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Recent advances in the treatment and prevention of equine peripheral caries

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ABSTRACT

Equine peripheral caries is a common condition characterized by demineralization and degradation of the clinical crown of equine cheek teeth. The condition can cause significant pain and morbidity, particularly in severe cases. Recent studies indicate that the condition is driven by environmental conditions within the mouth, as only the clinical crown of the tooth is affected (the reserve crown below the gingival margin remains unaffected). It is hypothesized that peripheral caries is driven by changes in oral pH, with risk factors for the condition including the intake of high-sugar feeds (oaten hay and feeding moderate levels of concentrate feed) and access to drinking water with an acidic pH. However, other identified risk factors include breed (Thoroughbred), limited pasture access, and concurrent dental or periodontal disease. Further studies have been able to show that affected teeth can recover from the condition if the inciting cause is removed and the unaffected reserve crown is allowed to replace the damaged clinical crown. Improvements in the condition can be observed within a few months. Signs of inactive (recovering) caries include a darker color and a smooth, hard, and reflective surface, and there is a new layer of unaffected cementum at the gingival margin, indicating that the newly erupted tooth is unaffected. Peripheral caries is a common and often overlooked condition in horses, which can often be treated with simple changes to equine management.

Introduction

Equine peripheral caries (PC) is a form of dental decay that primarily affects the outer layers (cementum) of equine cheek teeth. Affected horses are prone to dental fracture, increased dental wear, soft tissue lacerations, and periodontal disease,[1] causing significant morbidity and pain in many affected horses. Recent work[2] has shown that the condition affects the clinical crown and that the reserve crown remains largely unaffected. This indicates the condition is primarily environmental, as dental material not exposed to the oral environment remained unaffected. Therefore, if the inciting cause can be identified and removed, the condition can be resolved. In this case, as the tooth erupts the damaged clinical crown is replaced by the unaffected reserve crown and a normal clinical crown can again be achieved. This recovery has been demonstrated by Jackson et al[3] and Lundström and Birkhed.[4] This review article aims to summarize the recent literature on PC to provide clinicians with practical advice on managing and treating the condition and the secondary consequences in their patients.

Prevalence and Sequelae of Equine Peripheral Caries

Clinically PC appears as demineralization or degradation of the clinical crown. The prevalence and severity of PC are highly variable in different regions,[5] with an estimated prevalence of 6.1% in Sweden,[6] 8.2%[7] and 51.7%[5] in 2 UK studies, 58.8% in Western Australia,[8] and 91% in Scotland.[9]

The severity of the condition can be highly variable, depending on the affected tooth structures. The severity of peripheral caries is graded using the modified Honma grading scale, in which teeth are graded from 0 (no condition) to 4 (severe disease) **(Figure 1)**.[10] Milder cases (grade 1.1 and 1.2) only affect the peripheral cementum and may not cause significant pathology. However, in more severe cases (grade 3 and 4) the occlusal dentin overlying the pulps, or even the entire clinical crown can be affected,[10] which can have significant implications for the welfare of the horse. The condition affects the caudal cheek teeth more frequently and more severely than the rostral cheek teeth[5,8,9] **(Figure 2)**, which is likely due to the effects of saliva from the

