

Lack of Cementum in Infundibulum (Hypocementosis) in Equine Cheek Teeth

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Abstract

The aetiology for lack of Cementum in infundibulum in equine cheek teeth is primarily unknown for the non-traumatic cases but affect first of all 108, 109, 208 and 209.

The aim of the study was to elucidate reasons for affection these teeth. Examination of 64 pulsed or extracted and longitudinal sectioned cheek teeth showed that a considerable amount of the clinically normal cheek teeth had straw, forage debris and air in the infundibula. This material suggests that abnormalities in cementogenesis of the infundibulum and food impaction can be an internal route for infection via an incompletely cementum filled infundibulum. The exact time for formation of the apex of the infundibulum is unknown, but it seems to be after eruption of the maxillary cheek teeth, as the infundibulum is not filled totally with cement, and pressure on the root shows blood on the surface of the trans-sectioned infundibulum. Histological examination shows forage material embedded in histologically normal cementum as a sign of connection through the occlusion surface into the infundibulum during cement production.

This is in agreement with very recent research on the blood supply during development of the infundibulum which indicate that blood supply through the infundibular apex do continue after eruption of the teeth.

Keywords: Horse; Equine Dentistry; Equine Cheek Teeth; Infundibulum Development; Hypocementosis; Odontogenesis;

Introduction

Periapical infection is a common problem in the cheek teeth in the horse [1]. Lack of infundibular cement on the occlusal surface is observed before but generally it has been accepted, that a major reason for destruction of the infundibular cement in the maxillary cheek teeth is acids formed during bacterial fermentation of food particles [1,2]. Periapical infection is most often seen in 108,109,208,209 [1,3,4]. Traditionally, it is believed that apical infection of the teeth in horses begins with a gingival periodontal infection around these teeth or via the bloodstream [3]. Infundibular necrosis is a bacteria-induced disease characterized by demineralization of larger portions of the tooth [4,5]. The possibility of infection via an incompletely formed infundibulum, however, cannot be ruled out [5,6,7]. The

disease often manifests clinically with unilateral, malodorous nasal discharge, soft tissue swelling and/or malodour from the mouth and confirmed by radiography or ultrasonography or computed tomography (CT) scanning [8,9,10].

The definition of Caries is a molecular decay or death of bone/teeth. This study supports previous findings of hypocementosis in the infundibulum. The lack of cement in the infundibulum may be one of the reasons for infundibular infection and/or necrosis, and therefore "hypocementosis" is the proper name for the type of lack of cement in the infundibulum described in this material.

Materials and Methods

Teeth from 35 cadavers, 1 to 30 years old (mean age 6.5 years) were collected from horses donated to the Copenhagen Zoo and the University of Copenhagen. The cause of death was due to medical disorders unrelated to dental or oral diseases. Additionally, cheek teeth from clinical cases of apical infection (n=7), supernumerary teeth (n=2), and fracture (n=1) were included. A clinical inspection of the 35 cadavers checked for any maxillary cheek teeth abnormalities.

Thirty-two horses had maxillary cheek teeth showing lack of cementum and/or black stained cementum in the infundibulum in 108,109,208,209. If it was possible to insert an 18-gauge needle into the infundibular defect, the tooth was carefully extracted from the alveolar bone using a small surgical chisel on the palatal surface of the maxilla. The teeth were sectioned longitudinally and/or transversally by using a hacksaw (1.5 mm thick).

The horses with clinical signs of dental disease were radiographed. Three examples of horses with newly erupted teeth (13 -14 months of age) had CT scan, and one of these had a Magnetic Resonance Imaging (MRI) examination too.

The 109 from the cadavers of the horses with clinical signs of dental disease were carefully extracted and sectioned transversally midway on the length. Applying digital pressure on the apical area resulted in blood exiting from the infundibular cut-surface.

Ten diseased but clinical symptom-free teeth were radiographed after extraction to visualize the infundibulum.

