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Original Research Paper

PULPITIS AND PULP NECROSIS AS A SEQUEL TO PERIODONTAL DISEASE IN DOGS

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Summary: There is general agreement that pulpal disease can initiate and/or perpetuate periodontal disease; the opposite theory is controversial. Several investigators suggest that pulpitis could be a sequel to periodontal disease. If accessory canals are the main entrance for bacteria and their metabolites to the dental pulp, it is hypothesized that periodontal disease associated pulpitis is less likely to occur in dogs compared to humans, because the typical canine root canal anatomy has very few lateral ramifications away from the delta at the root apex.

The histopathology of dog teeth extracted because of moderate to advanced periodontal disease was studied to determine the presence and range of pulpitis or necrosis. A total number of 22 affected teeth were examined and changes were compared with 5 control teeth obtained from dogs with no clinically detected periodontal disease. There was obvious pulpitis in 27.3 % of periodontally affected teeth with mild inflammation in additional 18.2 %. Pulp necrosis was observed in 40.9 % of cases. Chronic apical periodontitis was confirmed in 44.4 % of teeth with pulp necrosis.

The finding of obvious chronic and acute pulpitis in a significant proportion of cases, despite the low number of lateral canals, was unexpected. Further study is required to determine pathogenesis of pulpitis. The possibility of pulpitis or pulp necrosis in periodontaly involved teeth should be considered when planning periodontal treatment.

Keywords: dentistry – veterinary; periodontal diseases; dental pulp diseases – pathology; dogs

Introduction

Periodontal disease is the most common chronic disease of dogs (1, 2), which locally affects periodontal tissues and leads to teeth loss (1, 3, 4). Additionally, evidence increases, that periodontal disease can be connected to different systemic conditions (2, 5 - 13).

Whilst there is general agreement that pulpal disease can initiate and/or perpetuate periodontal disease (14 - 18), the opposite theory is controversial (15, 17 - 24). Several investigators (4, 25 - 27) suggest that pulpitis could be a sequel to periodontal disease in humans and monkeys as bacteria and their metabolites may gain entry from an infected periodontal ligament through exposed accessory canals. However, this is less likely to occur in dog teeth as compared to human teeth because the typical canine root canal anatomy has very few lateral ramifications away from the delta at the root apex (15, 28 - 30). Dentinal tubules are also wide enough for bacteria and their products to pass into dental pulp tissue (3, 4, 31, 32), but the cementum and enamel play an important defensive role in such cases provided they remain intact (14, 23, 25, 31). Root planing may damage cementum and enhance exposure of dentinal tubules (15, 23) or damage blood vessels entering the pulp through the lateral canals (18). However, Bergenholtz and Lindhe (25) suggest that root planing has no effect on pathology of dental pulp. Bacteria and their products can invade the dental pulp via the blood stream, if sufficient enters the systemic circulation and evades the lymphoreticular filtering system (4, 33).

The aim of the study was to histologically examine the occurence of pulpitis and pulp

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necrosis in dog teeth extracted because of advanced periodontal disease.

Materials and methods

Population of dogs

Client-owned dogs in good general health except for spontaneous periodontal treatment that were presented to the Clinic for Small Animal Medicine and Surgery, University of Ljubljana, between October 2002 and January 2003 for treatment were included in the study. Dogs had not received any professional dental treatment within the previous three years and had received maximum three dental treatments in their lives.

In addition, control teeth, not affected with clinically detected periodontal disease, were immediately post mortem obtained for comparison from two seven- and eight-years-old cocker spaniels that had been euthanised for clinical reasons unrelated to the oral cavity. No previous periodontal treatments were reported for these two dogs.

Dogs were anaesthesied (premedicated with acepromazine 0.02 mg/kg, methadon 0.2 mg/kg and amoxicillin-clavulanic acid 20 mg/kg; induction of anaesthesia was performed with propofol 3 – 4 mg/kg and maintenance with 1 % to 2.5 % isofluorane) and the oral cavity of each dog was then assesed visually and with a William's periodontal probe and radiographed following the clinic's normal procedure.

Specimens selection

Teeth included in the study did not have any carious lesions or complicated crown fractures.

Furcation exposure in multi rooted teeth was graded on a 0 to 3 scale, depending on the penetration of the periodontal probe: 0 – none furcation involvement, 1 – lateral exposure, 2 – incomplete penetration, 3 – complete penetration.