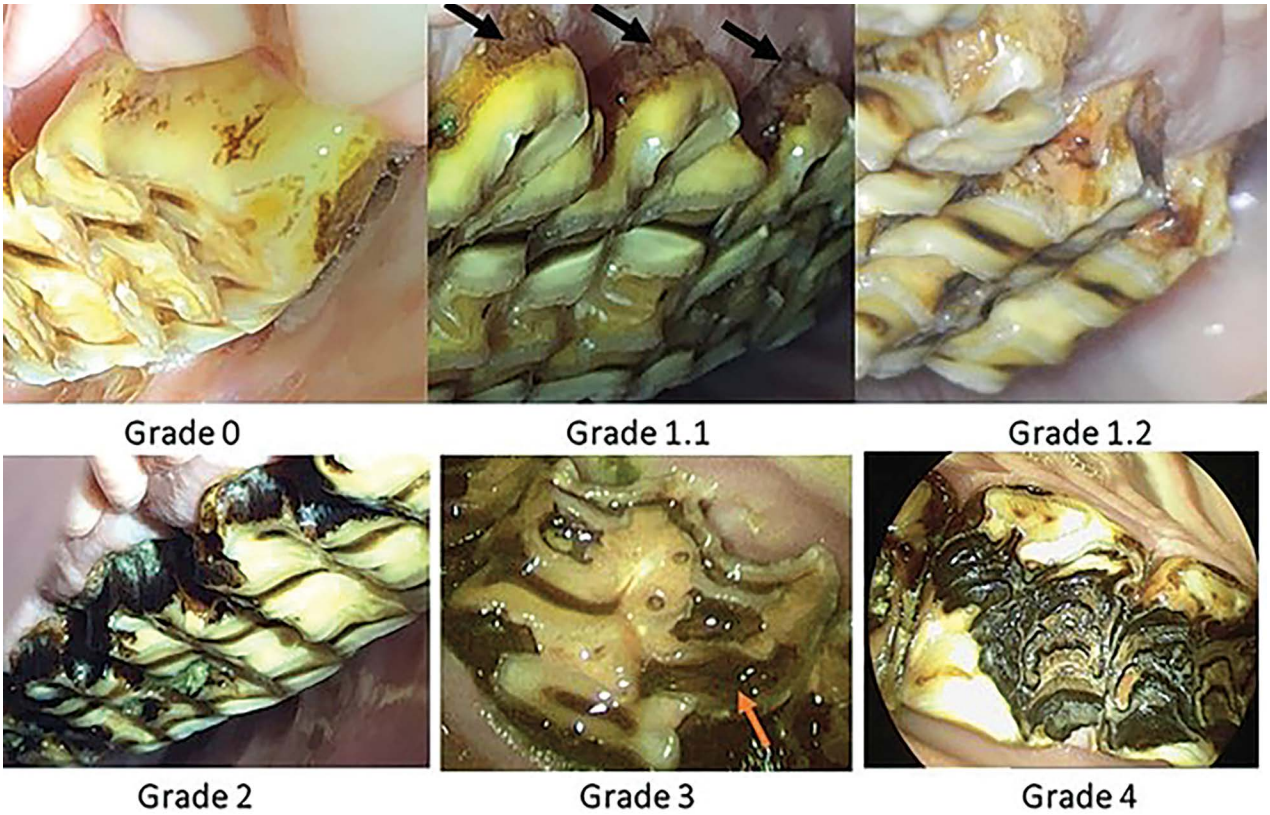

Figure 1—Grading equine PC, obtained as permitted by the publisher from Jackson K, Kelty E, Tennant, M. A new equine peripheral caries grading system: are the caries likely active or inactive? *Equine Vet J.* 2020;53:780–786.[10] Examples of the traditional peripheral caries grading system, and the definitions used in the survey; Grade 0: No peripheral caries; Grade 1.1: Pitting or partial loss of peripheral cementum; Grade 1.2: Almost total loss of peripheral cementum, some enamel is exposed; Grade 2: Involves cementum and enamel, enamel is completely exposed and discolored; Grade 3: Involves cementum, enamel and dentin; Grade 4: Loss of integrity of the tooth.

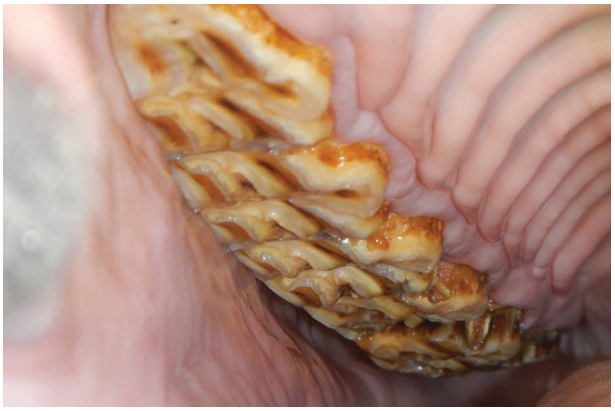

Figure 2—An example of equine peripheral caries (PC). The 106 and 107 are unaffected; there are some mild pitting lesions in the peripheral cementum at the gingival margin of the 108 and slightly deeper pitting on the 109, and the 110 and 111 are missing the peripheral cementum with the enamel being exposed.

parotid salivary duct that enters the mouth around the level of the Triadan 8s and drains rostrally, bathing the premolars in protective saliva.[8]

In the equine, the peripheral cementum contributes significantly to the size and strength of the clinical crown. Loss of this structure predisposes the teeth to dental fracture as the enamel is unsupported and the soft tissues are more susceptible to trauma from the exposed enamel.[1] Another sequela of PC is feed accumulation between the teeth.[2] With the loss of the peripheral cementum, particularly in the mandibular cheek teeth, where the peripheral cementum makes up the rectangular shape of the tooth **(Figure 3)**, loss of this dental material can create diastemata (gaps) between the teeth, which predisposes the horse to feed accumulation and periodontal disease.[2] Jackson et al[2] found that horses with moderate or severe PC (grade 2 and above) in the mandibular arcades were over 13 times more likely to have feed accumulation between the teeth than those without PC.

