Feline Oral Cavity Diseases

Part 1: Tooth Resorption Lesions

Introduction

Over the last thirty years, the veterinary world has become increasingly aware of the phenomenon of the dental resorative lesion in cats. Nomenclature varies but the term in common use now is Tooth Resorption lesions (TR). The species can be added as a prefix. The phenomenon may well be cyclical from the initial reports in the 1900’s to the present day. Most recent surveys could well indicate a reduction in the incidence level compared with the last three decades. (Wiggs 2003)

These lesions are seen in other species, specifically dogs, pigs, humans, rats, mice and marmosets to date (Du Pont 2002). However, the incidence in the feline has become, and remains, relatively high. The earliest contemporary reference to them in veterinary literature is often quoted as 1976 (Schenk 1976). However a paper from 1955 (Tholen 1987) quoted “erosions below the crown of the tooth”. The oldest known reference is by Hopewell-Smith in the Dental Cosmos journal of the University of Pennsylvania that describes them as long ago as 1930 (Hopewell-Smith 1930).

Surveys of cat teeth from the 1980’s onwards indicated initially an alarming rise in the incidence of this problem before the reduction in incidence reported since 2000 or so. Given the need for radiology for proper diagnosis, older surveys not using radiographs will mean under-diagnosing lesions.

In the surveys listed below approximately, 29% of cats within the general feline population and 54% of cats with demonstrable dental disease were shown to have at least one TR lesion. One study of 109 healthy cats indicated an incidence of 38% in mixed breed cats and 70% in purebred cats (Girard N 2008)

Under reporting of TR lesions is almost certainly a problem in these surveys given that many fragmented or missing teeth were lost due to resorptive lesions and that 81% of cats with one or more retained roots were also found to have a clinically evident resorptive lesion (Du Pont 2002). One study attempted a comparison between domestic, feral and captive exotic cats (Levin 1996). Statistical analysis is currently only available for one group (feral cats) but the indication is that the same disease process takes place with a much lower incidence than in the domestic feline population (9.5% of those younger than six years showing at least one affected tooth and 17% of those older than six years).

The location of TR lesions is approximately 90% on the labial/buccal aspect of the teeth. The most commonly affected teeth being the upper fourth premolar, lower first molar and lower third premolar.

Pathophysiology

Histologically, TR’s in cats are initially half moon concavities in the structure of the tooth starting on the cementum surface. (Burke 2000; Dubelzig 1990) The destruction of the dental tissues is associated with cellular digestion of the dental tissues by multi-nucleated giant cells analogous to osteoclasts (odontoclasts).
Stem cells are attracted to the periodontal ligament area by inflammatory cytokines and are transformed into clast type cells. These clast cells then erode the mineralised tissues such as cementum, dentine and enamel. The lesions are lined by these cells and are non-carious. The dental margins are fully mineralised, hard and scalloped to support this fact. Histopathologic examination (Gorrel 2002) shows that TR’s in cats are essentially an inflammatory disease of the periodontium that extends to the surrounding tissues. The inflammation affects large parts of the surface of the tooth and is characterised by both resorptive and reparative phases.

**Incidence**

The incidence of these lesions in cats has been documented in a number of surveys over the past few years (see further reading list). The populations defined in these surveys are both the general feline population and the feline populations with dentistry bias (i.e. those individuals who were presented for dental or oral cavity problems). Surveys on this subject have become more numerous and sophisticated over the years – mainly by the inclusion of radiographic diagnosis as a routine. The main surveys from 1982 to 2001 are as follows;

**General Population**  (average 29%)

Schlup (1982); Switzerland 28.5% of cats had one or more lesions. (n = 200)

Zetner (1987); Austria 28% of cats had one or more lesions. (n =?)

Coles (1989); Australia 52% of cats had one or more lesions. (n = 64) (2)

Mulligan (1989): California, USA 20% of cats had one or more lesions. (3) (n =?)

Harvey (1992): USA 26% of cats had one or more lesions. (n = 794) (4)

Zetner (1992): Austria 23% of cats had one or more lesions (n = 500) (5)

Wiggs (1993): Texas, USA 28% of cats had one or more lesions (n=214)

Okuda et al (1994): Japan 22.5% of cats had one or more lesions (n = 138)

Clarke (1994): Australia 29% of cats had one or more lesions (n= 168)

Verhaert (2000). Belgium. 25% of cats ha at least 1 lesion (n=753)

Ingham et al (2001): UK 29% of cats had one or more lesions (n=228)

Girard et al (2008): France. 38% (mixed breed cats) and 70% (pedigree cats) n=109

**Population with dentistry bias**  (average 54%)

Tholen (1987): New York, USA 65% of cats displayed at least one lesion. (n = 465) (6)

Zetner (1990): Austria 46% of cats with chronic oral disease & one lesion or more. (n=24)

Crossley (1991): UK 57% of cats displayed at least one lesion (n = 152). (7)

Remeeus (1991): Holland 43% of cats displayed more than one lesion. (n = 308)

Harvey (1992): USA 67% of cats displayed more than one lesion. (n = 78)
Van Wessum (1992): Holland 62% of cats displayed more than 1 lesion (n = 432)

Wiggs (1993): Texas, USA 42% of cats displayed more than one lesion (n=252)

Okuda et al (1994): Japan 48.1% of cats displayed more than 1 lesion (n = 81)

Harvey et al (2000): USA. 43% of cats with TR’s – mean number affected teeth 4.4 (n=162)

Lommer et al (2001): USA. 67% of cats displayed an ORL. Radiographs used. (n=147)

**AETIOLOGY.**

Research indicates that this is a complex disease with several causative factors interacting. Although diet is included in this list, many other factors may be responsible.

