Periodontal Disease

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Periodontal disease is the most commonly diagnosed problem in small animal veterinary medicine. In the vast majority of cases, however, there are little to no outward clinical signs of the disease process, and, therefore, therapy often comes very late in the disease course. Consequently, periodontal disease is also the most undertreated animal health problem. In addition, unchecked periodontal disease has numerous dire consequences both locally and systemically. These consequences are detailed in the article and should be utilized to educate clients and encourage compliance of therapeutic recommendations. The local consequences include oronasal fistulas, class II perio-endo lesions, pathologic fractures, ocular problems, osteomyelitis, and an increased incidence of oral cancer. Systemic diseases linked to periodontal disease include: renal, hepatic, pulmonary, and cardiac diseases; osteoporosis, adverse pregnancy effects, and diabetes mellitus. Before the discussion of consequences, this article covers the pathogenesis of periodontal disease, followed by clinical features and diagnostic tests.

Keywords: periodontal disease, gingivitis, periodontitis, pathologic fracture, endocarditis, class II perio-endo abscess, oronasal fistuala

Periodontal disease is the number one health problem in small animal patients.[1] By just 2 years of age, 70% of cats and 80% of dogs have some form of periodontal disease.[2] Periodontal disease is described in 2 stages, gingivitis and periodontitis. Gingivitis is the initial, reversible stage of the disease process in which the inflammation is confined to the gingiva.[2,3] This inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent home care.[3,4] Periodontitis is the later stage of the disease process and is defined as an inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms.[5] This inflammation results in the progressive destruction of these tissues, leading to gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets can be reduced or eliminated by proper removal of plaque and calculus.[6] However, periodontal bone loss is irreversible (without regenerative surgery).[2] Although bone loss is irreversible, it is possible to arrest its progression.[3,7] It is, however, much more difficult to treat and maintain periodontally diseased teeth as compared with healthy teeth. It is important to note that periodontal attachment loss may be present with or without active periodontal inflammation.[2]

Periodontal disease is initiated when oral bacteria adhere to the teeth in a substance called plaque. Plaque is a biofilm, which is made up almost entirely of oral bacteria, contained in a matrix composed of salivary glycoproteins and extracellular polysaccharides.[2,8] Calculus (or tartar) is essentially plaque that has become calcified by the minerals in saliva.[9] Plaque and calculus can contain up to 100,000,000,000 bacteria per gram.[10] Bacteria within a biofilm do not act like free-living or "planktonic" bacteria, and, in fact, they are 1000 to 1500 times more resistant to antibiotics than their planktonic counterparts.[8] The plaque on the tooth surface is known as supragingival plaque.[8] Once it extends under the free gingival margin and into the area known as the gingival sulcus (between the gingiva and the teeth or alveolar bone), it becomes known as subgingival plaque.[8] Supragingival plaque is thought to affect the pathogenicity of the subgingival plaque in the early stages of periodontal disease.[2] Once the periodontal pocket forms, however, the effect of the supragingival plaque and calculus is minimal. Therefore, control of supragingival plaque alone is ineffective in controlling the progression of periodontal disease.[11]

Initial plaque bacteria from healthy sites consist of predominately nonmotile, Gram-positive, aerobic facultative rods and cocci.[8] Gingivitis is caused by an increase in the overall numbers of bacteria, and this increase is primarily of motile Gram-negative rods (approximately 50%) and anaerobic species.[8] In established periodontal disease, Gram-negative rods account for approximately 74% of the microbiotic flora.[8] Finally, elevated numbers of spirochetes are found in almost all periodontal pockets, and anaerobic organisms compose 90% of the bacterial species in chronic periodontal disease.[12,13]

Classically, periodontal disease was thought to be caused by an increase in the overall numbers of bacteria. The nonspecific plaque hypothesis was based on the fact that peri-

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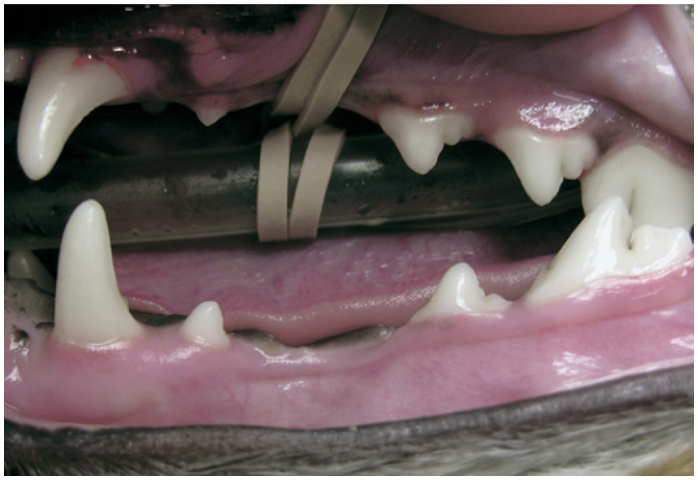

