Periodontal disease: Aetiopathogenesis

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Periodontal disease is a collective term for **plaque-induced inflammation** and is a series of diseases including **gingivitis and periodontitis**. It comes from the Greek ‘peri’ meaning around, and ‘odons’ meaning tooth. Periodontology is the branch of dentistry concerning the supporting structures or **periodontium** of the teeth.

ANATOMY

The periodontium (or periodontal tissue) consists of the **gingiva**, the **cementum**, the **alveolar bone** and the **periodontal ligament**.

GINGIVA

The gingiva surrounds each tooth and comprises the **free gingival**(gingival margin), **attached gingiva** and **gingival sulcus**. The attached gingiva is firmly attached to the underlying periosteum of the alveolar bone. It is continuous with the alveolar (or oral) mucosa and is demarcated from it by the **muco-gingival junction**. The **free (or marginal) gingiva** is that found coronal (ie towards the crown) to the cementoenamel junction (CEJ). The cementum covers the root, and enamel covers the crown of the tooth. A **gingival sulcus** is formed around the tooth and has a normal depth of 0-3mm in dogs and 0-1mm in the cat. This depth is measured with a periodontal probe. At the base of the gingival sulcus, the cells are **junctional epithelial cells** and are tightly attached to the enamel via hemidesmosomes. This is the epithelial attachment to the tooth. The alveolar mucosa is a loose, moveable non-keratinised epithelium. The epithelium of the attached and free gingiva is a keratinised stratified squamous epithelium with many rete pegs. About 70% of the connective tissue of the gingiva is collagen.

CEMENTUM

Cementum covers the roots, and is a hard, bone-like substance but does not contain blood, lymph or nerves. It is capable of continuous deposition throughout life. It consists of collagen fibres in a matrix, with about 65% mineral content in the form of hydroxyapatite. It is an important structure involved in reparative and resorptive processes. It also anchors periodontal ligament fibres to the root.

PERIODONTAL LIGAMENT (PDL)

This acts as a suspensory, shock-absorbing ligament. It allows minute movement of the teeth, for instance during mastication. It is designed to withstand sudden, sharp forces. Collagen fibres are arranged in functional groups and act to anchor the tooth to the alveolar bone. The PDL space is about 0.25mm wide. It also contains blood and lymph vessels, nerve fibres, elastic fibres and many cell types (including fibroblasts, cementoblasts, osteoblasts, osteoclasts, cementoclasts, epithelial rest cells of Malassez and undifferentiated mesenchymal cells).
ALVEOLAR BONE

The alveolar processes are the parts of the mandible and maxilla that form and support the tooth sockets. Depressions in the alveolar bone (alveolar sockets) contain the roots of the teeth. The alveolar bone consists of four layers; the periosteum, compact bone, cancellous bone and the cribriform plate lining the alveolar sockets. This appears as a radioopaque line, the lamina dura on radiographs.


Periodontal disease is a collection of diseases affecting these periodontal tissues, causing inflammation and loss of attachment for the tooth/teeth. It is caused by bacterial plaque.

What is plaque?

Plaque is a whitish, sticky substance. It is a biofilm. A biofilm is an aggregation of microorganisms in which cells are stuck to each other and to a surface. During the initial stages of plaque formation, a pellicle is formed, which is an acellular film of salivary glycoproteins, polypeptides and lipids. Specific bacteria with adhesion properties initially colonise this, allowing further bacterial colonisation by co-aggregation and co-adhesion. This matures to a biofilm of many different species of bacteria embedded in a matrix of host and microbial origin. As plaque matures, the composition changes contributing to the development of periodontal disease.

Initially plaque is composed of Gram-positive aerobic bacteria. These bacteria consume large quantities of oxygen and lower the oxygen content of the surrounding microenvironment, which is particularly important in the gingival sulcus. As the oxygen content drops, the growth of anaerobic bacteria is supported. As periodontal disease progresses, the bacterial population shifts from a Gram-positive aerobic community to a Gram-negative anaerobic population. Specific bacteria are known for their pathogenic role including Porphyromonas spp, Prevotella Spp and Spirochaetes.

What is calculus?

