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Popular Article

Periodontal Disorders and its Management in Small Animals

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Abstract

Periodontal disease is inflammation of some or all of the tooth's support structures (gingiva, cementum, periodontal ligament, and alveolar bone). When compared to gingivitis, periodontitis indicates bone loss caused by bacteria. Initially a pellicle forms on the clean tooth. The pellicle attracts aerobic gram-positive bacteria, primarily Actinomyces and Streptococci. Soon more bacteria adhere forming plaque. Within days the plaque thickens, underlying bacteria run out of oxygen and anaerobic motile rods and spirochetes begin to populate the sub-gingival area. Endotoxins released by the anaerobic bacteria cause tissue destruction and bone loss. Microscopic lesions have been found affecting dog's livers, kidneys, and brains in some animals with periodontal disease.

Introduction:

Periodontal disease is inflammation of the tooth's support structures, leading to bone loss due to bacterial infection, unlike gingivitis (Niemiec, 2008). A pellicle forms on the tooth, attracting aerobic bacteria, which then shift to anaerobes as plaque thickens. These anaerobic bacteria release endotoxins that cause tissue destruction and bone loss. In some cases, periodontal disease is associated with microscopic lesions in the liver, kidneys, and brain.

American Veterinary Dental College (AVDC) classification system

- Stage I (PD 1): Gingivitis only without attachment loss.
- Stage II (PD 2): Early periodontitis- <25% attachment loss or stage I furcation involvement in multi-rooted teeth.
- Stage III (PD 3): Moderate periodontitis- 25-50% attachment loss & stage II furcation of multi-rooted teeth.
- Stage IV (PD 4) – advanced periodontitis- >50% attachment loss & Stage III furcation present.

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Factors affecting periodontal disease

Multiple factors, including age, species, breed, chewing behavior, diet, grooming habits, occlusion, oral hygiene, and bacterial flora, contribute to the development of periodontal disease by promoting plaque accumulation and gingivitis.

Etiological agent

Dogs: Pigmented and non-pigmented Bacteroides (Porphyromonas Gingivalis, Prevoltella sp., Bacteroides sp.), Fusobacterium etc.

Cats: Peptostreptococcus sp., actinomyces, and porphyromonas sp.

Differential Diagnosis - Phemphigus, oral neoplasia, lupus, stomatitis

Diagnostic Procedures

Periodontal probing - "Probing depth" refers to the distance between the free gingival margin and the base of the pocket. In dogs, depths greater than 2 mm, and in cats, depths greater than 1 mm, are abnormal and indicative of periodontal disease (Niemiec, 2008).

Attachment loss- Measures between cement-enamel junction (CEJ) and apical extent of pocket. Normally the gingival sulcus is located at the CEJ, any attachment loss is abnormal.

Imaging - Radiography is a crucial diagnostic tool for evaluating periodontal disease, as up to 60% of the disease is hidden below the gum line. Early stage III disease is indicated by reduced density and sharpness of the crestal bone. As the disease advances, signs include loss of lamina dura mineralization and furcation involvement in multirooted teeth. No radiographic changes are seen in stages I and II. In severe cases, radiographs reveal significant bone loss around one or more roots, which may appear as horizontal, vertical, or oblique bone defects (Ellen *et al.*, 2010).

Treatment: The primary objective of periodontal therapy is to manage plaque.

Stage I and II - Professional cleaning includes hand scaling, polishing, irrigation, and fluoride application.

Stage III - For pocket depths of 3-6mm in dogs and 2-4mm in cats, treatment may include closed root planing, subgingival curettage, and the application of perioceutic gel.

Stage IV - In dogs, a pocket depth greater than 6 mm and in cats greater than 4 mm necessitates surgery for root treatment via open flap curettage or extraction.

With 2-3 mm of healthy attached gingiva, an apical reposition flap can reduce pocket depth in areas with alveolar bone loss (Ellen *et al.*, 2010). If insufficient healthy gingiva is available, options include a rotated flap, free gingival flap, or extraction.

Bone replacement procedures – Can aid in preserving teeth with two- or three-walled infra-bony pockets.

Guided tissue regeneration - Utilize tissue barriers to separate gingival tissue from the root surface.

Periodontal splinting – This technique is particularly useful in the incisor region to stabilize mobile

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teeth. Splinting criteria include normal periodontal support on both sides of the tooth, strict home care, and a cooperative patient who avoids chewing hard objects (Khatariya *et al.*, 2020).