Work by Gorrel and others indicates that development of TR on the cement root surfaces does NOT require inflammation (Gorrel 2002). All cats appear to develop cemental surface resorption in life and some cats heal with restoration of normal periodontal attachment whereas in others, the lesions deepen to affect dentine and go onto develop destructive ankylosis. Any factor that creates abnormal formation or results in a deficiency of dentine or cementum mineralisation could precipitate the development of resorptive lesions.

The quality of cementum may vary because of differing hormonal and dietary factors predisposing some cats to development of clinical lesions. Excessive vitamin D intake over many years has been considered as a significant factor (Reiter 2004) but this was contradicted by another study (Girard 2008).

Over the years, papers have occasionally considered the effects of occlusal stresses and/or periodontal disease as aetiological factors via external inflammatory or non-inflammatory processes (Burke T, Johnston NW 2000. Harvey 1992, Girard 2008).

One current direction of research is focusing on treatment using the bisphosphonate group of drugs (Alendronate™: Merck) that bind to hydroxyapatite in bone and root surfaces and inhibit the clastic activity that causes apoptosis (programmed cell death). In the proof of concept study (Mohn 2009) it was used at 9mg/kg twice weekly and appeared to slow or arrest the progression of lesions. This is based on the assumption that the periodontal ligament is an area of high uptake of factors that mediate clastic activity – otherwise why does “rubber jaw” not become “rubber leg”? (Harvey 2004)

**DIAGNOSIS**

Clinically these lesions are intensely painful (when on the crown and open to the oral cavity) and are often associated with irritative, gnawing movements of the jaws during mastication. In an anaesthetised animal, it is common to produce a reaction when a lesion is found accidentally during routine probing of the sulcus. Clearly, the pain from these lesions must debilitate the cat but, as with many painful dental conditions in animals, the host seems to “live” with problem without overt external stimuli. Often the only way to determine how much of a burden the pain was for the animal is to note the difference in demeanour before and after treatment.

There are three main methods for diagnosing the presence of TR’s in cats.
1) **VISUAL**
- Red spot zone of inflamed gingiva over lesion.

2) **TACTILE.**
- Using sharp explorer TR's look in the sulcus area
- Due to the vascularity of the lesion, this may cause pronounced bleeding.

3) **RADIOLOGY.**
- The definitive method and recommended mode of diagnosis
- Should be used if either of the above two methods gives rise to suspicion.
- Best performed with a dental x-ray machine due to the small size of mouth & film
- Areas of lysis of tooth tissues are known to be rapidly progressive.
- Beware the phenomenon known as “cervical burn-out” where excessive exposure can give rise to confusion at the tooth neck.
- Sentinel teeth (307/407). One study (Heaton 2004) indicted that if these teeth are missing or affected it is a 93% predictor of TR lesions elsewhere in the mouth.

**TYPING AND STAGING**

The definitive guide (2009) to this can be found on the American Veterinary Dental College website – [www.avdc.org](http://www.avdc.org)  [http://www.avdc.org/?q=node/29#resorption](http://www.avdc.org/?q=node/29#resorption)

Classification of lesion is based on severity (stage) and location (type).

Some lesions result in total root resorption whilst others degenerate less drastically retaining key organic element of the tooth - such as the periodontal ligament and pulp. If organic tissue is retained they need treated differently from those which do not retain these tissues. Therefore, to enable logical treatment planning they must first be staged and typed.

Lesions are staged 1-5 depending on severity of the actual lesion.

**Stage 1 (TR 1):** Mild dental hard tissue loss (cementum or cementum and enamel).
**Stage 2 (TR 2):** Moderate dental hard tissue loss (cementum or cementum and enamel with loss of dentin that does not extend to the pulp cavity). The tooth becomes painful if the sensitive dentine tubules are exposed to air.

**Stage 3 (TR 3):** Deep dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth retains its integrity. Stage 3 lesions are very painful if exposed to air and bleeding from pulp tissue will be evident on probing. Early “ghost images” of roots may be evident now on radiographs.
**Stage 4 (TR 4):** Extensive dental hard tissue loss (cementum or cementum and enamel with loss of dentin that extends to the pulp cavity); most of the tooth has lost its integrity.

*TR4a Crown and root are equally affected;*

*TR4b Crown is more severely affected than the root;*
**TR4c Root is more severely affected than the crown.**

**Stage 5 (TR 5):** Remnants of dental hard tissue are visible only as irregular radiopacities, and gingival covering is complete. The oral mucosa may well have healed over the tooth fragments and may or may not be sensitive.
Typing of Lesions

Typing of Lesions is dependent on their location as seen on a radiograph. They can present in three ways:

**TYPE 1**

On a radiograph of a tooth with type 1 (T1) appearance, a focal or multifocal radiolucency is present in the tooth with otherwise normal radiopacity and normal periodontal ligament space. *(Du Pont 2002)*

Concurrent periodontal disease is a common feature of type 1 lesions. *One study of 109 healthy cats indicated that 40% of the total lesions found were Type 1* *(Girard 2008).*

**TYPE 2**

On a radiograph of a tooth with type 2 (T2) appearance, there is narrowing or disappearance of the periodontal ligament space in at least some areas and decreased radiopacity of part of the tooth.