PC can also result in pulpitis and tooth death in severe cases,[11] particularly in cases with significant occlusal caries that affect the secondary dentin overlying the pulps. Erridge et al[1] found bacteria within the dentinal tubules in 63% of gram-stained sections, even in teeth with only superficial involvement of the peripheral cementum on gross examination. Similarly, Borkent et al[11] found histological evidence that caries had extended to the pulp, creating a tertiary dentin response in one area and infected,

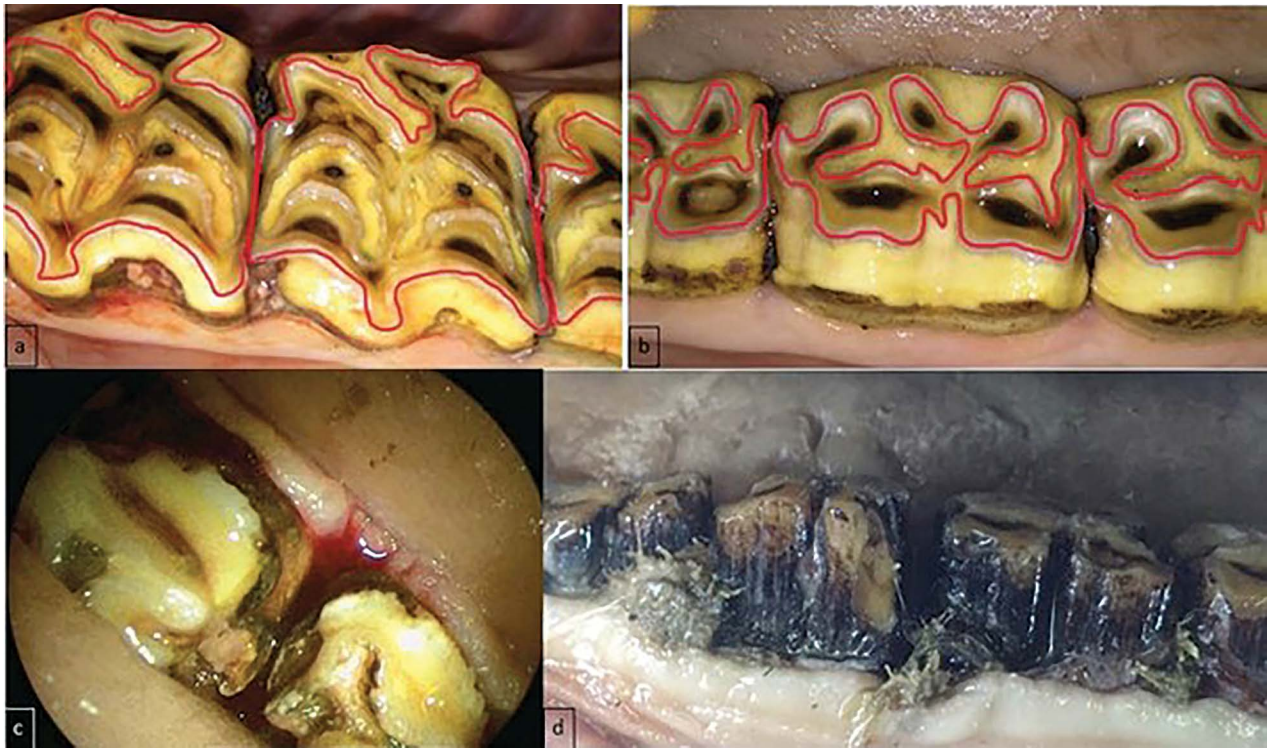

Figure 3—Cheek teeth anatomy and association with diastemata obtained as permitted by the publisher from Jackson K, Kelty E, Staszyk C, Tennant M. Peripheral caries and disease of the periodontium in Western Australian horses: an epidemiological, anatomical and histopathological assessment. *Equine Vet J.* 2019;51(5):617–624.[2] a) Maxillary cheek teeth and b) Mandibular cheek teeth with the peripheral enamel outlined in red. This highlights the close proximity of the peripheral enamel between adjacent teeth and the minimal content of peripheral cementum (pC) in the interdental position in the maxillary cheek teeth vs the more convoluted shape interdentally and the greater proportion of peripheral cementum in the interdental position in the mandibular cheek teeth. c) An endoscopic image of the interdental space between 410 and 411 (Triadan numbering system) of a 5 year old quarter horse with severe peripheral caries and gingival recession after the food remnants had been removed. The image shows a complete loss of peripheral cementum and resulting diastema. d) An abattoir specimen, mandibular molars buccal side, showing a severe case of peripheral caries with diastemata and feed accumulation (within the widened interdental spaces).

necrotic pulp tissue in other areas, indicating the significant implications of the condition in severe cases.

