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A study on the potential role of occlusal fissure fractures in the aetiopathogenesis of equine cheek teeth apical infections

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Keywords

Equine dentistry, fissure fractures, apical infection,

Summary

Thirty nine equine cheek teeth diagnosed as having anachoretic apical infections and also having occlusal fissure fractures, but without occlusal pulpar exposure, that had been orally extracted without causing occlusal damage and 10 control teeth were used in this study. The teeth were individually imaged by computed tomography, occlusally stained with methylene blue and visually re-examined, then sectioned subocclusally at 5 mm intervals until the fissure fractures could no longer be detected. A limited histological study was then performed on 7 apically infected and 5 control teeth. Standard computed tomography only detected 1/39 fissure fractures. Thirteen of the 39 stained teeth had subocclusal fissure fractures identified at circa 6mm beneath the surface and in nine of these 13 teeth the fissure fractures had deeper staining to a level immediately above or in a pulp horn, indicating a potential route for bacterial pulpitis. However, the extraction process, the long-term formalin storage or the processing of teeth for this study, may have caused or deepened some of the observed fissure fractures. Additionally, methylene blue may penetrate dental tissue more readily than bacteria can invade. Further studies on the potential role of fissure fractures in the aetiopathogenesis of cheek teeth apical infection are warranted.

Introduction

Cheek teeth bacterial apical infections, especially in younger horses often infect the adjacent alveolar and supporting bones causing osteomyelitis and/or paranasal sinusitis and thus are of great clinical and welfare concern.¹ A number of different routes of infection to the dental pulp and/or apical periodontal tissues have been documented in horses including; anachoresis, dental fractures including caries related midline sagittal, other idiopathic and traumatic fractures, infundibular caries, deep periodontal disease and dental dysplasia.²⁻⁶

Anachoresis, is essentially a default diagnosis made when no identifiable physical route of bacterial invasion from the oral cavity to the pulps or apex of an infected tooth is found and these infections are believed to be caused by blood or lymphatic borne infections.^{2-4, 7, 8} Anachoresis has been documented to be the most prevalent cause of equine cheek teeth apical infection, causing 51%-68% of infections.²⁻⁵ The proportion of apical infections diagnosed as being caused by anachoresis is much higher in horses than in human or small animal apical infections, both species where dental disorders have been much better studied.

Occlusal fissure fractures of cheek teeth, that are usually transversely oriented, have been recorded in 54% to 72% of horses, where they are generally believed to be innocuous.⁹⁻¹¹ One study found fissure fractures to cause 2/79 (2.5%) equine cheek teeth apical infections.⁴ The two mandibular cheek teeth fissure fractures these authors described as causing pulpar and apical infection were sagittally oriented, connecting the secondary dentin above pulp horns numbers 1 and 2.⁴ These fractures appear to be an early stage of “idiopathic” “slab” fractures,^{12,13} rather than the more common transverse pattern of fissure fractures. Casey et al. (2015) also noted the presence of occlusal fissures fractures in some apically infected teeth, without suggesting an aetiological role for them.⁵ These authors also found fissure fractures in the dentino-pulpar area of 20% of control teeth, where they were histologically identifiable as a linear defect in dentin.⁵

We hypothesise that a high proportion of apically infected equine cheek teeth diagnosed as having anachoretic infections are in fact caused by oral bacteria gaining access to the pulpar tissues from occlusal fissure fractures. The aim of this study was to examine cheek teeth with apical infections believed to have been caused by anachoresis for the presence of deep occlusal fissures that may have penetrated the pulp horns and caused pulpar and subsequent apical infection.

Materials and Methods

Apically infected teeth

Equine cheek teeth (N= 446; 272 maxillary and 174 mandibular) that were orally extracted because of apical infection at the Equine Hospital at the Royal (Dick) School of Veterinary Studies, Edinburgh and which had since been stored in 10% buffered formalin solution for 6 months to 15 years were utilised. The teeth were grossly examined, including following transverse sectioning towards their mid-crown and apical regions, that along with clinical records were used to determine the most likely route of the apical infection. The cause of apical infection was diagnosed as: anachoresis (i.e. no obvious physical route of infection from the oral cavity to the apex) (n=204; 45.7%; 116 maxillary, 88 mandibular); idiopathic or infundibular caries related fractures (n=132; 29.6%; 96 maxillary, 36 mandibular); deep periodontal disease (n=62; 13.9%; 20 maxillary, 42

mandibular); dental dysplasia, including 4 patent infundibulae (n= 33; 7.4%; 21 maxillary, 8 mandibular); infundibular caries (n=11; 2.5%, 11 maxillary) and (due to extensive tooth damage) to undetermined causes (n=4; 0.9%; 1 maxillary, 3 mandibular).