Type 2 lesions have extensive root replacement by alveolar bone. Progression will result in loss of periodontal ligament and pulp. *One study of 109 healthy cats indicated that 60% of the total lesions found were Type 2* *(Girard 2008).*
TYPE 3

On a radiograph of a tooth with type 3 (T3) appearance, features of both type 1 and type 2 are present in the same tooth. A tooth with this appearance has areas of normal and narrow or lost periodontal ligament space, and there is focal or multifocal radiolucency in the tooth and decreased radiopacity in other areas of the tooth.

Summary of Staging and Typing

Correct staging and typing provides diagnosis, prognosis and treatment planning for that individual tooth. Bear in mind that a number of teeth may be affected and will be at different stages of progression. Some teeth may already be lost. Treatment planning, therefore, includes not only a strategy for the current period but some form of monitoring for the future also.

Abbreviations: A tooth with a Stage 4b lesion that has a type 2 radiographic appearance would be abbreviated TR-S4b-T2

Treatment of TR Lesions

STAGE 1:

Thorough dental scaling and polishing of the teeth with non-fluoride flour grade pumice. The affected tooth is lightly air-dried and treated with an unfilled resin dentine bonding agent (e.g. Bond One: Jeneric Pentron) to seal off the dentine tubules. Fluoride material such as Duraphat (Colgate) can also be used subsequently. It should be noted that the use of fluorides in this manner is mainly anecdotal and requires extreme care in small body weight animals. However, their action of hardening of enamel, desensitising exposed dentine and limiting the influence of plaque bacteria is likely to help patient comfort in the immediate post-op period. The teeth should be charted, radiographed and re-examined by radiography at an interval of not more than three months. The client is obviously made aware of the lesion and the need to introduce homecare methods to reduce the level of plaque on the crowns. Success rates of restoration of stage 1 lesions are considerably higher than for other stages (Wiggs 2003).
STAGE 2, 3 and 4:

Stage 2 lesions and beyond are sensitive and most likely painful. They are treated by extraction (see below). The extraction technique used will be dependent on type as seen on radiograph. A

- **Teeth with retained organic tissues (periodontal ligament and/or pulp – mainly Type 1) need to be extracted in a conventional manner via a mucogingival flap and buccal plate ostectomy before splitting into component roots. As these teeth are more fragile than “normal” teeth, more care is needed.**

- **Teeth with no organic material present (mainly Type 2) can be treated less invasively by crown amputation via a small gingival envelope flap and crestal alveoloplasty following extraction to eliminate any sharp bony margins.**

- **Treatment for Type 1 and Type 2 is not interchangeable. The less invasive technique for Type 2 is not suited to Type 1q and vice versa.**

STAGE 5 - The stage 5 lesion consists of retained roots with no crowns. In many cases, these will be felt as hard swellings under healed oral mucus membranes. If there is no pathology of soft tissue over the swelling, no visible apical pathology on x-ray and no current sensitivity, no treatment is necessary. However, if soft tissue inflammation is present or if the cat feels pain or sensitivity in this area, exploration and curettage of the area may be desirable.

**Extraction Techniques**

1. **Conventional Extraction Technique (Type 1 lesions)**

Type 1 lesions are frequently linked with periodontal disease and a conventional extraction technique should be performed with the aim of removing all of the tooth tissue and diseased bone. It is not advisable to use a root retention technique with these teeth.

A mucogingival flap with vertical releasing incisions will provide superior visualisation of tooth and the buccal bone plate. The vertical releasing incisions should be made slightly off the target root(s) using a #11-scalpel blade along the long axis of the mandible/maxilla and perpendicular to the long axis of the tooth. Make a gingival flap on both sides of the tooth use a sharp periosteotome or periosteal elevator (Molt P2/4) to create good access to the target area.

Using a small round bur (#1) the buccal bone can be carefully removed to show the surface of the roots and the furcation point where they meet. Splitting the tooth into component roots can now take place. Further limited widening of the periodontal space can take place with the small bur before careful luxation of the roots with small luxators designed for this task. **See box below for recommended cat luxators.**

Once roots are removed the bone surface is smoothed with a rasp or rongeurs and flushed with sterile saline (10ml syringe/21g needle) before closure of flap with 5/0 Monocryl (W 3203).

**Atomisation of cat teeth with a high-speed bur is not acceptable.** This method of destroying the root does not give sufficient control and, in many cases, tissues beyond the root may be destroyed. Given that there is a sensory nerve beyond almost all teeth – infraorbital for maxilla or inferior alveolar nerve for the mandible; – it makes little sense to use this method. In normal circumstances, there is no substitute for careful elevation and removal of the whole root.
2. Crown Amputation Technique (Type 2 lesions)

In the case of Type 2 lesions only, a technique of crown amputation is permissible ([Du Pont 2002a: Du Pont 2002]).

A limited envelope flap reflecting gingiva from the tooth surface using a periosteotome may be sufficient. If not, releasing incisions may occasionally need to be made if an envelope flap provides insufficient visualisation.

The crown is removed with a small (#1) round bur perpendicular to the long axis of the crown to remove all tooth structure just apical (below) the level of the alveolar bone crest.

A fine round diamond or bone rasp should be used to create a smooth surface of bone before saline flushing and closure with single interrupted suture(s) of 5/0 Monocryl.