Specimens from 5 teeth (109, 209 from horses > 5 years of age) were prepared and decalcified before cutting and staining with haematoxylin and eosin (H&E) for histopathology. The Faculty of Odontology, Dept. for Oral Pathology and Medicine, University of Copenhagen performed the histopathological examinations (Jesper Reibel, prof., Dr. odont).

Results

In cases of clinical infection in the pulp area, the pulp showed dramatic changes in the extracted and sagittal sectioned teeth. Foreign bodies, including recognisable fibrous forage material appeared in the central channel of the infundibulum, extending even to the apex (Figure 1). The diameter of the channel "I" was broader at the apex compared to the occlusal area. In the apex, some reddish staining of the cementum was observed.

In the horses clinically imaged, reactive periapical bone was observed on radiographs, and sinusitis could be seen on radiographs as a sequela to the apical infection.

In the rest of the teeth an irregular open central channel were very often found in the infundibular cementum on the occlusal view - with the surrounding cement being stained black. When longitudinal sectioned, the opening could be followed into the former central vascular channel. This channel was often filled with straw/forage and/or geosediment and air along an irregular central channel (Figure 1,2,3). The opening varied in size, sometimes being quite narrow (1 - 2 mm) and sometimes being very broad (4-6 mm) (Figure 1,4). Often, it could be followed along the teeth towards the apex of the infundibulum - sometimes ending in a diverticulum (6 -7 mm in diameter) and occasionally with reddish staining of the surrounding cement (Figure 3).



Figure 1: (109) after pulsed and sectioned longitudinally from a 5-year-old horse with apical infection and sinusitis, showed typical changes on radiograph. A relatively broad infundibular channel extending from the occlusal surface containing forage and air. Toward the apex the channel (I) becomes very wide in diameter, and the surrounding cement is yellow/ reddish coloured. When the tooth was repulsed, the apex of the tooth was partly destroyed. The presence of reddish coloured cement could indicate inflammation of the pulp.



Figure 2: (109) after extraction and longitudinally sectioned from a 5-year-old horse without clinical dental signs. A thin infundibular channel from the occlusal surface extends towards the apex where it ends in a large irregular opening (I). The channel and the opening are filled with green coloured food and straw.



Figure 3: (208) after extraction and sectioned longitudinally from a 5-year-old horse without clinical dental signs. In the middle of both the mesial and the rostral infundibula, an irregular, quite thin infundibular channel (I) broadens pronouncedly at the apex containing forage debris and air.

In the partly erupted 109 teeth of 3 different foals the central channel had a diameter of up to 8 mm. It was filled with highly vascularized connective tissue (Figure 5) producing cement to fill out the infundibular lumen. Hypodermic needles could be pressed into some of the infundibula of 108 and 107 indicating lack of cement filling in these infundibula.

On radiographic and CT or MRI imaging, the partly air-filled infundibulum could be followed to its apex in newly erupted teeth, showing an air/forage filled infundibular apex-cavity next to the root area (Figure 6).

The extracted, partly erupted 109 and 209 teeth of these young foals (Figure 5) were cross-sectioned and blood could extrude from the transacted surface, when pressure was applied to the apical end of the teeth (Figure 7,8).

Histological examination supported the macroscopic anatomical findings and in many cases the presence of foreign bodies embedded in the cement (Figure 9), bacteria (Figure 10), and lack of cementum could be found.

The histopathological examination revealed foreign body material not only in the centre of the hypoplastic cementum of the infundibular “vascular” channel, but also inside the cementum-filled portions of the infundibulum (Figure 9). The cementum as such was normal in structure and there were no signs of caries at the cement surface of the pieces examined.



Figure 4: (109) after extraction and longitudinally sectioned from an 8-year-old horse without clinical dental signs. There is a broader, but not very deep opening in both infundibulae (I) at the occlusal surface. The defects are filled with straw and food remnants. This is a typical case of infundibular caries.



Figure 5: Occlusal surface of left maxillary cheek teeth-arcade of a 13-month-old colt without any clinical signs of dental diseases. (109) marked “M1” is the partly erupted. The inserted needles illustrate the soft openings of the infundibulae at the occlusion surface of 107, 108, 109.