The more occlusally located periodontal membranes and gingiva in the 204 teeth diagnosed with anachoresis appeared to have been intact prior to extraction as judged by the absence of additional staining of the peripheral cementum (albeit some may have been removed by formalin storage) or of areas of abnormal smoothness or loss of peripheral cementum and the absence of evidence of pre-extraction clinical crown damage. At least one pulp was occlusally exposed in 56/204 (27.5%) of these teeth, indicating they had a more chronic infection and/or thinner subocclusal secondary dentin. These 56 teeth were unsuitable for this study as were 82 other teeth that had extensive iatrogenic clinical crown damage, including of the occlusal surface, primarily caused by extraction forceps. The 66 remaining cheek teeth, included 25 maxillary and 41 mandibular cheek teeth from horses of mean age 8.4 years (range 3-16 years) with a mean dental age (age of teeth since they first erupted) of 5.8 years (range 2-12 years). All had fully intact occlusal surfaces with just some localised forceps damage to the clinical crown periphery. Examination of their occlusal aspects showed 39 of these 66 teeth to have occlusal fissure fractures and these 39 teeth were selected for further study.

Control Teeth

Four cadaver heads from horses that had been euthanized or died from non-dental related disorders were found by direct visual examination of their teeth to have occlusal fissure fractures affecting at least one cheek tooth. Ten teeth (5 maxillary and 5 mandibular) of mean dental age 10.3 years (range 6-22 years) with transverse fissure fractures affecting secondary dentin were extracted intact from these heads using a stainless steel bone chisel and mallet and were immediately used as controls for occlusal staining and histological studies.

Computed tomography

The 39 apically infected teeth with fissure fractures were individually imaged with a multi-slice scanner (Siemens® SOMATOM Volume Zoom, Siemens, Munich, Germany) using a 512 x 512 matrix, 120 kV, 300 mA, at a slice thickness of 0.5 mm with 0.5 mm overlap. Bone window CT data were transferred as DICOM images to Horos® imaging software.

Methylene blue staining of occlusal aspects of intact teeth

A pilot study where the occlusal aspects of control equine cheek teeth with fissure fractures were submerged in methylene blue for > 24 hours caused such intense dark staining of all of the clinical crown calcified tissues that fissure fractures that were visible prior to staining, were now not detectable. Subsequently, all 39 apically infected cheek teeth with occlusal fissure fractures were stained with methylene blue using modification of a protocol used to detect fine enamel fissure fractures in extracted human teeth.¹⁴ The occlusal surfaces of teeth were submerged in 2% methylene blue for 30 seconds, dried with paper towels and allowed to fully dry at room

temperature before photography. The apically infected and control teeth were then stored in formalin solution for a couple of days until sectioned, in order to prevent iatrogenic cracks from developing due to dehydration of cementum or dentin.

Sectioning

The teeth were then transversely sectioned subocclusally parallel to the occlusal surface (to allow for their variable occlusal angulation) at circa 5 mm thick intervals using a water-cooled tile saw with a 0.8-mm thick, continuous-rim, diamond tipped blade (Malvern Lapidary, Worcester, UK). Examination of these sections allowed assessment of the depth of the fissure fractures and for possible connections between fissure fractures and pulp horns. Further sections up to 5mm in thickness were made on each tooth until no further staining was detected or the pulp horns were reached.

Pilot histopathological study

Transverse 5mm thick subocclusal dental sections (as described above) of 7 apically infected cheek teeth (3 maxillary and 4 mandibular), and 5 control cheek teeth (3 maxillary and 2 mandibular) with transversely oriented fissure fractures (but otherwise normal) were decalcified and fixed as described.^{15,16} Histological slides (1-2 per tooth) were stained with H&E and examined by light microscopy.