Pathophysiology of Dental Caries

In a normal, healthy mouth with a neutral pH, there is a dynamic equilibrium between the mineral content of the teeth and the minerals in the oral fluid.[12] At a neutral pH, the mineral component of the teeth (the hydroxyapatite) will dissolve slightly, releasing calcium, phosphate, and hydroxyl ions into the oral fluid. The oral fluid is a rich reservoir of these minerals, so under normal circumstances the oral fluid becomes supersaturated with these minerals, causing precipitation of these minerals back into the teeth.[12] This creates alternating cycles of mineral loss and gain in the hydroxyapatite crystals.[13,14]

Dental caries form when certain oral bacteria ferment dietary carbohydrates and produce an acidic by-product, reducing the pH of the biofilm around the teeth.[12,15] At an acidic pH, the phosphate ions and hydroxyl ions react with the hydrogen ions, forming complexes such as HPO_4^{2-} and H_2O leaving the oral fluid undersaturated in phosphate ions. This causes the hydroxyapatite to dissolve to resaturate the oral fluid and reestablish equilibrium **(Figure 4)**.[12] This process of demineralization occurs when the pH drops below the "critical pH," which varies for different dental materials. Enamel's critical pH is around 5.5, so it is quite resistant to decay. Dentin's critical pH is around 6.2,[16] and cementum's critical pH is only 6.7,[15] which illustrates why equine cheek teeth, with their thick covering of peripheral cementum, are so susceptible to the condition.

Many factors influence this "dynamic equilibrium" and tip the balance toward demineralization or remineralization **(Figure 5)**. These include the type of microbial flora in the biofilm, dental anatomy, saliva flow and composition, composition and structure of the teeth, diet, genetics, as well as the use of fluoride or antimicrobial agents.[16] For example, Borkent et al[17] were able to show that acidogenic and aciduric microorganisms such as streptococcus were associated with PC, indicating the importance of the biofilm in the development of caries as has been shown in other species.[12] The importance of the parotid salivary gland was demonstrated by Jackson et al[18] when they showed the devastating