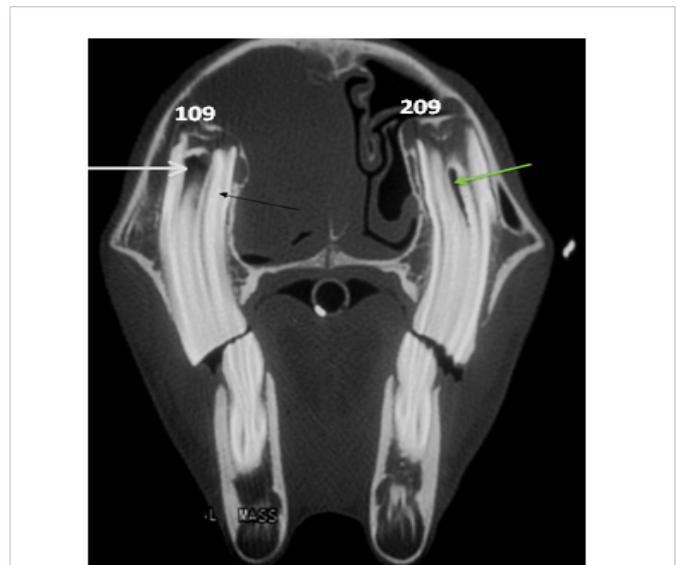


Figure 6: CT image of a newly erupted cheek tooth (109) in a horse where an open apical infundibulum is visualized (white arrow) as well as a narrow pulp (black arrow). The horse has a big mass-effecting soft tissue in the nasal cavity unrelated to the teeth. There is air in the infundibular apex in 209 (green arrow).



Figure 7: The extracted tooth (109) from Figure 5 was transversely sectioned in its mid region. Two infundibular channels (white arrows) are visible on the cut surface, and the five pulp-channels are the reddish areas

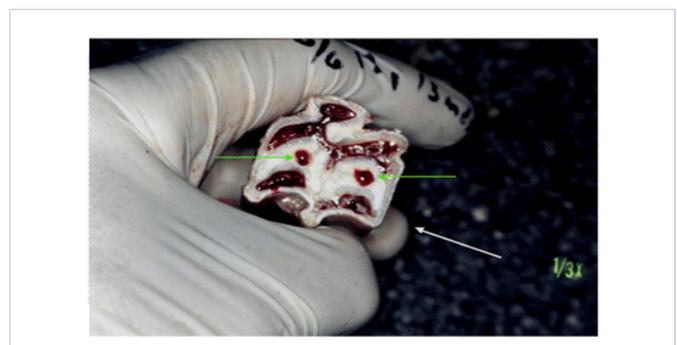


Figure 8: TA finger (thick arrow) is pressing upon the root area of the same tooth as in Figure 7, and now blood is more clearly shown in the blood-filled infundibula (thin arrow) than in Figure 7. This picture indicates that the infundibular apex has not mineralized and that there be some blood supply from the original root area through the infundibular apex.