Also known as tartar, it is mineralised plaque. Supra-gingival calculus (ie above the gingival margin, towards the crown) is not in itself pathogenic. However, it does provide a further attachment surface for plaque. Sub-gingival plaque (ie beneath the free gingival margin within the gingival sulcus) is of pathogenic significance as it worsens the inflammatory response due to its foreign nature. Therefore removing only supra-gingival calculus is a cosmetic exercise. The disease process will progress if sub-gingival plaque and calculus is left behind. There is no correlation between the amount of calculus and severity of periodontal disease.
What is gingivitis?

Plaque accumulation at the gingival margin and in the sulcus, causes inflammation of the gingiva. Initially this is termed *marginal gingivitis* as it involves the free edge of the gingiva only, and then progresses to include the whole gingiva. The flora of clinically healthy gingiva is mainly *aerobic and facultative anaerobic*. Aerobes consume large quantities of oxygen which increases the number of pathogenic gram negative *anaerobes*. Inflammation results from the action of bacterial enzymes, toxins (eg LPS) and antigens as well as the *host’s immune response*. An initial defence using plasma factors such as complement, and salivary enzymes will progress to a neutrophil response if unsuccessful in controlling the infection. Neutrophils are not able to engulf bacteria within a biofilm and so degranulate within the sulcus contributing to the inflammation. If neutrophils are unsuccessful, monocytes are recruited which develop into macrophages which can present to lymphocytes, so that chronic inflammation is present. There is no tissue destruction and this phase is *reversible* by diligent home care (ie tooth brushing). In experiments performed in the 1960s, human volunteers stopped brushing their teeth for 7 days, developing intense gingivitis (and no doubt horrendous halitosis). This was *reversible* once the subjects resumed toothbrushing.

As the gingiva becomes inflamed, it appears swollen, red and oedematous. It has a tendency to bleed either spontaneously, or upon probing with a periodontal probe.

It is important to assess gingivitis for *every tooth* when examining a patient undergoing dental treatment. This is performed by visually assessing the gingiva, and assessing the gingival sulcus by probing with a periodontal probe.

This will induce bleeding in an inflamed gingiva. A gingivitis *score* for each tooth should be assessed and noted on the patient’s dental chart. Løe and Silness (1964) developed a useful gingivitis scoring system still in use today.

<table>
<thead>
<tr>
<th>Appearance</th>
<th>Bleeding</th>
<th>Inflammation</th>
<th>Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>-</td>
<td>-</td>
<td>0</td>
</tr>
<tr>
<td>Mild redness, oedema</td>
<td>No bleeding on probing</td>
<td>Mild</td>
<td>1</td>
</tr>
<tr>
<td>Redness, oedema</td>
<td>Immediate bleeding on probing</td>
<td>Moderate</td>
<td>2</td>
</tr>
<tr>
<td>Marked redness, oedema</td>
<td>Spontaneous/profuse bleeding</td>
<td>Severe</td>
<td>3</td>
</tr>
</tbody>
</table>
What is periodontitis?

Inflammation and irreversible destruction of the periodontal tissues (remember: gingiva, cementum, periodontal ligament, alveolar bone) resulting in attachment loss of the tooth. Periodontitis is irreversible. Periodontitis develops with the sub-gingival extension of plaque-induced inflammation. Sulcular epithelium becomes inflamed and breaks down, allowing bacteria and their by-products access to deeper periodontal tissues. Bacteria accumulate in periodontal pockets further reducing the oxygen tension and favouring pathogenic anaerobes such as Porphyromonas spp (eg P. gulae) Prevotella spp, Fusobacterium spp and Spirochaetes. So that periodontal disease develops by the change of aerobic flora, to more pathogenic gram negative anaerobes. Connective tissue under the epithelial attachment becomes affected, and the epithelial attachment migrates apically. Think of this as a zip being undone. Thus a periodontal pocket is created.

The gingiva may also be destroyed, creating areas of gingival recession. Gingival recession is measured from the cement-enamel junction to the level of the free gingival margin. Alveolar bone becomes re-absorbed by the action of osteoblasts and osteoclasts which are activated by bacteria, their by-products and by inflammatory cells. PMNs cannot engulf bacteria within a biofilm, and so degranulate in the periodontal pocket thus increasing the inflammation.