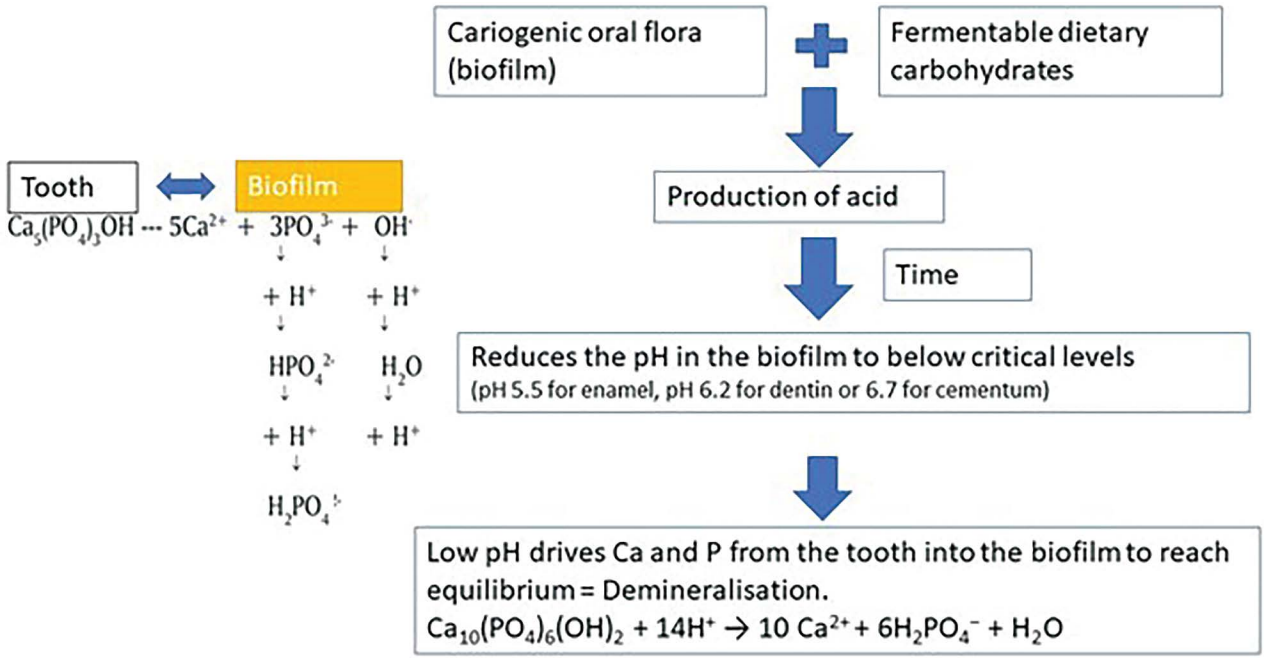

Figure 4—The pathogenesis of dental caries formation, where the normal dynamic equilibrium between the minerals in the hydroxyapatite in the teeth and the minerals in the biofilm around the teeth is disrupted. With the ingestion of fermentable dietary carbohydrates, cariogenic oral bacteria metabolize the sugars, causing the production of acids that can lead to demineralization.[12,16]

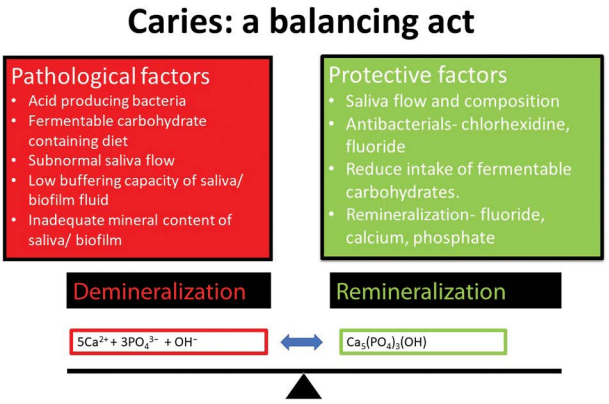

Figure 5—The development of dental caries occurs when there is an imbalance between the demineralization and remineralization cycles between the teeth and biofilm, with demineralization of the teeth predominating. Many factors influence this, and if the oral environment contains more of the pathological factors on the left, the balance will tend more toward demineralization and the development of dental caries. If the protective factors predominate, it will favor the balance toward remineralization and caries are unlikely to form.[16]

impact that the loss of saliva had on the affected side after traumatic injury to the parotid duct or gland on that side. Additionally, Lundström et al[19] were able to show a lower resting pH and a more pronounced pH drop in carious teeth compared to sound teeth after the application of 10% sucrose to the tooth surface, indicating the caries process in equine cheek teeth appears to be similar to that of human teeth.

Chronic fluorosis in horses can present similarly to PC and should be considered as a differential diagnosis in horses kept in high fluoride areas, particularly if on well water.[20] Some key differences are that fluorosis affects all teeth (incisors and canines as well) and can extend below the gingival margin, affecting the reserve crown, alveolar bone, gingiva, and periodontal soft tissues as well. In comparison, PC only affects the clinical crown exposed to the oral environment. Additionally, chronic fluorosis can be associated with hypercementosis, and dental lesions will not respond to a diet change with chronic fluorosis.[21]

In human dental caries management, the approach is shifting away from restorations, toward looking for the causal factors and focusing on preventative measures.[13] A similar approach should be taken in equine dentistry, although in many cases severe occlusal caries may benefit from restorations to protect the secondary dentin and underlying pulps as a temporary measure. However, it should be noted that with continued eruption of the tooth, the restorations will be worn away, so if the causal factors are not addressed, then the process will continue on the newly erupted crown. As such, it is of the utmost importance to discover the inciting causes and address the above influences to encourage an environment tipped toward remineralization rather than demineralization as this is most likely to provide a long-term solution to the problem.

Risk Factors

A number of risk factors have been associated with PC in horses, including diet, management, water source, breed, and concurrent dental disease.[5,8] The hay source has been found to be an important risk

factor in some areas with Jackson et al[8] finding that horses on oaten hay were almost 3 times more likely to be affected by PC than those not on oaten hay and those on meadow (grass) hay were less than half as likely to be affected. This was investigated and believed to be due to the significantly higher average water soluble carbohydrate (essentially the "sugar") levels in the oaten hay. The same study[8] found that horses with access to quality pasture (enough to reduce the amount of hay fed) for 8 or more months of the year were significantly less likely to be affected by PC, so access to grazing appeared to be protective. In fitting with this, Borkent et al[5] also found an association between feeding moderate levels of concentrate feed and an increased risk of PC, while Lundström and Birkhed[4] found that feeding regime and supplement changes, as well as environmental factors such as reduced grazing or acidic water, appeared to be risk factors for PC. Severe PC has also been associated with feeding silage with high levels of acid and with feeding high levels of processed maize feeds in Sweden,[6] so diet has been a significant factor in multiple studies. The breed of horse was also significant with Thoroughbreds being significantly more likely to be affected than Western breeds (e.g. Quarter Horses and stock horses) or warmbloods.[8]