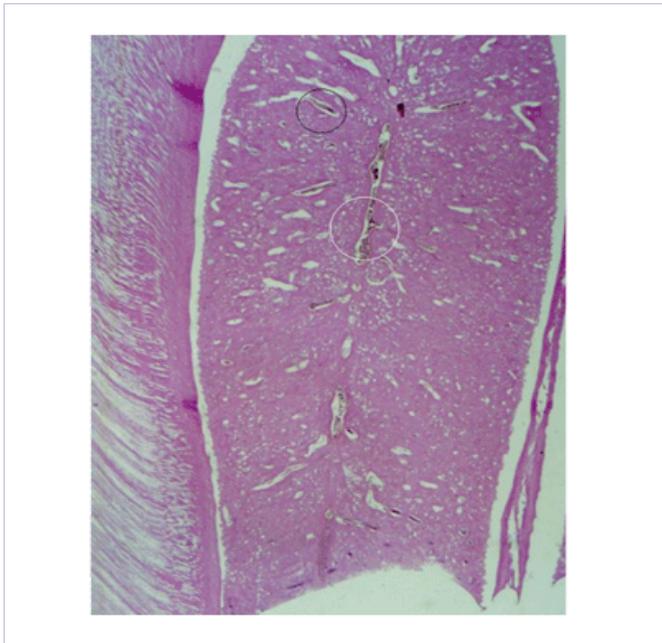


Figure 9: Histomicrograph of a stained decalcified section of the apical part of the infundibular cement from Figure 1. The small remnant of the infundibular channel is seen (white circle) and more perpendicular straw-enclosed in the cement (black circle). The cementum is regular and normal in structure. Stained Haematoxylin and Eosin (H&E).

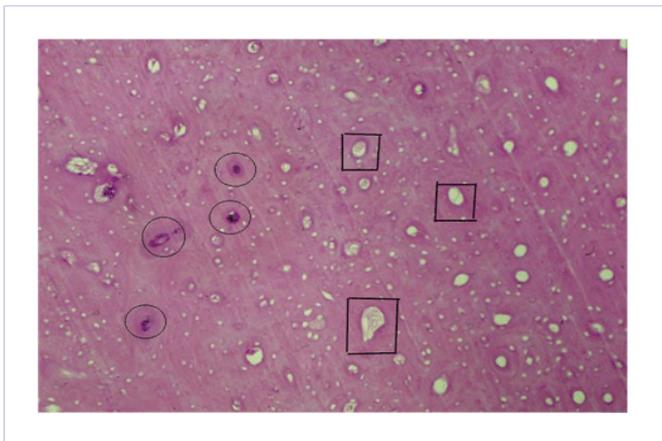


Figure 10: Higher magnification of Figure 9. Straw embedded in the cement (squared boxes) and areas with bacteria (round boxes) are present in the otherwise normal infundibular cementum. Stained Haematoxylin and Eosin (H&E).

Table 1 shows the diameter of the longitudinal sectioned 108,208 and 109,209 teeth measured at the occlusal and the apical end of the infundibulum. Twenty percent of the 108/208 (12/62) had a diameter more than 4 mm without cement in the infundibulum. Only 13 percent (8/64) of 109/209 had a diameter of the hypocementosis infundibulum greater than 4 mm. Two-thirds of all 108/208 teeth had not fully developed cement in the infundibulum and half of the 109/209 teeth had lack of cement at the apical end of infundibulum.

Table 1: The size of the infundibular diameter in maxillary cheek teeth 108/208 and 109/209 from 32 horses (64 teeth all together) at the occlusal area (OA) and at the apical area (IA) (3 – 10 mm from the apex). Only 12 of all 108/208 had fully developed cement in their infundibulum (OA) and half of the 109/209 had lack of cementum at the apical end of infundibulum (IA).

Infundibular channel diameter (OA)	108/208	109/209
0	12	14
< 2 mm	22	24
2 – 4 mm	18	14
> 4 mm	12	12
Root area Infundibulum (IA)		
0	28	32
< 2 mm	8	10
2 – 4 mm	16	14
> 4 mm	12	8

Discussion

Kilic describes the ultrastructure of the teeth in the horse. A developmental assessment of the infundibulum in the cheek teeth is described in earlier studies, some using advanced imaging-technique as CT and MRI [3,6,7,11]. An extensive description of the forage-remnants in the infundibulum is presented for the first time in this study. The aetiopathogenesis of both infundibular necrosis/caries and periapical lesions in the horse teeth remains unclear. However, cases with normal uninfected pulp may be a result of lack of cement in the infundibulum extending to the apex of the infundibulum [5,12]. Fractures of the teeth are also seen in connection with severe caries destruction of the infundibular cement and enamel. One study using CT showed infundibular hypocementosis in more than 70% of diseased cheek teeth [7]. These findings are in agreement with several other studies [6,13,14,15], but according to the present material, the aetiology appears to be more a question of lack of cement production, than acid destruction of the cementum. The infectious material is already present when the cement is produced, as bacteria and fibrous forage material is found embedded in the cementum – not only on the surface - as seen in the histological findings (figure 9).