Figure 1. Normal gingival tissues. Photograph of the maxillary right arcade of a dog, revealing normal gingival tissues. Note the absence of calculus on the teeth, the coral pink color of the gingiva, and the lack of erythema or edema (inflammation) to the gingiva. Also, note the massing second premolar (106).

odontal disease is associated with an increased level of plaque and calculus.[8,14] It was thought that low levels of plaque bacteria were controlled by the host response. In addition, research found that the concentration of bacteria in periodontally diseased sites is twice as high as in healthy sites.[15] Recent studies, however, point to a few, virulent strains of bacteria as being responsible for the attachment loss seen with periodontal disease.[16-18] The specific plaque hypothesis is based on the fact that a few species are seen in virtually all cases of established, chronic periodontal disease.[8] These findings have led to the development of the "one-stage full-mouth disinfection" treatment as well as a vaccine against these organisms. However, the cornerstone of therapy is still meticulous plaque control.

The bacteria in the subgingival plaque secrete toxins as well as metabolic products.[19] Also produced are cytotoxins and bacterial endotoxins, which can invade tissues on their own and, in turn, cause inflammation to the gingival and periodontal tissues.[2] This inflammation causes damage to the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis, ie, the destruction of the attachment between the periodontal tissues and the teeth.[2] In addition to directly stimulating inflammation, the bacterial metabolic byproducts also elicit an inflammatory response from the animal. White blood cells and other inflammatory mediators migrate out of the periodontal soft tissues and into the periodontal space due to increased vascular permeability and increased space between the crevicular epithelial cells.[20] White blood cells fight the infection by phagocytizing bacteria but may also release enzymes to destroy the bacterial invaders either by design or after their death. When released into the sulcus, these enzymes will cause further inflammation of the delicate gingival and periodontal tissues. In fact, the progression of periodontal disease is determined by the virulence of the bacteria combined with the host response.[20] It is the host response that often damages the periodontal tissues. However, patients with deficient immune systems typically have more severe periodontal disease than those individuals in good health.[2,20] Human immunodeficiency virus, diabetes, and stress are significant risk factors for severe periodontitis in humans, and could likely be extrapolated to our animal patients.[20]

The inflammation produced by the combination of the subgingival bacteria and the host response damages the soft tissue attachment of the tooth, and decreases the bony support via osteoclastic activity.[19] This causes the periodontal attachment of the tooth to move apically (toward the root tip).[2,5]

As periodontal disease progresses over time, the attachment loss continues in a nonlinear pattern as active stages of destruction are followed by quiescent phases (burst).[21] The end stage of periodontal disease is tooth loss; however, the disease has created significant problems before tooth exfoliation.

Clinical Features

It is important to be familiar with normal features to identify abnormal findings. Normal gingival tissues are coral pink in color (allowing for normal pigmentation), and have a thin, knife-like edge, with a smooth and regular texture (Fig. 1). In addition, there should be no demonstrable plaque or calculus on the dentition. Normal sulcal depth in a dog is 0 to 3 mm and in a cat is 0 to 0.5 mm.[22]

The first clinical sign of gingivitis is erythema of the gingiva (Fig. 2).[23] This is followed by edema (Fig. 3), gingival bleed-

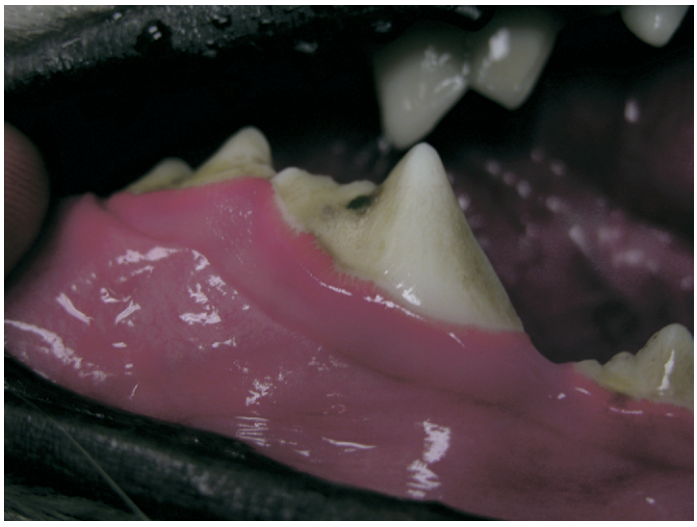

Figure 2. Early gingivitis. This clinical photograph of the maxillary left fourth premolar (208) of a dog reveals erythema to the gingival margin and mild calculus on the teeth, which is consistent with early gingivitis. Note the lack of edema of the gingiva.