antibiotics, corticosteroids, and chemical cauterising agents, such as 2-5% silver nitrate solution, but the results of such topical treatments are usually disappointing. Greater success has been achieved by injecting a long-acting corticosteroid directly into the lesion but these can be difficult to administer. Systemic corticosteroids are probably the best first line of treatment. Quite high doses are needed, and owners should be prepared for possible recurrence, but a 75% success rate has been achieved with systemic corticosteroids used at a dosage rate of 1mg/kg prednisolone orally twice daily for 1 to 2 weeks, followed by a gradual tapering of the dosage over the next two weeks. Oral progestagens (e.g. 2.5mg megoestrol acetate twice weekly) have also been found to be useful. Antibiotic cover, for example with lincomycin, should also be provided. Other people have succcessfully used surgical excision and radiotherapy, but the best second line of defence after steroid therapy is probably cryosurgery.

Moving on to oral neoplasia - malignant tumours of the mouth are relatively common in the cat, and although they rarely metastasize to other parts of the body, they usually rapidly recur after attempted surgical removal. The most common tumours are squamous cell carnicomas, and much more rarely other tumours such as fibrosarcomas; they tend to occur in older cats (average age 10 years); and they most commonly occur on the ventral surface of the base of the tongue, the palate, and the gums. Tonsillar squamous cell carcinoma, although it has been reported from some geographical areas in cats, is not nearly as common as in the dog. Squamous cell carcinomas are often proliferative and ulcerated and should always be differentiated by biopsy from chronic inflammatory conditions and perhaps the occasional granulomatous lesion caused by actinomycosis. Gingival squamous cell carcinomas also tend to invade the underlying bone. Benign oral tumours such as periodontal fibrous epulis are rare in the cat; occasionally fibromas occur on the lips and face and these can be removed successfully surgically.

The third aspect of chronic feline stomatitis is dental disease. Dental caries, as occurs in humans, does not seem to occur in the cat. The cat does however sometimes show cavitation of the neck of some teeth which progressively undermines the crown and can lead to tooth loss. These lesions are hard and, unlike true caries, a dental probe does not stick. They seem to be caused by excessive osteoclast activity. The classic progression to dental disease in the cat however starts with calculus formation on the surface of the tooth. Calculus tends to occur in older cats, particularly those fed soft food. It can occur on any tooth but is commonest on those closest to the major salivary ducts (eg. lat. aspects of premolars and molars; lingual aspect of incisors). Calculus = mineral (which is mainly calcium hydroxyapatite) + bacterial plaque (bacteria + salivary protein), and it tends to form on the surfaces of the teeth close to the gingival sulcus. The adjacent gum then becomes inflamed, and ulcerated, and eventually recedes, so predisposing the tooth to infection and periodontitis. There may also be periodontal pocket formation. Treatment obviously consists of removing the calculus, but if there is

marked gingivitis or periodontis then the cat should also be treated with antibiotics, preferably starting several days before calculus removal to reduce the risk of septicaemia. In severe cases, affected teeth should be removed. Calculus does tend to reform quite quickly, however, probably because in removing the tartar, micro-abrasions are made on the enamel surface of the tooth. There is therefore little that can be done to prevent the disease, but the feeding of dry cat food is probably helpful.

Finally I would like to consider the most intractable group of all, that is "non-specific stomatitis and gingivitis." In young cats, a simple hyperaemia rather than gingivitis may occur at the margin of the gum, but it is usually an owner complaint rather than a cat complaint and it usually resolves satisfactorily without treatment. However, more important is the syndrome of chronic inflammation and ulceration, which occurs in all ages of cats, often in the mucosa of the fauces at the angle of the jaw, sometimes on the hard palate, on the gums, and occasionally on the tongue. It is not known if the more unusual condition of hyperplasia of the mucosa of the angle of the jaw, which often encroaches around the base of the molars, is the same syndrome or a separate entity. Probably it is a sequel to the chronic inflammatory and ulcerative lesions where the gingival tissue tends to proliferate in an attempt at healing. Unfortunately the hyperplastic tissue is rather friable, and thus is easily traumatized further during mastication.

The aetiology of these chronic inflammatory conditions is unknown - bacterial infection with anaerobes such as Bacteroides or Fusobacteria, or with a variety of aerobes has been suggested as a possible cause. In occasional cases, fungi such as Candida spp. may be involved, particularly if the cat has been on prolonged antibiotic therapy. Certain viruses have also been implicated in chronic stomatitis, particularly feline calicivirus. The role of this virus in acute stomatitis is well established, but there is limited circumstantial evidence that it may be involved in the aetiology of more persistent problems as well. Recently a small series of cases was reported from Australia where a significantly higher isolation rate of calicivirus was obtained from cases of stomatitis, compared to matched controls (Thompson et al., 1984). However, this possibility needs to be examined further, since the association of this persistent infection with cases of stomatitis may simply represent a general immune deficit on the part of the host, and not cause and effect.