The procedure of sectioning the teeth of 13-months old foals demonstrates that the infundibular cavity can be open at the developmental stage when the 109/209 begins to erupt and wear (Figure 5). When the eruption is completed, the nutrition to the secondary cement in the infundibulum from the dental channel via the occlusal aspect must cease in order to close the occlusion surface. If it does not cease, hypocementosis will appear. In another study, accessory blood supply to the infundibula were actually found [6].

Though it has been pointed out; that no more cement can be deposited in infundibulum following tooth eruption, this theory is probably not valid any longer. Figures 1, 2, 3 shows

that there sometimes may be some forage material in the apex of the infundibulum before the cementum fills up with cement. This theory supports the infundibulum development according to Dacre, who describe a possible remnant of the apical vascular channel [5,6,14]. The exact time for this closure of the infundibulum at the occlusal surface is not proven by the current study, but it seems to be after eruption of the cheek teeth in the 4 young foals in this material.

The histological findings have been confirmed in another study [11]. The old vessel channels contains grass-fibre, forage material, and some materials are embedded in the cement outside the central channel of the infundibulum (Figure 9,10). Therefore, there must have been an open central channel in the infundibulum as the forage material was present before and during the cement was formed.

Figures 1 and 2 shows a slim tract from the occlusal surface to the apex of one and two infundibulum in different teeth. This is not a typical caries cavity conformation as showed in figure 4 of an 8 years old horse.

A study [3] has shown that up to 80 % of older horses to some extent may be affected by caries/infundibular necrosis. The current material questions, if the aetiology of infundibular necrosis primarily is due to the effect of bacteria and acids resulting in formation of the eroded cavity. It seems that lack of cementum production in the infundibulum plays a more important role in younger horses. This theory could explain why so many of the relatively young horses, 4 - 6 year of age, suffer from infectious apical periodontitis around the roots of 108/208 and 109/209. The persistent open channel from the occlusal surface and long central channel reaches almost to the apex area (Figure 1-2-3). If bacteria and acids caused these channels, then the channels would not have been as narrow and long but rather forming a wider defect of the infundibular cavity [15]. This theory is supported also by the fact that no histological signs of caries decay was found (i.e. no plaque covered and plaque undermined cement in the infundibulae) with formation of slab-like lesions and cementum disintegration [5,17]. The bacteria and forage is observed inside the normal structured cementum as seen in figure 9 in this study. The bacteria and acid theory is more reasonable when broader cavities in the occlusal surface are formed (Figure 4), and it could result in severe destruction and/or fracture of the cheek teeth. The extended destruction of the cementum and enamel is often seen in relation to peripheral dental caries in older horses and is also seen in the mandibular cheek teeth [12,18,19].

Treatment of infundibulum from the occlusion surface with composite as suggested [1] could be right. Though technically difficult, it may save the tooth and prevent further development of decay. This study confirms what has been described in cases of upper cheek teeth caries [9]. The findings are also similar to previous studies that found the infundibulum often air filled on CT [17].

The hypocementosis can be visualized on radiographs if a contrast study is performed on the emptied infundibular channels

or better in CT or MRI scanning (Figure 9), as also described by other authors [13,17,18,20]. Similar descriptions as in this study in normal teeth and in cases with infundibular infections have been reported [9] too.

The theory about hypocementosis resulting in apical infection has also been mentioned before [18,20], and a case in a 5-year-old horse with straw filled infundibulum have been reported [19].

Conclusion

This study shows that the development of the infundibulum in filling out the cavity with cement in the maxillary cheek teeth is not always completed at the time of eruption and often partly filled with forage debris and air. Therefore, the study supports the possibility that lack of sufficient cementum production before eruption can be the predisposing factor for developing infundibular necrosis.

Acid producing microorganisms and/or sugar containing food may destroy the cement and enamel in the infundibulum, resulting in infection and/or necrosis in the infundibulum and alveolar bone as a sequelae to the hypocementosis in equine developing cheek teeth.

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