

PERIODONTAL DISEASE

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Introduction

Periodontal disease is the number one health problem in small animal patients. By two years of age, 70% of cats and 80% of dogs have some form of periodontal disease. However, there are generally little to no outward clinical signs of the disease process, and therefore, therapy typically comes very late in the disease. Consequently, periodontal disease may also be the most undertreated disease in our patients. Additionally, unchecked periodontal disease has numerous local as well as systemic consequences. Local consequences include: oronasal fistulas, class II perio-endo lesions, pathologic fractures, ocular problems, osteomyelitis, and increased incidence of oral cancer. Systemic diseases which have been linked to periodontal disease include: renal, hepatic, pulmonary, and cardiac diseases; osteoporosis, adverse pregnancy effects, and diabetes mellitus.

Pathogenesis:

Periodontal disease is generally described in two stages, gingivitis and periodontitis. Gingivitis is the initial, **reversible** stage in which the inflammation is confined to the gingiva. The gingival inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent homecare. 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. The inflammation results in the progressive destruction of the periodontal tissues, leading to attachment loss. This can be seen as gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets may be reduced or eliminated by proper plaque and calculus removal. However, periodontal bone loss is **irreversible** (without regenerative surgery). Although bone loss is irreversible, it is possible to arrest its progression. However, it is more difficult to maintain periodontally diseased teeth in comparison to healthy teeth. Additionally, periodontal attachment loss may be present with or without active periodontal inflammation.

Periodontal disease is initiated by oral bacteria which 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. Calculus (or tartar) is basically plaque which has secondarily become calcified by the minerals in saliva. **Plaque and calculus may contain up to 100,000,000,000 bacteria per gram.** Bacteria within a biofilm do not act like free living or “planktonic” bacteria; and in fact are 1,000 to 1,500 times more resistant to antibiotics than are planktonic bacteria. Plaque on the tooth surface is known as supragingival plaque. 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 is called subgingival plaque. Supragingival plaque likely affects the pathogenicity of the subgingival plaque in the early stages of periodontal disease. However, once the periodontal pocket forms, 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.

Initial plaque bacteria consists of predominately non-motile, gram-positive, aerobic facultative rods and cocci. Gingivitis is initiated by an increase in the overall number of bacteria, which is primarily motile gram negative rods and anaerobes. In established periodontal disease, gram negative rods account for approximately 74% of the microbiotic flora. 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.

Classically, periodontal disease was thought to be caused by an increase in the overall numbers of bacteria. The *non-specific plaque hypothesis* was based on the fact that periodontal disease is associated with an increased level of plaque and calculus. It was thought that low levels of plaque bacteria were controlled by the host response. It was further noted that the concentration of bacteria in periodontally diseased sites is twice as high as in healthy sites. However, recent studies point to a few, virulent strains of bacteria as being responsible for the attachment loss seen with periodontal disease. The *specific plaque hypothesis* is based on the fact that these few species are seen in virtually all cases of chronic periodontal disease. 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. 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. This inflammation causes damage to the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis, i.e. the destruction of the attachment between the periodontal tissues and the teeth. 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. 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. 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} HIV, diabetes, and stress are significant risk factors for severe periodontitis in humans, and could likely be extrapolated to our animal patients.

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. This causes the periodontal attachment of the tooth to move apically (towards the root tip).

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

Clinical Features:

It is important to be familiar with normal features in order 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. In addition, there should be no demonstrable plaque or calculus on the dentition. Normal sulcal depth in a dog is 0 to 3mm and in a cat is 0 to 0.5mm.

The first clinical sign of gingivitis is erythema of the gingiva. This is followed by edema, gingival bleeding during brushing or after chewing hard/rough toys, and halitosis.