Results

Fissure fracture patterns in apically infected teeth

The long-term formalin storage of some of the apically infected teeth had stained them a light tan colour which made direct visual identification of occlusal fissure fractures more difficult (Fig. 1) than in unfixed control teeth. Fissure fractures were more apparent in all 39 apically infected teeth on direct examination with a magnifying eyepiece (Fig. 1). Fissure fractures identified in this study all had the same appearance, i.e. hairline fractures of the occlusal surface with one end of the fracture involving secondary dentin (overlying the site of a pulp horn) and the other extending peripherally to enamel and or even to peripheral cementum. In the 17 mandibular cheek teeth, 28 detected fissure fractures ran transversely through pulp horns: no. 1 (n=10); no. 2 (n=12); no. 3 (n=2); no. 4 (n=2); no. 5; (n=1), i.e. were termed type 1a fissure fractures using the Pollaris et al.(2018) classification.¹¹ One long, sagittally oriented fracture in a mandibular tooth connected the secondary dentin overlying pulp horns Nos. 1 and 2, i.e. a Type 1b fissure fracture.¹¹ In the 22 maxillary teeth, 28 fissure fractures ran

transversely through pulp horns: no.1 (n=4); no. 2 (n=6); no. 3 (n=8); no. 4 (n=5); no. 5 (n=5) (all type 1a fractures).

Using the above-noted standard CT imaging technique, fissure fractures were identified in transverse images of only one (a mandibular) of the 39 teeth (Fig. 2), but not on longitudinal sections where in contrast, definitive CT evidence of pulpar and apical disease was usually very apparent (Fig. 3).

Following staining (Fig. 4), the fissure fractures became more prominent occlusally in only 18/39 (46%) of the apically infected teeth (Fig. 5), with deep staining of the surrounding calcified dental tissues in the other 21 teeth sometimes obscuring the original fissure fracture. The mean occlusal length of stained fissures fractures was 12 mm (range 5-26 mm). Stained fissure fractures were identified below the first subocclusal section (circa 6mm subocclusally) in 13/39 (33%) infected teeth (Figs. 6, 7), but never below the second subocclusal section (circa 12mm deep). The most prominent methylene blue staining occurred in irregular secondary dentin (over the central aspect of the former pulp horns). In 9/13 teeth (6 maxillary and 3 mandibular) with subocclusal detected fissure fractures, the staining occurred directly above (n=8) or within the adjacent pulp horn (n=1).

Histological findings

Histologically, a layer of darker blue staining was present between primary and secondary dentin around every pulp horn site, in both control and apically infected teeth, whether adjacent to a fissure fracture or not, as seen in Fig. 8. This slide shows an area of dentin adjacent to a fissure fracture, where the longitudinally sectioned dentinal tubules contain rod-like bacteria. The fissure fractures histologically identified in 11/12 (infected and control) teeth contained blue stained plaque and or vegetable material. (Figs. 9, 10). However, the one histological slide in an apically infected tooth that showed a direct connection of a fissure fracture to an infected pulp contained no plaque/vegetable material nor any necrotic pulp debris more axially in the fracture, and so it appeared like a recent, possibly iatrogenic fracture (Fig. 11). Histological examination of one control tooth with a fissure fracture (that was of similar appearance to other control teeth fractures) showed dentin adjacent to the fissure fracture to contain pale blue, rod-like bacteria (pioneer organisms) in its dentinal tubules, some which were markedly disrupted (Fig.12).

TABLE 1: Equine Cheek Teeth Fissure Classification System of Pollaris et al (2018) ¹¹

Type 1	Fissures that involve the secondary dentin overlying pulp canals
	Type 1a: Fissure orientation is perpendicular to the surrounding enamel fold, variably involving the adjacent enamel or even the peripheral cementum
	Type 1b: Fissure orientation is sagittal (rostro-caudal), sometimes connecting adjacent pulp horns
Type 2	Fissures that never involve secondary dentin
	In primary dentin they run parallel to the enamel folds, can also occur in enamel or peripheral cementum

Discussion

The prevalence of fissure fractures (66.6%) in apically infected cheek teeth diagnosed with anachoresis in this study is very similar to the prevalence of fissure fractures in the general equine population of 54-72%. ⁹⁻¹¹ The location and position of the fissure fractures within the teeth (mainly buccally located in mandibular and palatally located in maxillary cheek teeth) as previously described, ⁹⁻¹¹ indicates a link with equine masticatory forces and this topic has been recently reviewed. ¹¹ In apically infected teeth all fissure fractures except one, were transversely oriented and involved primary and secondary dentin (i.e. were Pollaris et al Type 1a fractures), with only one sagittally directed (Type 1b) fissure fracture identified (in a mandibular tooth). ¹¹

