Periodontal Disease Research and Treatment – UK Experiences

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Periodontal disease (periodontitis, paradontal disease, alveolar disease and alveolar periosteitis) describes inflammation of the supporting structures of the tooth, i.e. the gingiva, periodontal ligaments, cementum and alveolar bone. In addition to its obvious great importance in human dentistry, periodontal disease is also very important in dogs and cats, and also in farm animals including sheep and cattle. Colyer who worked on London draught and riding horses in the early 1900s described periodontal disease as a most significant equine dental disease (“the scourge of the horse”) and found a prevalence of 33% of this disease in an abattoir survey of 484 horses. However, examinations of photographs of Colyer’s equine skull specimens showed the periodontal disease to be secondary to interdental food impaction caused by other dental disorders such as diastemata and displaced cheek teeth (CT). Other early studies both in South Africa and the UK have also reported the presence of periodontal disease in horses.

Baker (1970) and Wafa (1988) found that 60% of horses over 15 and 20 years of age respectively, suffered from periodontal disease of their CT. However, this periodontal disease was usually secondary to other disorders such as displaced teeth or CT diastemata. Recent clinical studies on referred cases have also shown virtually all equine periodontal disease to be associated with abnormalities of the interdental (interproximal) spaces, such as CT diastemata.

Figure 1. Normal interdental area of mandibular cheek teeth – showing tight apposition of the adjacent cheek teeth without any space for food trapping. Note the pale-coloured normal gingival, tightly attached to the peripheral cementum.

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with minimal evidence of periodontal disease associated with normally apposed teeth such as illustrated in Figure 1. Periodontal disease in donkeys is also significantly associated with diastemata, overgrown teeth, displaced teeth and increasing age. Both Baker and Wafa also recognised a mild transient periodontitis associated with dental eruption, in 40-50% of immature skulls. Little pathological research has been performed on equine periodontal disease until recently and it is likely that the constantly remodeling periodontal tissues because of their hypsodont teeth makes them differ from those in brachydont species – see papers by C. Staszyk. in these proceedings.

Periodontal disease in brachydont teeth is initiated by the adherence of organic dental plaque and bacteria to teeth. Later, the plaque often becomes calcified to form dental calculus that consists of 70-90% minerals. All teeth including equine teeth have a covering of organic matter (mainly mucopolysaccharides and glycoproteins of salivary origin and attached oral bacteria) (Fig. 2). If this is a thin layer, it can be described as a normal organic pellicle. If it becomes thicker it can then be classified as dental plaque. A thick layer of dental plaque can be present in the interdental spaces even in apparently normal horses (Fig. 3). Unlike in brachydont species, equine dental plaque generally does not become calcified, an exception being the plaque around the lower canine teeth, and less commonly on the buccal aspects of the rostral maxillary CT and incisors of horses with intercurrent dental problems. The main component of equine dental calculus is calcite that has a chalky appearance. The associated, usually low-grade periodontal disease adjacent to the calculus around canine teeth usually resolves following its removal. In general dental calculus is not a significant problem in horses that do not have intercurrent dental disorders and this low prevalence may be due to the prolonged time horses spend masticating fibrous food that prevents a buildup of excessive plaque that could later calcify.
In brachydont teeth, periodontal disease starts as gingivitis, with loss of the normal tight gingival attachment to the adjacent teeth. This gingiva then becomes inflamed due to mechanical irritation, e.g. to impacted food particles and from chemical irritation from bacteria, food and plaque, and later even due to the body’s own immune reaction, e.g. inflammation caused by neutrophil breakdown products. As the gingival destruction continues, the progressively larger gingival and later periodontal defect becomes further impacted with food and the process perpetuates itself, with the periodontitis extending deeper into the periodontal ligament and later also spreading along the buccal and lingual/palatal margins of teeth, forming large periodontal food pockets11,12 (Figs. 4 & 5).
Figure 5. The diastema between these two cheek teeth in an old horse has caused severe periodontal disease, including destruction of the periodontal ligaments and deep erosion of the peripheral cementum (black and pitted).

This inflammation and concurrent bacterial infection may even extend to involve the alveolar and supporting bones, causing bone remodeling, or even bone necrosis and infection. Eventually so much periodontal ligaments are lost that the tooth becomes loose and may even be spontaneously lost.\(^1\) More localised extension of the periodontal disease in an apical direction can lead to infection of the periodontal ligaments at the apex, ischaemia or infection of the pulp and death of the tooth.\(^3\) Wide and “open” diastemata are less likely to trap food and cause periodontal disease (Fig. 6). A periodontal disease grading system (0-4) used in small animals that is based on the percentage of dental attachment loss\(^3\) could be used in equids (Table 1).

Figure 6. This specimen has wide and open (same width at its occlusal and gingival aspect) diastema that does not tightly trap food and consequently it has normal epithelium deep in its base. This latter finding in this size and shape of diastema is the rationale behind diastema widening treatment.
Table 1. Equine Periodontal Disease Grading System

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
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<tbody>
<tr>
<td>Stage 0</td>
<td>normal</td>
</tr>
<tr>
<td>Stage 1</td>
<td>gingivitis</td>
</tr>
<tr>
<td>Stage 2</td>
<td>early periodontal disease (up to 25% attachment loss)</td>
</tr>
<tr>
<td>Stage 3</td>
<td>moderate (25-50% attachment loss)</td>
</tr>
<tr>
<td>Stage 4</td>
<td>severe (greater than 50% attachment loss)</td>
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</tbody>
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In the author’s experience, almost all equine periodontal disease is associated with abnormal spacing between teeth (Figs. 4 & 5) including due to primary diastemata, displaced or supernumerary teeth as discussed in other proceedings of this Focus meeting. Additionally, with generalized, severe dental disease such as wavemouth and shearmouth, especially in geriatric horses, restricted masticatory action can lead to generalized peripheral caries and associated periodontal disease. Periodontal disease also occurs adjacent to incisors or canine teeth affected with the EOTRH syndrome as discussed elsewhere in this proceedings. Some clinicians claim that CT overgrowths that have caused “shifting” of teeth to be the main cause of diastema in their patients. Such occlusal overgrowths are rare in the diastemata cases examined at our clinic. Additionally, a recent histological study of equine periodontal disease found periodontal disease to be associated with physical abnormalities of the interdental spaces.11

Treatment

Cases with periodontal disease should be examined for predisposing factors and these factors should be addressed whenever possible. Overgrowths that may have caused dental drift should be reduced in stages – in order to prevent pulpal exposure or thermal damage. Food should be removed from diastemata and periodontal pockets. Some clinicians claim that the removal of impacted food alone is curative – but if so – why did food become impacted at these sites in the first place? The author widens all valve-like diastemata (Figs. 7 & 8) that are causing food impaction and has reported a good success in most, even longstanding cases13 and the techniques have been well described.14 There is a significant risk of damaging the adjacent pulps – that are closest to the distal aspect of the rostral tooth15 and therefore as much tooth as possible should be removed from the mesial aspect of the caudal tooth. The widening should not be performed for more than 5 seconds at a time – constantly cooling the teeth and assessing the site with a dental mirror or oral endoscope.
Figure 7. Inserting a diastema widening burr into the base of a valve diastema.

Figure 8. Moving occlusally with diastema burr – taking care not to damage adjacent pulps.

References