---

**Dental Luxators for Cats**

- Feline Luxator – EX5  (2mm)
- Feline Luxator – EX5S (2mm serrated)
- Feline Luxator – EX5H (2mm notched)
- Feline Luxator – 100C  (2mm gentle back curved)
- Feline Root Tip Pick – WA1

Source: [www.drshipp.com](http://www.drshipp.com) (current cost 2010: $37.95 each)

---

**SUMMARY**

- The problem of feline tooth resorptive lesions can be demonstrated to affect at least 29% of the general cat population and at least 54% of those cats presented to veterinarians for oral cavity disease.

- The lower premolar 2’s (307/407) act as sentinel teeth and provide a 93% predictor for lesions in other teeth.

- Treatment of TR’s requires staging and typing by radiography to select the correct method.

- Bisphosphonate treatment (Alendronate: Merck) show promise to prevent progression of existing lesions and retard the formation of new lesions.
REFERENCES


HOPEWELL-SMITH, A. (1930) the process of osteolysis and odontolysis, or so-called absorption of calcified tissues: A new and original investigation. The Dental Cosmos 1xxii, 1036-1048


RIETER AM: (2004). Further evidence for a possible role of vitamin D in the aetiology of feline Odontoclastic Resorptive Lesions (FORL).


Part 2: Feline Chronic Gingivo-Stomatitis (FCGS)

Introduction

The condition currently, and most commonly, known as *Feline Chronic Gingivo-Stomatitis (FCGS)* is a relatively common and frustrating problem to the small animal practitioner. A number of synonyms are found in the literature for the same conditions. Examples are “Feline Lymphocytic Plasmacytic Stomatitis”, Plasma Cell Stomatitis”. The reported incidence varies with severity but a figure of 3% of all feline dental conditions for the most intractable cases may be considered reasonable. Many cases prove to be extremely frustrating with a number of different combination treatments in current use.

The syndrome is characterised by persistent and severe inflammation and ulceration of the oral soft tissues. Many times this includes the pharyngeal and lingual mucosa.

The two most common sites are:

- **Tissues lateral to the palatoglossal folds (palatoglossitis)**
- **Tissues (gingiva and mucosa) overlying the cheek teeth (buccostomatitis).**

The condition is often present in the absence of significant accumulation of calculus on the teeth. Inflammation in these areas occurs commonly around the time of kitten vaccination or when temporary teeth eruption, when permanent teeth erupt or, most commonly, much later in life.

Purebred cats have long been considered to be more susceptible. There is also an inverse relationship between the age of onset of disease and the number of cats in the household and this may implicate social stress or increased exposure to infectious agents as predisposing factors.

This syndrome is best considered as part of a full oral cavity examination and the presence of Tooth Resorption (TR’s) lesions frequently adds to and confuses the picture. Additionally, it is clear that carriage of calici virus is a co-factor in the induction or progression of the complex. Although the relationship between calici infection and FCGS appears strong, there is also a reported incidence of 50% of cats infected with FIV also having FCGS. Another source indicates that around 15% of cats with FCGS are positive for FeLV/FIV. *(Knowles 1989)*

One consistent feature of all cases is a hypergammaglobulinaemia. This implies B lymphocyte proliferation and therefore *no humoral immune response depression*. It is probable that *affected cats are intolerant to even small quantities of bacterial plaque* on the tooth surface and elsewhere in the mouth. The main problem is that not all FCGS cases are alike. Some respond to routine periodontal therapy and improved hygiene while others will respond poorly to any treatment. The implication is that some cats have a very low threshold to the trigger factors(s) whilst others have a higher threshold approaching the level for normal cats. *Most intractable cases (87%) improve with elective tooth extraction and a few cases (13%) do not respond to any treatment.* *(Hennet 1997, Girard & Hennet 200, Hennet 2010)*
Clinical Signs

The main sign in all cats is dysphagia and pain due to extensive oral inflammation and ulceration of soft tissues. Inflammatory lesions can be focal or diffuse and may involve all oral tissues - most commonly the tissues lateral to the palatoglossal folds, gingiva and mucosa overlying the cheek teeth. Other tissues in the pharynx, tongue and the mandibular molar salivary glands are also affected in severe cases.

Other reported signs are:

- **Severe halitosis.**
- **Weight loss - chronic or acute**
- **Lack of (or an inability) to groom.**
- **A reluctance to eat hard food is common.**
- **Submandibular lymphadenomegaly – often dramatically increased in size and painful when palpated.**
- **Variable, sometimes minimal, accumulation of plaque and calculus.**
- **Teeth may be missing, affected by “tooth resorption” or suffering from furcation exposure and excessive mobility after recession of the periodontal tissues.**

Aetiology

There is no simple aetiological agent for this syndrome. Certain factors are known to have an effect but the most commonly held view is that these cats suffer from an immunological over-reaction to low levels of oral antigens – dental plaque mainly. Factors involved are:

- **Breed:** Some breeds may appear to have more affected individuals. Purebred cats are anecdotally more often affected with Siamese, Burmese, Abyssinian, Persians, Tonkinese, and Main Coons all over represented.

- **Environmental Factors:** Colony cats or those in multi-cat households appear to be more commonly affected. **Stress** is considered the main factor with also the close proximity of animals allowing transmission of microorganisms also being significant.

- **Plaque bacteria:** The oral bacteria present in the plaque matrix drive the abnormal non-specific inflammatory response. Although individuals are thought to be plaque intolerant, there is a variable threshold to the bacterial load among these individuals. Specific bacteria as seen in periodontal disease have been reported in these cats and pure cultures of *Pasteurella multocida* are common in the authors’ experience.