Ramzan and Palmer (2010) found that 6% of cheek teeth with identified disorders had occlusal fissure fractures on oral endoscopic examination. However, the depth of these fractures could not be determined in these clinical cases to assess the possible relationship of the fissure fractures with the intercurrent dental disorders. ¹⁰ These authors suggested that fissure fractures are unlikely to cause dental apical infection, but could possibly predispose maxillary cheek teeth to develop “slab fractures” (through 1st and 2nd pulp horns). Van den Enden and Dixon (2008) found that fissure fractures caused 2.5% of apical infections in equine cheek teeth. ⁴ The type of fracture they described, i.e. mandibular cheek teeth with sagittal fractures through pulp horns Nos. 1 and 2, with necrotic pulps beneath the occlusal surface, was also found in one tooth in the current study, that also had underlying necrotic pulps. Dacre et al (2007) showed that 25% of sagittally oriented “idiopathic” cheek teeth fractures had decreased secondary dentin thickness, indicating the presence of long-term pulpar damage, i.e. that these were pathological dental fractures. ¹² However, the longer term endodontic infection may have been initially caused by oral bacteria gaining pulpar exposure via fissure fractures, with these fissure fractures later becoming gross fractures. These sagittal fissure

fractures may have the same aetiology as the more common, transversely oriented fissure fractures, but are more likely to widen and cause endodontic disease because their fracture plane is parallel with the main equine masticatory forces.

High-resolution computed tomography (image slices of ≤ 1 mm) as performed in the current study provides high spatial resolution and less partial volume artefacts to allow detection of subtle lesions.^{5, 17} However, CT imaging identified fissure fractures in only 1/39 teeth with visible occlusal fractures showing its lack of sensitivity for this type of lesion. Micro-computed tomography as used by Suske et al (2016) would be a more sensitive imaging technique for detecting these subtle lesions.^{6, 18}

In suspect human teeth where the crown enamel appears intact on visual examination, dyes are used to detect enamel micro-cracks which could allow deeper bacterial access.^{14, 19} Detection of these early-stage fissure fractures (sometimes termed “craze lines”) may allow their early treatment and thus prevent enamel and dentinal caries and possible pulpar infection from developing.²⁰ Dyes are also used on extracted human teeth to detect minute enamel defects.¹⁴ The multiple layers of the three different calcified dental tissue on the equine occlusal surface makes the use of methylene blue dye for fissure fracture detection more difficult. The secondary dentin of equine teeth absorbs more exogenous colours (i.e. as occurs in “dental stars”) than the other calcified dental tissues and this may explain its increased staining with methylene blue. Standardised techniques need to be developed for diagnostic equine dental staining.

The fact that the secondary dentin immediately overlying pulp horns or the occlusal aspect of the underlying pulp horns of 13/39 apically infected teeth were stained with methylene blue suggests that the fissure fractures present in these teeth could allow oral bacteria to penetrate from the occlusal surface into the occlusal aspects of the pulps. However, it is possible that particles of methylene blue dye, can penetrate dentin more readily than bacteria, as seen in the pilot study where the complete crowns of control teeth became intensely stained dark blue. Additionally, oral extraction of cheek teeth is a prolonged (can be many hours long) and forceful procedure that could cause new fissure fractures or deepen existing fractures and so the deep dentinal staining noted in some of these 13 teeth could be artefactual. The effect of the long term formalin storage of the infected teeth is unknown, but dry storage would have allowed dehydration of dentin and cementum and subsequent development of micro-fractures of all three calcified dental tissues, that would have prevented this study from being performed.

Only a limited number of slides of apically infected and control cheek teeth with fissure fractures were examined in this pilot histological study. This study found darker, blue-staining present at the boundary between primary and secondary dentin in all samples, as previously described and this innocuous staining is likely due to H&E staining. Most fissure fractures were filled with amorphous, plaque-like material sometimes also containing vegetable material and pollens all which was stained pale blue by methylene blue dye and/or haematoxylin and eosin. In only one tooth, was a fissure fracture observed to directly connect with an infected pulp. However, as this fracture had particularly sharp margins, contained no plaque or food material, nor necrotic pulp material more axially (Fig. 11) it may have been caused by or deepened iatrogenically during dental extraction, tooth sectioning or histological slide preparation.

Histologically, the dentinal tubules in one apically infected (Fig 8) and one control cheek tooth (Fig. 12), had focal, pale blue, rod-shaped, bacteria-like structures indicating that fissure fractures can allow a direct route of infection from the oral cavity deep into the adjacent dentin and then possibly on to the pulp. This localised, histologically identified dentinal infection was not grossly visible in either tooth. There was no evidence of pulpar infection in the control tooth, however if this horse had lived, the affected tooth may well have developed pulpar infection later. This chance histological finding in a control tooth provides further evidence that fissure fractures are a potential route of pulpar infection.