The source of drinking water has also been found to be associated with PC. Horses on bore water (untreated groundwater) were the least likely to be affected, followed by scheme (fluoridated and treated town water supply), and then those on rainwater and dam water were significantly more likely to be affected.[8] When this was further investigated, it was found that the average pH of rainwater was 5.9 (well below the critical pH of cementum), where bore water averaged around pH 7.4 and scheme water was pH 7.9. This acidity was likely an important factor in the development of PC, as also demonstrated by Lundström and Birkhed.[4] The other interesting finding in the study of Jackson et al[8] was that the mineral contents of the water sources were very variable with the average calcium levels of bore water being almost 70 times higher than that found in rainwater. If we remember the process of caries development, and the ability for remineralization, having the teeth bathed in higher levels of these minerals may increase the opportunity for remineralization to occur and may explain why these teeth are less likely to be affected by PC.[8] It is important to note that these were average levels for these water sources in Western Australia and that even within this area the levels in the groundwater and rainwater were highly variable (the pH of the groundwater varied from pH 4.0 to pH 8.0, so some groundwater can also be highly acidic).[22] As such, testing the pH and mineral content of the horse's water source is recommended in affected cases. PC is also associated with concurrent dental abnormalities[5] and with interdental feed accumulation and periodontal disease, particularly in the mandibular arcades.[4,8]

There are indications that some horses may be more prone to the condition than others, with breed being a risk factor in some studies[6,8] and sex being a factor in another study[9] (however, sex as a risk factor has not been replicated in other studies).[5,7] Some of these findings may be attributed to differences in diet between horses of different breeds[6]; however, in the study of Jackson et al[8] (where Thoroughbreds were significantly more likely to be affected), only 3 of the 127 Thoroughbreds in the study were racehorses. As such, most were not on a high-concentrate, low-roughage diet so it is likely that certain breeds or individuals may be more susceptible to the condition.

Saliva and the parotid salivary gland and duct have also been shown to be important in the development of PC.[18] In a case study[18] of 2 horses with unilateral damage to their parotid salivary gland or duct, severe caries were observed on the same side as the injured salivary gland **(Figure 6)**, demonstrating that saliva amount and quality are factors in the development of PC. Individually variable saliva production could be another reason for individual variations in susceptibility. It is important to note, however, that many of the cases in the recovery study[3] appeared to be highly susceptible to the condition (often more affected than other horses on the property with similar diets and management) and yet they were still able to recover. As such even if horses are more susceptible to the condition, if the inciting cause is removed, recovery is still possible in most cases.[3]

Peripheral Caries Recovery

As only the exposed tooth is affected, it is possible for the condition to resolve if the inciting causal factors are modified or removed, allowing new tooth to erupt and replace the diseased tooth. Improvement in the condition can typically be observed within 6 months with new healthy tooth visible at the gingival margin, and complete resolution occurring within around 2 years.[3] While improvements are generally observed with the removal of the cause, complete resolution may not be possible in some horses, particularly senior horses with minimal reserve crown remaining, in horses with minimal/no saliva production, or where the cause cannot be determined so cannot be removed.

Jackson et al[3] followed 42 cases over many years following a diet change of removing oaten hay (typically with a high-sugar content) and found a significant improvement in PC in the molar teeth. **Figure 7** shows an example of a recovering case.[3] Lundström et al[4] were also able to show improvement or resolution in 4 cases after dietary and management changes.

Treatment and Prevention

Treatment and prevention of PC begins with investigating risk factors that can be modified, (such as diet and management factors). The diet should be assessed for items that are high in sugar or acid, in particular cereal hays, sweet feeds, and acidic water sources. Once identified, these contributing factors