- **Feline Calici Virus:** Many clinicians, including the author, find a level approaching 100% of chronically affected individuals (> 6months) showing positive testing to virus isolation following oropharyngeal swabbing for Feline Calici Virus. The significance of this within the syndrome is not known. It is possible that the virus damages cell membranes allowing easier antigenic penetration by other agents. However, other co-factors are required before this virus can cause disease as FCV carriage in the cat population is around 30% (*Zicola 2009*). One research study (*Hennet & Boucrault-Baralon 2005*) considered that chronic palatoglossitis lesions, as opposed to buccostomatitis lesions, to be calicivirus associated. FIV particularly may have a role in producing oral lesions by predisposing the cat to secondary infections. Both FIV and FeLV may contribute to an aberrant immune response to oral antigens. However, this is not the hyperimmune response that characterises the main syndrome.
**Dental Disease:** The presence of any concurrent dental disease is important. Either periodontal disease or Tooth Resorption lesions (TR’s) or both can have an exacerbating effect on the syndrome.

**Diagnostic Testing**

A standard diagnostic approach is advocated for all cats affected. This should comprise:

- **Virus testing:** for FIV, FeLV. Oral swab for FCV and FHV

- **Bacteriology:** for both aerobic and anaerobic bacteria. A high proportion of cats tested show pure cultures of Pasteurella multocida (*Dolieslager 2010*).

- **Routine Haematology and Biochemistry** screening for underlying systemic disease. One study (*Hennet 1997*) reported 10% of affected cats with chronic renal failure. Any underlying systemic disease may significantly affect the prognosis or the safety of anaesthetic protocols and other drugs (e.g. long term NSAID’s)

- **Biopsy** of affected areas - necessary to eliminate neoplasms (e.g. Sq. Cell Carcinoma, Lymphoma etc) and other immunopathologies. Very important if lesions not symmetrical.

- **Dental chart and full mouth dental radiographic survey** to assess periodontal status of teeth, bone quality and locate broken root tips or tooth resorption lesions.

**Treatment**

The main aim is zero tolerance of both existing dental disease and of bacterial plaque. There are three underlying principles underpin treatment with the aim to *reduce the oral antigen burden.*

- **Control plaque**

- **Control existing dental disease – periodontal disease and tooth resorptive lesions mainly**

- **Control inflammation**

All affected individuals should be treated in the same manner. The ultimate aim is to improve the overall hygiene of the oral cavity and reduce the antigen burden by, (initially) a thorough dental scaling, periodontal debridement and polishing followed by aggressive home care with 0.12% chlorhexidine gluconate gel twice daily. (Parodongyl™: Virbac).

**Base-line Treatment - all cases**

**Antibiotics** - often necessary pre-operatively to control excessive inflammation and improve quality of soft tissue before and after surgery. The following drugs have been described for use.

*Clindamycin (Antirobe: Pfizer) at 11mg to 22mg/kg sid,*

*Ampoxycillin/Clavulanic Acid at 12.5mg/kg bid*

Use pre-op as required to improve tissues and post-op for a minimum 8-10 days. Ensure owner can comply with treatment. This may mean using an antibiotic in an acceptable form. Powder can be hidden in frozen butterballs or in pilchards with tomato sauce.
Dentistry

- **Dental chart**: a dental chart is an essential record of the mouth. Discourage shortcutting of exam.

- **Scale & polish**: every case starts by improving basic hygiene.

- **Treat diseased teeth**: in almost all cases, this means extraction.

- **Initiate chlorhexidine**: twice daily application of chlorhexidine (Parodongyl: Virbac) wiped inside lips twice daily or brushed if cat will allow it.

**Additional Treatment - Selected Cases**

**Gingivectomy**

Necessary in cases where hyperplastic gingiva has created a pseudo-pocket deeper than the normal sulcus depth of 0.5-1.0 mm. Use a #11 scalpel and a bevelled cut 120° to the long axis of the tooth or an electrosurgery machine on cut/coagulation mode or a high-speed tissue bur.

These methods require extreme care not to damage tooth surface. It is necessary to “scallop” the gingiva at the inter-dental area. Remember that up to 1mm may slough at the margin of an electrosurgery incision.

**Repeat Scale / Polish**

For juvenile patients it is important to avoid permanent anatomic changes in the first two years of life. If the immune system is substandard in the early months, the provision of excellent hygiene can help considerably. Although little calculus may be visible, continued inflammatory changes in either the whole of the gingiva or the marginal gingiva is an indication to repeat the surgical cleaning - especially the hand curettage subgingivally. This may mean surgery every three months.

**Elective Tooth Extraction**

This is now firmly established; by both peer reviewed publication and dental specialists, as the logical option to take if base line treatment (see above) alone is insufficient to provide resolution of the inflammation. In the author's opinion, if the tissues fail to respond to the best hygiene you can provide within 2-4 weeks, by reduction of inflammation and improvement in comfort, elective surgical extraction of all the cheek teeth should follow without delay. Owners and many veterinary surgeons are often reluctant to take this step. The prognosis for this procedure can broadly be estimated that 50% of all cases will resolve without further need for treatment, 37% will improve but will require less medication than before but varying degrees of continuing anti-inflammatory treatment and 13% will not improve (Girard, 2005).

Elective surgical extraction of whole cheek teeth quadrants should not be undertaken lightly as there are several complications that may associated with it.