Another facet to consider with respect to the aetiology of chronic stomatitis is the possibility that it may be immune-mediated in some way. There are three aspects to this. The first is the association of persistent feline leukaemia virus infection with some cases of chronic stomatitis, presumably because of the accompanying immunosuppression. The second aspect comes from a report by Johnessee and Hurvitz (1983) who noted in a series of cases that the lesions were predominantly infiltrated with plasma cells, and all cats had a polyclonal gammopathy. These authors therefore suggested that the cats may be predisposed to over-respond immunologically to otherwise normal bacterial flora. The third way in which chronic stomatitis may be immune-mediated is by means of the pemphigus complex, more specifically pemphigus vulgaris, where auto-antibodies form to intercellular cement substance and to the cell wall of the epidermis, leading to lesions at mucocutaneous junctions and also on mucosal surfaces. The diagnosis of this condition is by a specific

immunofluorescent test, but its relative importance in the feline stomatitis complex is not as yet known.

Finally, diet should be considered in the aetiology of chronic stomatitis in the cat. Soft foods can of course predispose to the condition, particularly by encouraging tartar formation. It is also worth noting however that diets with excess Vitamin A (e.g. high in liver) can lead to gum hyperaemia and tooth changes. However it is probable that other skeletal abnormalities would be noted associated with this hypervitaminosis A,, before the mouth lesions.

Treatment of these persistent inflammatory conditions is, unfortunately, often unrewarding: a number of treatments has been suggested - for example, specific antibody therapy based on bacterial culture and sensitivity tests - and if spirochaetes and Fusiformis are implicated then metronidazole therapy may be indicated, though it has been suggested that the curative effect of this may be non-specific in its action. Corticosteroids may be useful, particularly a long-acting corticosteroid injected directly into the lesion but although temporary remission may follow, there is often little long-term success with such treatment and the prognosis is not good. Another suggestion has been the use of intramuscular corticosteroids followed by oral progestagens, or oral progestagens alone (Wildgoose, 1979). Other measures that have been suggested include antiseptic mouth washes, (eg. 0.2% chlorhexidine, widely used in human gingivitis), chemical cautery (e.g. 2-5% silver nitrate solution) to stimulate epithelial regeneration, and vitamins A, B and C. Sloppy and aromatic foods are helpful in encouraging the cat to eat although it should be remembered that such foods encourage tartar formation in the long run. Teeth should be checked for tartar formation and sometimes where the inflammation appears to be associated with particular teeth, it does seem that the removal of some or all of the teeth may be helpful even though no dental disease may be evident. However this is a somewhat extreme measure, and difficult to achieve in cats whose teeth are otherwise healthy, and by no means all cases will respond to what is after all pretty radical therapy. Finally, cryosurgery has recently been advocated quite strongly by some as a possible treatment for chronic stomatitis, particularly for ulcerated or hyperplastic lesions in the fauces. It does seem that with care, it can be successful, but the mechanism by which it works is not understood.

References

Gaskell, R.M., Gaskell, C.J., Evans, R.J., Dennis, P.E., Bennett, A.M., Udall, N.D., Voyle, C. and Hill, T.J. (1983). Natural and experimental poxvirus infection in the domestic cat. Vet.Rec. 112, 164-170.

Johnessee, J.S. and Hurvitz, A.I. (1983). Feline plasma cell gingivitis-pharyngitis. J.Amer.Anim.Hosp.Ass. 19, 179-181.

Johnson, R.P. and Povey, R.C. (1982). Effect of diet on oral lesions of feline calicivirus infection. Vet.Rec. 100, 106-107.

Lane, J.G. (1977). Pharyngostomy intubation of the dog and cat. In: Grunsell C.S.G. and Hill F.W.G. (ed) The Veterinary Annual, 17th Issue, Bristol, Wright-Scientechnica, p. 164.

Martland, M.F., Fowler, S., Poulton, G.J. and Baxby, D. (1983). Pox virus infection of a domestic cat. Vet.Rec. 112, 171-172.

Pederson, N.C., Laliberté, L. and Ekman, S. (1983). A transient febrile limping syndrome of kittens caused by two different strains of feline calicivirus. Feline Pract. 13(1), 26-36.

Studdert, M.J., Martin, M.C. and Peterson, J.E. (1970). Viral diseases of the respiratory tract of cats: isolation and properties of viruses tentatively classifed as picornaviruses. Am.J.Vet.Res. 31, 1723.

Thompson, R.R., Wilcox, G.E., Clark, W.T. and Jansen, K.L. (1984). Association of calicivirus infection with chronic gingivitis and pharyngitis in cats. J.Small.Anim.Pract. 25, 207.

Wildgoose, W.H. (1979). Treatment of gingivitis in cats. Vet.Rec. 104, 86.

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Gaskell, R.M. and Gruffyd-Jones, T.J. (1977). Intractable feline stomatitis. In The Veterinary Annual, 17th Issue, Eds. C.S.G. Grunsell and F.W.G. Hill, Wright-Scientechnica, Bristol, p.195-199.