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MANAGING MOUTHS IN CATS

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INTRODUCTION

The importance and recognition of dental disease in companion animals is increasing due to increased awareness and demand by clients, overall improved diagnostics and higher delivery of medicine by practitioners. Most literature and dental information focuses on dogs, but there are a few common, and not so common, clinical disorders we should be aware of in cats and be able to manage in our practices. This manuscript will focus on two very common disease processes (periodontal disease and resorptive lesions), two moderately common diagnoses (stomatitis and endodontic disease), and two uncommon disorders (juvenile hyperplastic gingivitis and caudal palate defects) and their management. Each will be broken down into clinical signs, diagnosis, and treatment options.

COMMON DISORDERS IN THE FELINE MOUTH

Periodontal Disease

Until owners are able to provide routine homecare for their pets, periodontal disease will likely continue to be a frequent diagnosis. Most is gingivitis, but it can extend to periodontal pocket formation, periodontal tissue loss and eventually, if not treated, tooth loss. The underlying etiology of periodontal disease is bacterial plaque formation. Clinical signs for periodontal disease include gingivitis, visible plaque accumulation, calculus, halitosis, bleeding gingiva, gingival recession, periodontal pocket formation, and mobile teeth. One specific type of periodontal disease in cats is chronic alveolar osteitis, commonly seen around the canine teeth. In this condition, the inflammatory reaction within the bone around the canine teeth results in protrusion of the alveolar bone and in some instances loss of periodontal tissue attachment. Probing for periodontal pockets, oral examination, and dental radiography aid in the diagnosis of periodontal disease.5 Dental radiographs help assess the level of alveolar bone support remaining or lost due to periodontal disease and allow us to stage periodontal disease based on bone loss.12 Stage I is gingivitis with no bone loss, stage II is gingivitis with 0-25% bone loss along a root, stage III is gingivitis with 26-50% alveolar bone loss, and stage IV is greater than 50% bone loss. At stage IV periodontal disease, we should consider extraction. At any of the previous stages, treatment includes cleaning the teeth and providing some sort of periodontal pocket debridement with or without perioectics and homecare to try to re-establish the normal physiologic anatomy.

Resorptive Lesions

Resorptive lesions occur as a result of tooth structure loss due to the actions of the odontoclast.13 The stimulus of this odontoclast to initiate resorption of healthy tooth structure is still unknown. The prevalence of such lesions has been shown to be between 20-67%, and this appears to be increasing over the past decade.7,11 Clinical signs are typically associated with a sensitive tooth (facial pawing, head shaking, jaw chattering) with gingivitis along the gingival margin on the buccal surface of teeth, but in some cases, no clinical signs are visible. In cases where no clinical signs are noted, these lesions are detected subgingivally with dental radiography. 90% of these lesions have been reported to be on the buccal surface, and are most common in the premolars, molars, canine and incisor teeth, in that order.11 Recent literature has grouped these lesions into two categories with “Type I” being root resorption associated with periodontitis and “Type II” being root replacement resorption.1 Type I lesions appear radiographically as radiolucent areas within the crown or root with clear periodontal ligament space and discernable roots. Type II lesions appear radiographically as loss of root structure, blending into the alveolar bone. At the present time, treatment of these lesions depends on the type of resorption occurring. Type I lesions indicate extraction of the tooth and entire visible root to prevent possible nidus for infection. Whereas recent studies have shown that Type II lesions can be successfully treated with crown amputation and intentional root retention.1,2,7 The theory is that these roots are basically dissolving into bone and will continue to do so once the crown is removed without further problems. This modality of treatment is not to be undertaken if any radiographic evidence of periapical disease or periodontitis is present or if this is a case of feline stomatitis, in which case all dental tissue must be removed.

MODERATELY COMMON DISORDERS

IN THE FELINE MOUTH

Feline Stomatitis

One of the most painful and frustrating conditions cats can develop is feline stomatitis.4,14 The cause is unknown, but appears to be an immune reaction to plaque and the tooth structure itself, or the basement membrane of the periodontal tissues. Clinical signs include severe chronic gingivitis, with or without the following: faucitis, pharyngitis, or palatitis. Other signs include ptysialism, reluctance to eat, bleeding gingiva, extreme oral sensitivity, and weight loss or anorexia. Diagnosis is based on clinical signs and biopsy of the affected gingival tissue. The histopathology usually finds abundant lymphocytes, plasmacytes and occasional neutrophils. Although many organisms have been cultured or found in affected cat’s mouths, none has been proven to be the cause. As such, oral culture and sensitivity or viral isolation has been of little benefit. One recent interesting relationship is that of Bartonella sp. with chronic feline diseases such as gingivitis, stomatitis, and conjunctivitis. The test for Bartonella is available through National Veterinary Labs, Franklin Lakes, NJ, (201) 891-2992. If positive, the recommended treatment is 10mg/Kg Azithromycin once daily for 21 days. Bartonella sp. have zoonotic potential and thus are important from a public health standpoint. Case management of a cat with stomatitis should include CBC, full chemistry profile, urinalysis, T4 and free T4, FeLV and FIV testing, and Bartonella serology. Cleaning the teeth, homecare/brushing, oral antibiotics and corticosteroids are helpful initially, but their effectiveness for treatment usually wanes within 3-6 months. The only treatment thus far shown to have long-term results without the need further medication is either caudal or full mouth dental extraction. In the only study to report long term results of caudal or full mouth extractions, 60% had significant improvement, another 20% had some improvement, and a final 20% had little to no clinical improvement.4 Dental radiographs are essential when performing these extractions to ensure the entire root of every extracted tooth is removed. Post-operatively, antibiotics are given for 14-28 days. Pain management is...
paramount in these patients and is accomplished with pre-
anesthetic opioid administration (either Buprenorphine or
Butorphanol), intraoperative local anesthetics (Bupivacaine),
and postoperative NSAID and opioid given orally for 5-7
days. For those patients with anorexia prior to presentation,
nutritional support via esophagostomy or gastrostomy tube
may be warranted either pre or post-operatively until eating
well again. Re-evaluation at one month should show some
improvement, and further follow-up at three months should
be indicative of success or failure of treatment. If there is
little to no response, the remainder of the teeth may need to
be extracted or dental radiographs taken to ensure all
tooth/root remnants have been removed and that there are
no areas of reactive alveolar bone borders.