Medications: Clindamycin, an antibiotic approved for dental infections, may be administered a week before periodontal treatment, perioperatively 15 minutes before anesthesia, postoperatively for 7-10 days, or in pulse therapy during the first five days of each month.

Home Care: Fluoride - Stannous fluoride preparations help control periodontal disease by reducing plaque through enamel deposition and decreasing dental pain. A 0.4% strength is recommended for patients with stage 3 and 4 periodontal disease, particularly those with exposed root surfaces.

Chlorhexidine- Chlorhexidine is the most effective product for inhibiting plaque formation, with bacteriostatic and bactericidal effects against bacteria, fungi, and some viruses. Once absorbed, it remains effective for up to 24 hours. For maximum efficacy in humans, chlorhexidine should be swished for one minute twice daily, as contact time is crucial for binding to the tooth and gingival sulcus. It can be applied using a gauze sponge, cotton-tipped applicators, spray, or finger brushes (Khatariya *et al.*, 2020).

Stages of periodontal disease:

Gingivitis:

Periodontal disease is classified from stages I to IV based on radiographic and clinical severity. Stage I, known as gingivitis, presents with clinically inflamed and swollen gingiva. At this stage, no bone loss is evident, and dental radiographs appear normal.

Early periodontitis

Stage II disease, or early periodontitis, is marked by the initial appearance of radiographic abnormalities. It involves loss of alveolar supporting bone, apical migration of the gingival fibers, and junctional epithelium. Bone loss can be generalized, appearing as horizontal loss across some or all tooth surfaces. Radiographically, the earliest sign is a loss of the crestal bone's sharp definition, leading to a blunted and irregular alveolar crest with potential localized erosion. In the incisor regions, the alveolar crests become blunted, while in premolar and molar regions, the sharp angle between the lamina dura and alveolar crest may be lost (Niemiec, 2008).

Established periodontitis

Stage III periodontal disease is characterized by pocket formation and significant bony destruction, often extending to the buccal and/or lingual alveolar bone plates. Radiographically, it may present as horizontal or vertical bone defects. Horizontal bone loss refers to the reduction in bone height across several adjacent teeth and can be classified as localized or generalized. It is further categorized by severity: mild (<10% bone loss), moderate (10-30%), or severe (>30%). In horizontal bone loss, both the buccal and lingual plates, as well as the interdental bone, are resorbed (Khatariya *et al.*, 2020).

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Vertical bone defects, also known as proximal intra-bony defects, extend apically from the alveolar crest and are enclosed by three walls of bone: two marginal (lingual/palatal and facial) and a hemiseptum (the interdental septum bone that remains on the adjacent, unaffected tooth after destruction of the distal or mesial interproximal bone) (Watson, 2006). As the disease advances, the defect may become two-walled. Radiographically, vertical bone defects appear V-shaped and sharply outlined, adjacent to the root surface, with surrounding bone showing a normal appearance. Small intra-bony defects may not be visible on radiographs, but using a gutta-percha point in the pocket can help assess the defect's extent.

Inconsistent bony margins

An inconsistent bony margin results from uneven resorption of the alveolar cortical plate on the lingual or facial surfaces. This is common in advanced periodontitis, where thin marginal bone may not be fully resorbed by inflammation. Radiographically, inconsistent margins can be difficult to detect, as lesions may be superimposed on the root of the affected tooth.

Advanced periodontal lesions

Stage IV periodontal disease is characterized by deep pockets, tooth mobility, gingival bleeding, and pustular discharge, with extensive bone loss. Furcation exposure, resulting from bone loss at the furcation of multi-rooted teeth, may precede advanced periodontal disease. Radiographically, it can be challenging to identify interradicular involvement unless a radiolucent area is present at the furcation. Advanced furcation exposures, where both cortical plates are lost, are more readily visible on radiographs (Watson, 2006).

Class I (incipient) furcation exposure is when a probe tip can just enter the furcation area (<1 mm), with most of the bone intact. Class II (definite) exposure occurs when the probe extends more than 1 mm horizontally into the furcation area. Class III (through and through) lesions, caused by advanced periodontal disease, allow an explorer probe to pass unobstructed through the defect due to significant bone loss. Alveolar dehiscence is characterized by complete resorption of the alveolar cortical bone along the entire root length, visible radiographically as radiolucency around the affected root (Watson, 2006).

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