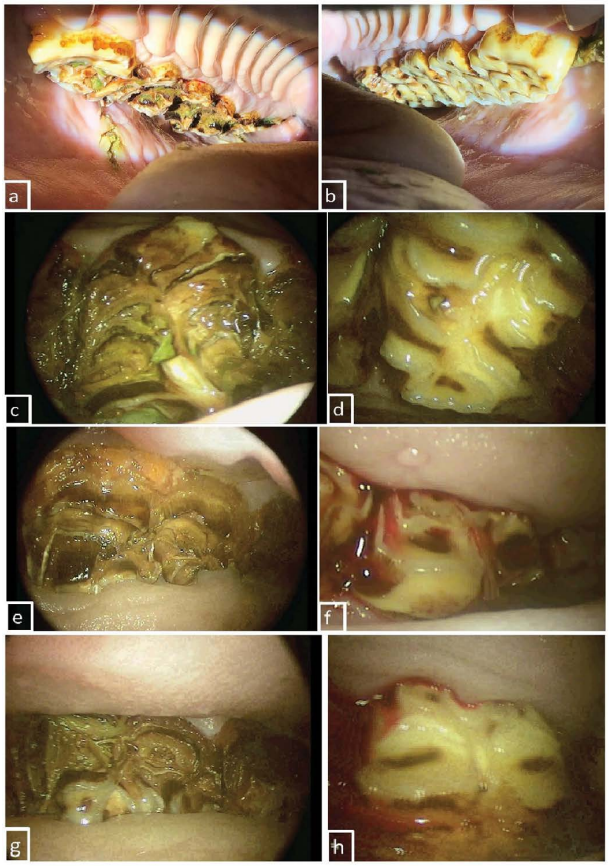

Figure 6—Obtained as permitted by the publisher from Jackson K, McConnell E, Kelty E, Tennant M. Traumatic injury to the parotid salivary gland or duct and the subsequent development of ipsilateral severe peripheral dental caries in two horses. *Equine Vet Educ.* 2019;33:e39–e44.[18] a) Oral view of the 100 arcade and b) the 200 arcade of Case 1 showing the severe peripheral caries and buccal ulceration, affecting all dental components and extending across the entire occlusal surface of the teeth 108–11 and also affecting the teeth 106 and 7, compared to the mild to moderate peripheral caries on the 200 arcade. c) Endoscopic view of the occlusal surface of the 109 and d) 209 teeth. The difference in severity is clearly evident with all dental structures on the occlusal surface of the 109 tooth being affected, compared to just the loss of peripheral cementum and some interdental material on the 209 tooth. e) Endoscopic view of the buccal aspect of the 409 and f) 309 teeth, comparing the complete loss of peripheral cementum, as well as denuded enamel and dentin on the 409 to the 309 teeth which still maintain a large percentage of the peripheral cementum and normal dental architecture. g) Endoscopic view of the 410 and h) 310 teeth showing the stark contrast between the two arcades with almost complete loss of the buccal clinical crown of the 410 tooth, compared to the 310 tooth which is missing the peripheral cementum but maintains its structural integrity.

should be removed or replaced with lower sugar and less acidic alternatives. Increasing access to pasture may also be beneficial. Addressing concurrent dental disease, in particular periodontal disease through regular dental care, is also very important.

Figure 7—Recovering peripheral caries image obtained as permitted by the publisher from Jackson K, Kelty E, Tennant M. Retrospective case review investigating the effect of replacing oaten hay with a non-cereal hay on equine peripheral caries in 42 cases. *Equine Vet J.* 2020;53(6):1105–1111.[3] Case 10, an example of dentition affected by peripheral caries recovering over a period of 15 months after a diet change. a) 100 arcade and b) 200 arcade on 28 July 2016 show active peripheral caries with plaque and feed adhering to the teeth up to the gingival margin at the initial visit. c) 100 arcade and d) 200 arcade on 7 February 2017 show no plaque or feed adhering to the teeth, shiny/glossy enamel and a thin line of unaffected newly erupted cementum at the gingival margin. e) 100 arcade and f) 200 arcade on 13 October 2017 showing substantial covering of the clinical crown with normal cementum.

These cases will often require more frequent dental visits (often every 6, or even 3 months if the periodontal disease or overgrowths are severe) to address the dental pathology.

In addition, for more severe cases with significant loss of the clinical crown from caries on the occlusal surface of the teeth, more proactive treatment may be justified to assist in protecting the underlying pulps. In these cases, the hollowed-out "cave" shape and rough, demineralized surface can lead to feed becoming trapped and adhering to the occlusal surface. The survival of micro-organisms in the oral environment is dependent on their ability to adhere to a surface, as free-floating organisms are quickly flushed away by salivary flow and swallowing.[16] Therefore, having this roughened hollow can provide a "safe haven" for pathogenic bacteria and once a mature plaque biofilm is established on the teeth, this can have a tremendous ability to rapidly metabolize any available carbohydrates,[16] allowing for rapid continued progression of the disease. Early intervention is particularly critical when the condition is affecting the occlusal surface, and therefore the secondary dentin overlying the vital pulps. In these cases, the extension of the caries into the pulps has been shown to cause pulp necrosis[11] and tooth death, so the consequences for the horse can be severe. In these cases, removal of the feed material, conservative removal of demineralized and

infected dental material with dental drills, and placement of a resin composite restoration may provide a physical barrier to prevent progression, as well as provide a smooth surface to prevent the feed from adhering to the teeth.