Gingivitis is typically associated with calculus on the involved dentition, but is primarily elicited by PLAQUE and thus can be seen in the absence of calculus. Alternatively, widespread supragingival calculus may be present with little to no gingivitis. It is critical to remember that calculus itself is essentially non-pathogenic. Therefore, the degree of gingival inflammation (not the amount of calculus) should be used to judge the need for professional therapy. As gingivitis progresses to periodontitis, the oral inflammatory changes intensify.

The hallmark clinical feature of established periodontitis is attachment loss. In other words, the periodontal attachment to the tooth migrates apically. As periodontitis progresses, alveolar bone is also lost. On oral exam, there are two different presentations of attachment loss. In some cases, the apical migration results in gingival recession while the sulcal depth remains the same. Consequently, tooth roots become exposed and the disease process is easily identified on conscious exam. In other cases, the gingiva remains at the same height while the area of attachment moves apically, thus creating a periodontal pocket. This form is typically diagnosed only under general anesthesia with a periodontal probe. It is important to note that both presentations of attachment loss can occur in the same patient, as well as the same tooth. As attachment loss progresses, alveolar bone loss continues, until tooth exfoliation in most cases. After tooth exfoliation occurs, the area generally returns to an uninfected state, but the bone loss is permanent.

Severe local consequences:

In addition to tooth loss, there are six proven local severe sequela of severe periodontal disease.

The most common of these local consequences is an **oral-nasal fistula (ONF)**. ONFs are typically seen in older, small breed dogs (especially chondrodystrophic breeds); however they can occur in any breed as well as felines. ONFs are created by the progression of periodontal disease up the palatal surface of the maxillary canines however; any maxillary tooth is a candidate. This results in a communication between the oral and nasal cavities, creating an infection (sinusitis). Clinical signs include chronic nasal discharge, sneezing, and occasionally anorexia and halitosis. Definitive diagnosis of an oronasal fistula often requires general anesthesia. The diagnosis is made by introducing a periodontal probe into the periodontal space on the palatal surface of the tooth. Interestingly, this condition can occur even when the remainder of the patient's periodontal tissues is relatively healthy (including other surfaces of the affected tooth). Appropriate treatment of an ONF requires extraction of the tooth and closure of the defect with a mucogingival flap. However, if a deep periodontal pocket is discovered prior to development of a fistula, periodontal surgery with guided tissue regeneration can be performed to save the tooth.

Another potential severe consequence of periodontal disease can be seen in multi-rooted teeth, and is called a **class II perio-endo abscess**. This occurs when the periodontal loss progresses apically and gains access to the endodontic system, thereby causing endodontic disease via bacterial contamination. The endodontic infection subsequently spreads through the tooth via the common pulp chamber and causes periapical ramifications on the other roots.

This condition is also most common in older small and toy breed dogs, however, this author has personally treated a case in a Labrador Retriever. The most common site for a class II perio-endo lesion to occur in small animal patients is the distal root of the mandibular first molars.

The third potential local consequence of severe periodontal disease is a **pathologic fracture**. These fractures typically occur in the mandible (especially the area of the canines and first molars), due to chronic periodontal loss, which weakens the bone in affected areas. This condition is again, most commonly seen in small breed dogs, mostly because their teeth (especially the mandibular first molar) are larger in proportion to their jaws as in comparison to large breed dogs. Pathologic fractures occur most commonly as a result of mild trauma, or during dental extraction procedures. However some dogs have suffered from fractures while simply eating.

Although this is typically considered a disease of older patients, this author has personally treated three cases in dogs less than three years of age.

Pathologic fractures carry a guarded prognosis for several reasons. Adequate healing is difficult to obtain due to lack of remaining bone, low oxygen tension in the area, and difficulty in rigidly fixating the caudal mandible. There are numerous options for fixation, but the use of wires, pins or plates is generally required. Regardless of the

method of fixation, the periodontally diseased root (s) MUST be extracted for healing to occur.