1) **The underlying bone may be sclerotic and poorly vascularised.**
2) **The roots may be ankylosed to the alveolar bone**
3) **Teeth affected by Tooth Resorption lesions (type 2) may have roots in an advanced state of destruction with no true morphology. For type 1 TR lesions the teeth may be fragile and hard to extract without flaps.**
See pp10-11 above for technique guide.

**Post-Op management of elective extractions**

Multiple extractions require consideration as to analgesics, antibiotics and nutrition post-op. Some cats may be best hospitalised for 2-3 days if owners are unwilling or unable to administer medication per os.

**Analgesia:** Buprenorphine (Vetgesic™; Alstoe) is considered good for moderate to severe pain in cats at 1ml per 15kg every 6 hours. *This can be used either parenterally or per os/sublingual.* Owners can administer this analgesic very easily. An alternative regime in the hospital would be to use a selective μ-agonist opioid such as morphine. Morphine is very useful for severe pain at 0.1mg to 0.2mg/kg im or sc. every 6-8hrs. Carprofen or meloxicam is useful n addition to, but not instead of, opiates.

**Antibiotics:** Although the primary condition is a hyperimmune reaction to mixed oral antigen, antibiotics by themselves give minimal success. In the perioperative period, they will guard against opportunist infection and should be started pre-operatively. The selected drug should have good activity in bone and on anaerobic bacteria. Clindamycin and/or potentiated Amoxicillin are the drugs of choice.

**Feeding:** Nutritional assistance may be necessary short or medium term. It may be necessary to consider pharyngostomy feeding in extreme cases and assisted oral feeding in hospital in others. If fluid intake is suboptimal, this should be addressed also. Most cats do better at home if the owner is able to provide active help. Soft foods (pilchards in tomato sauce) are necessary for three to five days post-op. In some circumstances it may be necessary to use a convalescence diet immediately post-op such as Hill’s a/d™, Waltham Feline Concentration Diet™ or Nutrigel™ (Virbac).

**Other Anti-inflammatory or Immunomodulation Therapies**

**Interferon**

A number of veterinary surgeons report using interferon and some studies are now appearing to indicate that it exceeds the potential of other treatments for this condition.

A consensus statement by a group of European specialists in late 2010 indicated feline recombinant interferon omega is most effectively used in the group of cats which are FCV positive and are long-term non-responders to extraction. Our own studies over three years indicate that, not only is interferon very helpful in reducing inflammation and improving comfort levels, it also allows practices to drop other treatment. *Results in a recent study of 39 cats indicate that feline recombinant interferon is an effective treatment particularly in the group of cats, which are FCV positive and are non-responders to elective extraction.* (Hennet 2010).

**Oral use:** Interferon given per os is believed to work by initiating a cytokine cascade when it comes into contact with cells to provide an immunomodulatory effect over a long period of time. The cascade then has distant effects.

A 10MU vial is initially injected into a 100ml bag of sterile saline and ten fractions of 10ml created, which are then frozen. When frozen they have a reported shelf life of one year. The first 10ml fraction is used to give a dose of 1ml per os per cat per day resulting in a daily dose of 100,000 units of interferon. This fraction can be refrigerated normally and will have a shelf life of three weeks.
The owner continues to give 1ml per day until all the fractions are used. Ideally, treatment lasts for 100 days but longer may be required. After three months, the progress should be reassessed using our Stomatitis Disease Activity Index (SDAI) scoring system. Cats can be rechecked for calici virus carriage in the oropharynx at this time.

**Initial intralesional use:** The consensus statement (October 2010) by a group of European specialists indicated *that intralesional treatment is not probably necessary to initiate therapy.* In some very severe cases an initial treatment total dose of 5 MU injected locally into multiple sites at the junction between healthy gum and a diseased tissue can provide an initial boost to a treatment course. Using a 10MU vial, enough saline or sterile water is drawn into the syringe to provide a reasonable volume for use - normally 1-2ml depending on area to be injected. The contents are administered in fractions of 0.1 - 0.2ml over the areas inflamed. For severe cases, we make five injections in each side of 0.2ml each. In less severe cases, we concentrate higher volumes into a smaller area.

Monitoring the cat's weight, along with a number of other indices as per our standard evaluation forms (SDAI), is a useful objective way of assessing response to treatment. We can supply you with an initial assessment form and one for ongoing evaluation.

Note that feline interferon omega should always be stored in the fridge and will remain viable once reconstituted for up to 21 days at 4°C. when used for low dose oral administration – not for injection.

**Subcutaneous injections:** This method of administration is described but appears to be less effective than submucosal administration for oral carriage of FCV and is not used by the author.

**Other treatments**

Many drug therapies are advocated for this condition and most have no proven efficacy. Some of these are based on case reports, anecdote or small-uncontrolled studies. Given that some of these drugs are highly toxic in cats, familiarity with the drugs is recommended. The internet also provides many owners with information which may or may not carry any reasonable validity.

**Anti-inflammatory therapy:**

**Corticosteroids:**

These drugs are used, by some practitioners, principally to control inflammation in refractive cases which have had elective cheek teeth extraction and are not sufficiently controlled by feline recombinant interferon. If their use is justified on welfare grounds, the overriding principle must always be to use the minimum effective dose rate. This means using a short acting molecule (prednisolone) at the lowest effective dose rate such as 5mg twice weekly or 2mg every other day tapering downwards.

**NSAIDs:**

The first choice option is meloxicam, to be prescribed with respect to the appropriate guidelines for use of long term NSAIDs in cats. Some new molecules such as robenacoxib may show promise.