The use of fluoride varnishes in the treatment of equine PC has also been discussed previously.[18] In the human literature, it has been shown to increase the resistance of the tooth structure to demineralization and is recommended by the American Dental Association Council on Scientific Affairs as a caries prevention agent.[16,23] While its use in horses requires further investigation, it is relatively inexpensive, easy to apply, and has significant potential benefits, which make it a viable option in these more severe cases (although ruling out the differential diagnosis of chronic fluorosis first is advised). Requesting that the owners flush the mouth regularly with the hose may also assist with feed stagnation and periodontal disease. The addition of buffering agents to the feed and/or water source may also be of benefit, but water consumption must be monitored closely to ensure they are drinking. More work in this area is required.

Active and Inactive Caries

Improvements in PC can often be observed within a few months, and a close examination of the teeth to assess for these changes is imperative to evaluate the effectiveness of implemented treatment strategies. PC can be classified as "active" (deteriorating) or "inactive" (improving/recovering).

In the human literature signs of active caries include the following[10,16,23]:

- Lighter in color, dull, or lackluster in appearance.
- Plaque adhered to the tooth surface.
- Consistency is soft, more friable, rough on probing.
- Dental structure is cavitated and weak.
- A chalky opacity at the gingival margins.
- Painful to cold, sweet, and acidic foods.
- Progress rapidly.
- Close to the gingival margin.
- Surface zone has a lower mineral content and is heavily infiltrated with bacteria.

Signs of inactive (arrested) caries in humans include the following[16,23]:

- Darker in color.
- Smooth and shiny/reflective.
- Hard/densely mineralized surface layer.
- Nonpainful.
- Dental structure is remineralized and strong.

In equine dentition, the signs are similar; however, we have the benefit of dental eruption. As such, one of the most important signs of inactive caries is a clear shelf of unaffected cementum at the gingival margin, indicating that the newly erupted tooth is unaffected. **Figure 8** shows examples of active and inactive PC. It is important to note that horses (and humans!) can have "mixed" caries with elements of both active and arrested caries [23] (for example, unaffected cementum at the gingival margin but with some areas with plaque adhering to the teeth). This can make them more difficult to classify and may warrant closer monitoring, but if the newly erupted tooth is unaffected then it is likely on the path to recovery. Jackson et al[10] introduced a grading system to account for whether the caries were currently active or inactive and recommended adding an (a) after the traditional grade if the caries were active, or an (i) if they were inactive and to simplify and standardize the grading, the paper looked at which aspect of the tooth was most severely affected. If the third of the tooth closest to the gingival margin was the most severely affected then the caries are likely active, if the middle or occlusal third was more severely affected, with a clear shelf of unaffected cementum at the gingival margin then they were likely inactive.

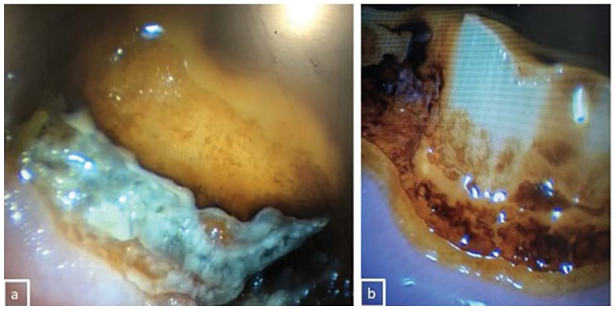

Figure 8—a) An example of active peripheral caries. There is significant plaque adhered to the tooth surface; the affected area is lighter in color; the surface is softer, weaker, and more friable with a cavitated appearance; there is a chalky appearance at the gingival margin; and the lesion extends all the way to the gingival margin. b) An example of inactive peripheral caries. They are darker in color with a hard, densely mineralized, reflective surface layer. You can see that the dental structure itself appears remineralized and strong and most importantly there is a clear shelf of unaffected cementum at the gingival margin indicating that the newly erupted tooth is unaffected.

Conclusion

Recent literature has shown we need to change our perception of PC, from horses that "just have bad teeth" to thinking of it as a treatable, preventable, environmental condition. We need to look closer at affected mouths to ascertain whether the caries are currently active or inactive, and if currently active, then the diet and management should be assessed for risk factors and eliminated if possible. More work is needed looking at the role of fluoride varnishes and feed or water additives of buffering agents to assist with cases that are difficult to manage with dietary modification alone.

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