Conclusions

Following staining with methylene blue, some occlusal fissure fractures in cheek teeth diagnosed with anachoretic apical infection had staining extending into secondary dentin directly above or in the underlying pulp horn, indicating a possible route for oral bacteria to cause pulpar and thus apical infection of these teeth. However, it is possible that extraction damage may have caused or extended some of these fractures towards the pulp horns. Additionally, oral bacteria may not penetrate as readily as the methylene blue dye did in these studies. Nevertheless, fissure fractures should be considered as a possible route of pulpar/apical infection in equine cheek teeth. Further studies on diagnostic occlusal cheek teeth staining and dental histology is warranted in cheek teeth with fissure fractures.

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Figure legends

Fig. 1. Tan-coloured occlusal surface of an apically infected maxillary cheek tooth with a fissure fracture involving the 4th pulp horn (arrow) that was evident on direct visual assessment (left). The same fissure fracture (arrow) is more readily detected under a bright light and magnifying eyepiece (right). The transversely oriented fissure fracture runs from the secondary dentin overlying pulp horn No.4 in a palatal direction through the peripheral enamel.

Fig 2. Longitudinal computed tomographic image of a maxillary cheek tooth with anachoretic apical infection and a fissure fracture. The fissure fracture was not detected in this image. Extensive hypoattenuation (gas) is present in the left pulp horn and common pulp chamber, along with apical cemental remodelling and thickened periodontal membranes.

Fig. 3. A transverse computed tomographic image of a mandibular cheek tooth with a fissure fracture present in the peripheral enamel and dentin on the buccal aspect of pulp horn No. 2 (arrow).

Fig. 4. Mandibular cheek teeth extracted because of apical infection (note granuloma on one root). Limited extraction damage is present on the clinical crown above the intact periodontal membranes. Note the methylene blue staining of some of the clinical crown calcified tissues.

Fig. 5. The occlusal surface of an apically infected mandibular cheek tooth stained with methylene blue that has highlighted a fissure fracture (arrow) running transversely from its buccal aspect through peripheral cementum, enamel, primary and secondary dentin overlying pulp horn No. 1.

Fig.6. Subocclusal section of an apically infected mandibular cheek tooth with two methylene blue stained fissures fractures on its buccal aspect (arrows) that extend into the darkly stained secondary dentin overlying pulps horns Nos. 1 and 2. The other three pulp horns (without adjacent fissure fractures) do not have darkly stained secondary dentin in this section.

Fig 7. A subocclusal, transverse section of a maxillary cheek tooth with two transversely oriented fissure fractures (arrows) on its palatal aspect, extending from peripheral cementum to secondary dentin overlying pulp horns Nos. 3 and 5, with deep staining of the secondary and lighter staining of the primary dentin. Secondary dentin overlying the 2nd pulp horn, which does not have an adjacent fissure fracture (at this level) is also darkly stained.

Fig. 8. Microscopic image an apically infected cheek tooth subocclusal section adjacent to a fissure fracture, with primary dentin located peripherally and a darker blue line (arrows) separating it from the more centrally placed secondary dentin. This feature was present around every pulp horn in every tooth and was considered insignificant. There are some blue-stained, rod-like bacteria in some primary dentin tubules that are longitudinally sectioned. On transverse sections, they completely filled the dentinal tubules.

Fig 9. Microscopic image of a sub-occlusal fissure fracture through transversely sectioned primary dentin tubules in an apically infected tooth. This fracture contains amorphous, plaque-like material and plant debris including pollens indicating a connection of this fissure fracture to the oral cavity. The surrounding dentin looks normal.

Fig. 10. Microscopic image of a control equine cheek tooth with two separate fissure fracture (arrows) partially filled with amorphous blue material, likely plaque stained by methylene blue and or H&E. The fissure fractures extends from both sides of the slide to secondary dentin overlying a former pulp horn. There is no sign of infection in this secondary dentin.

Fig. 11. Micrograph of an apically infected cheek tooth with an empty fissure fracture (with sharp margins) extending through primary and secondary dentin (arrows) into the pulp horn that contains abnormal pulp tissue due to pulpar and apical infection.

Fig.12. This subocclusal histological image shows a fissure fracture in the dentin of a control cheek tooth that contains blue-stained plaque-like material (green arrows). The fracture is wider and has and surrounded by disrupted dentin at the right side of slide. Many adjacent and distant dentinal tubules contain stained rod-like bacteria.

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