As alveolar bone is re-absorbed, the attachment of the tooth is reduced. It will also start to reveal the space between roots on multi-rooted teeth, the furcation. An exposure of the furcation is evidence of horizontal bone loss. Vertical bone loss also occurs and will allow increased probing depth.

Vertical bone loss will allow increased probing depths into the periodontal pocket.

The normal gingival sulcus is <2mm in dogs (3mm canine tooth) and 1mm for cats. This is measured with the periodontal probe. The sulcus of every tooth should be examined in at least 6 locations, 3 buccal and 3 lingual/palatal or inserted into the sulcus and walked around. The probe is inserted into the sulcus to check for pockets, abnormal pits and depressions in the root surface, and subgingival calculus. The depth and location of pockets should be accurately recorded on the patient’s dental chart. The probe should also be used to assess the status of the furcation (remember three rooted teeth have 2 furcations).

It is important to understand the concept of clinical attachment loss (CAL). For instance, gingival recession of 2mm, plus a pocket depth of 4 mm, actually means attachment loss of 6mm.

Periodontitis is therefore characterised by periodontal pocket formation, bone resorption and inflammation. This is essentially irreversible. The aim of treatment is to stop progression of disease. Periodontal therapy can be performed by a veterinary dentist, which can re-grow bone in some cases. This would only be attempted where homecare is optimal.
It is also important to remember that periodontitis can be localised and is not always generalised. It can be site specific, so may be only 1 site round one tooth. Hence the importance of charting and examining every tooth. Remember: There is no correlation between the amount of calculus and severity of periodontal disease.

It is important to understand that although the aetiological agent is bacterial plaque, the progression and severity of periodontal disease is influenced by a number of factors. There is a complex interaction between bacteria and their toxins and by-products, plus the host’s immune system, genetic predisposition and other local factors.

**CLINICAL SIGNS**

- Halitosis (oral malodour/’doggy breath’) caused by volatile sulphur compounds produced by oral bacteria (mostly anaerobes). These VSC’s are also toxic to the oral epithelium and contribute to disease progression.
- Salivation/blood tinged saliva
- Variable amounts plaque+calculus
- Inflamed, bleeding gingiva
- Purulent discharge from periodontal pocket
- Loss of normal gingival contour (gingival hyperplasia/recession)
- Furcation exposure
- Tooth mobility
Dysphagia/pain on eating – Rarely. Dogs and cats with periodontal disease often show no symptoms until it is in the advanced stages which is when owners may notice a problem and seek professional help. The earlier in the disease process we can spot it, the more likely we are to be able to reverse or halt the disease process.

Incidence

Periodontal disease is arguably the most common disease in small animal practice today. Even in 1899 Talbot found that 75% dogs in post-mortem studies had periodontitis. In 1965 Bell found a similar percentage of dogs affected, and in 2001 a similar percentage of cats (Lommer et al 2001). It is essential that the mouth be thoroughly examined in any clinical examination or health assessment.

Local consequences

Periodontal disease can cause severe local consequences if left untreated:

- Pain, discomfort (dogs and cats will rarely stop eating or show any signs of oral pain)
- Periodontal abscess (painful)
- Periapical abscess (very painful!)
- Oronasal fistulae leading to chronic rhinitis
- Pathologic mandibular fracture
- Osteomyelitis
- Strategic tooth loss

Systemic consequences

Chronic periodontitis is also responsible for serious systemic consequences. Animals with periodontal disease are more likely to have increased levels of systemic cytokines and inflammatory mediators. Furthermore, it has been shown dogs and cats with periodontal disease are more likely to have histological changes associated with heart, liver and renal tissues. The presence of periodontal disease may also impact other diseases, for instance diabetes mellitus. Severe periodontal disease will contribute to insulin resistance and making stabilising patients hard. Similarly, treatment of periodontal disease may lower a patient’s insulin requirements.

In humans there are proven links with cardiovascular disease (including stroke and heart attack), diabetes, chronic kidney disease, pneumonia, premature births and low birth weights, cancer and overall younger death.
References and further reading


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Rawlinson JE, Goldstein RE et al. Association of periodontal disease with systemic health indices in dogs and the systemic response to treatment of periodontal disease. Journal of the American Veterinary Medical Association 2011; 238 (5); 601-609