**Azathioprine:**

There is insufficient data to recommend the use of azathioprine in the management of FCGS syndrome. The potential side effects are not insignificant and excessive use of generalised immunosuppressive options can be a problem long term.

**Chlorambucil:**

There is insufficient data to recommend the use of chlorambucil in the management of FCGS syndrome.
CO2 laser surgery:
There is insufficient data to recommend its routine use in the management of FCGS syndrome.

Cyclosporine (Atopica®, Novartis):
Some data has been published on this molecule as part of a dermatology study suggesting 4 out of 8 cats treated responded and could be maintained on every second day dosing. However, other studies have been equivocal about the benefits and a placebo-controlled trial in a small number of cats did not show a significant difference from placebo. Some suggestions for use have been provided but monitoring of blood levels to avoid toxicity is deemed essential due to erratic absorption differences. In general, the currently available data is not sufficient to support a recommendation to use this drug.

Doxycycline:
There are anecdotal reports of the use of long-term doxycycline at sub-antimicrobial doses as an anti-metalloprotease to aid healing of ulcerated tissues. This is a result of its use in this manner to manage recurrent oral aphthous ulceration in some human patients. However, there is currently no data to support the use of this option in cats.

Gold salts:
There are insufficient data to recommend their use. Additionally, the reported side effects, including oral inflammation and ulceration, are high.

Additional nutritional support:
It is necessary to ensure good quality nutritional support to encourage an effective immunological response and post-extraction healing process. Various diets and supplements have been suggested including vitamin preparations and omega-3 EFAs, but there is no option which has data to allow a recommendation for any specific product. The beneficial effects of a recovery food post surgery has been demonstrated in cats with FCGS syndrome. Additive free food has also been suggested but the results are anecdotal at best.

Vaccine:
A multi-site placebo controlled trial is taking place to evaluate the effects of their T-Cell Receptor (TCR) peptides for feline stomatitis. The TCR Peptides are immune modulating biologics, which have been shown to modulate T-helper cell function and Th1/Th2 cytokine profiles. T-helper cells appear to play a central role in feline stomatitis, causing inflammation and inappropriate immune reactions. Preliminary studies in cats have shown significant results in advanced, refractory feline stomatitis. Preliminary results were due to be reported in late 2009.

Summary
This is a poorly defined syndrome of unknown aetiology characterised by focal or diffuse chronic inflammatory response involving the gingiva, oral mucosa, and often the pharynx and tongue.

Commonly described clinical findings include elevated serum globulins and a submucosal infiltrate of plasma cells, lymphocytes, neutrophils, and macrophages. Potentially various viral agents and bacterial species are involved. There is no doubt that atypical hyperimmune responses are the basis of the problem. Multiple mechanisms will be acting concurrently.

Successful management of this complex requires a logical approach. The need for base-line data before treatment alters the host response cannot be over-stated. Once this data is available, a treatment plan and prognosis can be considered. The role of bacterial plaque is crucial whatever the state of the host immune response. Diligent professional scaling, polishing and subgingival debridement - zero tolerance to any dental disease - underpins any treatment in tandem with aggressive homecare by the owner. Cases failing to respond to simple plaque control should be considered for elective cheek teeth extraction and adjunctive
treatments at an early date. Those cases still non-responsive but FCV positive may be helped by interferon therapy.

It is important that the owner is involved at an early stage with discussions as to aetiology, treatment plans and help with homecare. A highly motivated owner is a strong ally in the provision of successful treatment.

**Selected references and further reading.**


Hennet P. Chronic gingivostomatitis in cats: Long-term follow-up of 30 cases treated by dental extractions *J Vet Dent* 1997; 14: 15-21


PART 3 - FELINE OROFACIAL PAIN SYNDROME

Introduction

Feline Orofacial Pain Syndrome (FOPS) is a pain disorder of cats with behavioural signs of oral discomfort and tongue mutilation. (Rusbridge et al 2010)

FOPS is suspected to be a neuropathic pain disorder and the predominance within the Burmese cat breed suggests an inherited disorder, possibly involving central and/or ganglion processing of sensory trigeminal information.

The disease is characterised by an episodic, typically unilateral, discomfort with pain-free intervals. The discomfort is triggered, in many cases, by mouth movements. The disease is often recurrent and with time may become unremitting – 12% of cases in this series were euthanased as a consequence of the condition.

Sensitisation of trigeminal nerve endings as a consequence of oral disease or tooth eruption appears to be an important factor in the aetiology - 63% of cases had a history of oral lesions and at least 16% experienced their first sign of discomfort during eruption of permanent teeth. External factors can also influence the disease as one or more FOPS events could be directly linked to a situation causing anxiety in 20% of cats. FOPS can be resistant to traditional analgesics and in some cases successful management required neurogenic analgesics such as phenobarbitone and other anti-epileptic drugs.

The investigating group, who cover the specialities of Neurology, Ethology, Dentistry and Internal Medicine, became aware of this syndrome in 1997 and it prompted an ongoing investigation into the character of the syndrome, possible aetiology, and treatment that continues.