Endodontic Disease
Cats that are hit by cars or fall from high places may
inadvertently break the cusps of one or more of their canine
teeth, exposing the pulp canal. This is initially very painful
since the nerve supply to the tooth is exposed. Over time
and exposure to the oral bacteria, the pulp tissue becomes
non-vital and necrotic. Infection ensues and can travel to the
apex of the root, through the apical delta, or vascular
channels at the tip of the root into the periapical tissues,
periodontal ligament and alveolar bone. Seldom do cats
show obvious signs of endodontic disease, but this area is a
chronic osteomyelitis and source of infection. The most obvious
clinical signs would be a purulent draining tract either ventral
to the orbital rim, or under the chin, depending if it is either a
maxillary or mandibular canine tooth. These draining tracts
typical respond to antibiotic therapy, only to return shortly
after treatment is discontinued. The diagnosis can be made by
oral examination, using a fine probe or endodontic file to
look for any pulp canal opening and dental radiographs to
detect periapical bone loss. Radiographs can also be used to
evaluate the width of pulp canal when compared to the
same tooth (assuming it is vital) in the opposite arcade. The
non-vital tooth may have a larger/wider pulp canal indicating
that there was pulp death some time previously that resulted
in the death of the tooth. Treatment should be aimed at
removal of the source of the infection, the infected pulp
tissue. This can be accomplished either by extraction or root
removal. If the upper canine tooth is extracted, the
upper lip may, as a result, fall more medially allowing the
cusp tip of the mandibular canine tooth to strike the margin of
the upper lip, leaving ulceration. In these instances, using a
white stone bur to remove the tip of the cusp of the lower
canine tooth (2mm) and sealing any exposed dentin with a
dentinal bonding agent may help reduce the upper lip trauma.
When extracting the mandibular canine tooth, a significant
void will be left in the rostral mandibular bone, and bone
augmentation/alveolar ridge maintenance techniques such as
placing a bioactive glass particulate (Consil™, Nutramax
Labs) prior to gingival closure can help bone fill in this
defect.13

UNCOMMON DISORDERS IN THE FELINE MOUTH
Juvenile Hyperplastic Gingivitis
Early development of gingivitis, shortly after the permanent
teeth have erupted may be the onset of feline juvenile
hyperplastic gingivitis.14 Although this condition is most often
seen in Persian and Abyssinian cats, it can be seen in any
breed. The clinical signs are just as the name suggests,
inflamed and overabundant gingiva, usually seen in the
premolar/molar region. Typically, there is minimal calculus,
but the hyperplastic gingival tissue creates pseudopockets
adjacent to the crowns of the affected teeth, which produce a
breeding ground for plaque. Treatment involves cleaning the
teeth every 3-6 months, gingivectomy of the hyperplastic
gingiva, and good oral hygiene by the owners. Many of these
cats will revert to a more normal gingival architecture within
1-2 years of treatment. After improvement, these patients
may be maintained as any other cat.

Caudal Palate Defects
Defects in the palate can either be congenital or acquired.
Many of these lesions in cats are acquired either through
trauma such as a fall from high elevation, foreign body
penetration, hit by car, or electrical cord injury. The result is a
communication of the oral cavity with the nasal cavity
creating chronic rhinitis and possibly aspiration pneumonia.
Closure of these defects can be challenging due to the small
working space, lack of adjacent tissue, and thin palate
mucosa. Caudal palate defects are especially difficult due to
their location and lack of tissue to work with. One way
describe to close these lesions is the “Split-U” palate
surgery.8 The advantage of this procedure is the
maintenance of palate mucosal flap blood supply by the
major palatine arteries and complete closure of the defect.
Another way to close these defects, if tissue is available, is a
double-flap repair, as is described for oronasal fistulas in
canines.5 The first mucosal flap is created such that it is
inverted and sewn into the palate defect with the mucosa in
the nasal cavity. The second flap is a large rotating palate
flap that covers this initial flap and the defect to provide a
second layer of protection having the mucosa of this flap
in the oral cavity. If possible, the palatine artery may be
maintained in the second flap, further ensuring the blood
supply to the rotating flap. Esophagostomy or gastrostomy
tube placement and utilization for at least 4 weeks will help
prevent inadvertent damage to the repair while healing.
Follow-up should be at one month and three months to look
for persistent fistulation, which should be much smaller and
easier to manage if a second surgery is necessary.

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