Awareness of the risk of pathologic fractures can help the practitioner to avoid problems in at risk patients during dental procedures. If one root of an affected multi-rooted tooth is periodontally healthy, there is an even greater chance of mandibular fracture due to the increased force needed to extract the healthy root. An alternate form of treatment for these cases is to section the tooth, extract the periodontally diseased root, and perform root canal therapy on the periodontally healthy root. In cases where periodontitis involving a mandibular canine or first molar is identified during a routine prophylaxis, it is best to inform the owners of the possibility of a jaw fracture prior to attempting extraction of the offending tooth.

The fourth local consequence of severe periodontal disease results from inflammation close to the orbit which could potentially lead to **blindness**. The proximity of the tooth root apices of the maxillary molars and fourth premolars, places the delicate optic tissues in jeopardy. In cats (especially brachycephalic), the apices of the maxillary canines lie in this area and can create similar issues.

The fifth local consequence is described in recent studies which have linked chronic periodontal disease to **oral cancer**. The association in this case is likely due to the chronic inflammatory state that exists with periodontitis.

The final significant local consequence of periodontal disease is chronic **osteomyelitis**, which is an area of dead, infected bone. Dental disease is the number one cause of oral osteomyelitis. Furthermore, once an area of bone is necrotic, it does not respond effectively to antibiotic therapy. Therefore, definitive therapy generally requires aggressive surgical debridement.

In some cases, the bacterial infection may also result in a septicemia. In one case treated by this author, the patient had white blood cell counts of >50,000 on several occasions, which responded transiently to antibiotics. However, each time the antibiotics were discontinued the infection returned. On clinical presentation, the patient had halitosis, but no obvious dental disease other than a small fistulous tract. A dental radiograph revealed significantly mottled bone in the area, which prompted aggressive surgical debridement of the area of necrotic bone. Histopathology was consistent with osteomyelitis, and after a course of clindamycin, the patient did not relapse.

In another case treated by this author, the patient presented with an entire hemi-mandible which was necrotic secondary to osteomyelitis. In this case, the patient required a complete hemi-mandibulectomy.

Severe systemic manifestations:

Systemic ramifications of periodontal disease are also well documented. The inflammation of the gingiva and periodontal tissues that allows the body's defenses to attack the invaders also allows these bacteria to gain access to the body. Recent animal studies suggest the possibility that these bacteria negatively affect the kidneys and liver, leading to decrease in function of these vital organs over time. Furthermore, it has also been suggested that these bacteria can become attached to previously damaged heart valves (IE valvular dysplasias) and cause endocarditis, which in turn can result in

intermittent infections, and potentially thromboembolic disease. Other studies have linked oral bacteremias to cerebral and myocardial infarctions and other histological changes. Additional human studies have linked periodontal disease to an increased incidence of chronic respiratory disease (COPD) as well as pneumonia. There are many studies that strongly link periodontal disease to an increase in insulin resistance, resulting in poor control of diabetes mellitus as well as increased severity of diabetic complications (wound healing, microvascular disease). Additionally, it has been shown that diabetes is also a risk factor for periodontal disease. Periodontal disease and diabetes are currently viewed as having a bidirectional interrelationship where one worsens the other. Finally, it has been proven in animal studies that periodontal disease can elicit an increase in inflammatory lipids as well as an overall lipidemic state. This is described as a state of overall body inflammation leading to chronic disease processes and an abnormal immune response.

While some of these studies are not definitive, we know that periodontal disease is an infectious process and that affected patients must deal with these bacteria on a daily basis, which in turn can lead to a state of chronic disease. Therefore, we must learn to view periodontal disease as not just a dental problem that causes bad breath and tooth loss, but as an initiator of more severe systemic consequences. As one author states, "Periodontal disease is clearly an important and potentially life threatening condition, often underestimated by health professionals and the general public". Only by thinking in these terms can we fully appreciate the scope of the disease process and discuss the problem with clients so that they can appreciate the depth of the problems their pets face. This information will significantly increase client compliance with homecare and dental prophylaxis, as well as advanced dental procedures.