Clinical signs

- Exaggerated licking and chewing movements
- Pawing at the mouth.
- Discomfort is unilateral
- Episodic or continuous. In the episodic version, the distress usually occurs after eating and lasts between 5 minutes and 2 hours.
- Cat remains alert and can be distracted with difficulty.
- Some cats have continuous discomfort that increases in intensity when excited or stressed. These cats are often anorexic and in considerable distress, requiring paw bandaging and/or an Elizabethan collar to prevent severe mutilation.
- Some cases appear to be associated with oral disease, which can be divided into 5 groups:
  a) Mouth ulceration, especially as a consequence of Calici virus infection or primary vaccination
  b) Erupting permanent teeth
  c) Dental disease, most commonly periodontal disease and dental resorptive lesions.
  d) Recent routine dental treatment including difficult/traumatic extraction
  e) After tooth atomisation with drills when neurovascular bundles beyond the roots have been damaged.

Treatment of, or natural resolution of the lesions, can result in improvement. However many cases have
recurrences which proved more difficult to successfully treat. In the kittens, the problem often resolves when the mouth ulceration/teething does, however these cats may have a recurrence when adult. Other possible influences include stress, from systemic disease, pregnancy, or environmental factors e.g. a multi-cat household. Spontaneous remission and recurrence is common.

Hypothesis of pathogenesis

Orofacial pain disorders are well described in humans. The feline orofacial pain syndrome shows some similarities to trigeminal neuralgia. This disease is characterised by paroxysmal bouts of pain in the distribution of the trigeminal nerve, usually the jaw region. The pain is precipitated by trigger factors of which the most common is facial movement e.g. chewing. For trigeminal neuralgia to occur there must be a combination of peripheral disturbance or damage, e.g. dental disease, together with a cerebral brainstem disinhibition of the trigeminal apparatus. This results in a paroxysmal discharge and reverberation of pain impulses when a trigger point is elicited1. It is for this reason that anti-epileptic drugs are one of the most effective treatments. More unusual human facial pain syndromes include glossodynia (burning mouth syndrome)5. This is described as a burning or prickling sensation of the oral mucosa most commonly the anterior tongue6. In many of the affected cats, tongue discomfort seems to be the primary problem and several cases severely mutilated the tongue. There are also facial pain syndromes primarily involving missing teeth (atypical odontalgia or phantom tooth pain), the temporal mandibular joint (temporomandibular pain and dysfunction syndrome) and one where taste is the trigger (gustatory neuralgia)5,6.

As Burmese cats are predisposed, this raises the question of a hereditary susceptibility. The original study group involved in the investigation of this condition proposed that these cats are susceptible to the problem because of a disorder of central processing. They subsequently acquire a peripheral lesion such as dental disease resulting in the orofacial pain disorder. Stress may be a contributory factor. Treatment may be effective initially however there is a high chance of recurrence and treatment may become less effective with time. Many of the affected Burmese cats are closely related. A familial trigeminal neuralgia is recognised (rarely) in humans2,3,4.

Investigation

Diagnostic work up of affected cats includes ruling out predisposing medical problems, especially dental disease. Specialist opinion and good quality dental radiographs are recommended. Tooth resorption lesions (TR’s) are one of the more common associated diseases and the questionable practice of dental atomisation of fractured or retained roots can aggravate the problem. It is also important to explore the history for possible psychological factors, e.g. stress from multi-cat household, and implement appropriate management.

Treatment

Medical treatment is dependent on the underlying disease, if there is one. Some cases with gingivitis have appeared to respond to antibiotics, although spontaneous remission could not be ruled out. NSAID’s were an effective analgesia for some mildly affected cases. Opioids were very useful for severe hospitalised cases. In the main, anti-epileptic drugs (diazepam or phenobarbitone) gave more sustained and consistent relief. Some cases, especially those with chronic dental disease, responded to steroid therapy

Phenobarbitone is the preferred first choice drug for serious cases because of the greater risk of idiosyncratic hepatic failure with diazepam. An initial dose rate of 2-3mg/kg should be used with a reduction over time to the lowest effective dose rate. Occasionally life-long therapy is required. Somnolence, weight gain, decreased grooming, and transient cystic calculi are reported as possible adverse effects.
**Gabapentin** (Neurontin: Pfizer) has been used more recently to good effect in milder cases. The dose rate is in the range of 10-20mg/kg every 8-12 hours. In general, lower doses are best to start with. If no effect is seen after four weeks, the dose can be increased. This drug is used for chronic nerve root pain in humans (e.g. Shingles). Carbamazine & amitriptyline are also reported to have been used in some cases.

**Selegiline** is effective for some and is probably more appropriate for those with a behavioural component or contributing stressful environment. *The dose used is 1mg/kg p.o. sid.* For these cases alterations to the environment and application of behavioural modification is also essential.

**Conclusion**

Feline Orofacial Pain Syndrome (FOPS) is a recently recognised condition affecting mainly Burmese cats. The most common triggers appear to be oral lesions especially dental disease. There are similarities to trigeminal neuralgia and other orofacial pain disorders in humans. The Burmese cat is predisposed and a hereditary susceptibility is suspected. As a general guideline, NSAID’s are the most appropriate first line therapy for mild cases and phenobarbitone for more severe acute or chronic cases. New cases are still seen in the UK on an occasional basis. There are reports of cases in Australia and New Zealand where the genetic basis of the Burmese cat population is similar to the UK but no reports from other countries up to late 2006.

**References**

1. Lee KH  Facial Pain: trigeminal neuralgia *Annals of the Academy of Medicine, Singapore* 1993 22 193-6
3. Braga FM, Bonatelli AD, Suriano I, Canteras M Familial Trigeminal neuralgia *Surgical Neurology* 1986 26 405-8