Tooth mobility was graded on a 0 to 4 scale as sugested by Rateitschak et al. (34): 0 – physiological, 1 – detectably increased, 2 – clearly visible, 3 – severe, 4 – extreme.

Radiographic examination was performed if clinical evaluation was not diagnostic. Teeth with maximal furcation involvement or extremely mobile teeth were not radiographed.

Treatment included routine periodontal therapy (supra- and subgingival scaling followed by polishing and gingival lavage) and extraction of compromised teeth (primarily those with excessive mobility, furcation penetration, loss of one third or more of their periodontal attachment, or deep periodontal pocketing). Extraction was performed using standard extraction technique (sectioning of multirooted teeth using a cutting bur in a high speed dental handpiece with copious water spray, raising mucogingival access flaps and alveolar bone removal, as required to facilitate use of elevation/luxation instruments) (35).

Processing of the specimens

Each sectioned extracted tooth was immediately placed in >15-times its own volume of buffered (pH 7.2) 10 % formalin solution. Where the crowns were intact following extraction a hole was made at the cusp tip, using the high speed handpiece, to assist penetration of formalin into the pulp prior to immersion in fixative.

The extracted teeth were fixed for between one and two weeks during which time they were maintained at room temperature and the formalin changed twice weekly. After fixation the teeth were thoroughly rinsed with water prior to demineralisation in >15-times their volume of 12 % ethylenediamine-tetraacetic acid (EDTA). The teeth were maintained at room temperature with gentle agitation and the EDTA solution was changed every seventh day until two weeks after there was no radiographic indication of remaining mineral content. Following demineralisation the teeth were rinsed three times in distilled water and re-fixed for at least three days in buffered formalin prior to further processing.

The fixed, demineralised teeth were processed and embedded in wax in the mesiodistal or buccolingual plane for histological sectioning at thicknesses of 5 to 10 microns. Sections were adhered on glass slides and dried in an oven at 42 şC for 24 hours before being routinely stained with haematoxylin and eosin and mounted under a cover slip. Mesial and distal roots (palatal root being appropriate just in one case) of multirooted teeth were processed and examined.

Each sample of control and diseased teeth was serially examined by light microscopy (the same examiner for all specimens NA), presence of inflammatory cells characteristic for acute and chronic inflammation being recorded in odontoblast layer, in pulp blood vessels and outside blood vessels. Changes in blood vessels were also recorded and photomicrographs were obtained of representative sections. The pulps were then scored on a 0 to 3 scale (0 – no changes, 1 – mild changes, 2 – moderate changes, 3 – severe changes), which is based on the protocols for histological classification of pulp diseases used by

Czarnecki and Schilder (22). Pulps were then evaluated regarding histological evaluation of the pulp described by Seltzer and Bender (4): intact uninflamed – unaltered cells, minimal quantity of collagen fibres, normal blood vessels, altered uninflamed – no inflammatory cells or some present in blood vessels with no margination, altered odontoblast layer/predentin, abnormal disposition of dentin, altered blood vessels, increased amount of secondary/reparative dentin, increased collagen fibres amount, transitional stage – polymorphonuclear (PMN) cells in blood vessels with/without margination, some plasmacells in pulp tissue (not infiltrates), blood vessels/capillary congestion, inflamed pulp – acute pulpitis – increased numbers of PMN cells in blood vessels with margination, some plasma cells may be in pulp tissue, inflamed pulp – chronic pulpitis – increased numbers of plasma cells in tissue – if infiltrates then *partial chronic pulpitis*/if scattered then *total chronic pulpitis*, total necrosis – no structures distinct in the pulp chamber.

Changes in the pulp related to pulp exposure during or immediately after extraction were assessed but excluded from the scoring.

Classifying specimens

Each tooth was classified according to the extent and nature of its periodontal disease involvement to enable investigation for correlations between periodontal parameters and histological findings.

According to probing depth teeth were classified in four groups as suggested by Rubach and Mitchell (27): A: >0 -2 mm, B: 3 – 5 mm, C: 6 – 8 mm, D: >8 mm.

According to furcation involvement teeth were classified in four groups: 0: none furcation involvement, 1: lateral exposure, 2: incomplete penetration, 3: complete penetration.

According to tooth mobility five groups were formed: 0: physiological mobility, 1: detectably increased mobility, 2: clearly visible mobility, 3: severe mobility, 4: extreme mobility.

Statistical analysis

The results were tested for correlations with periodontal parameters using univariant analysis and test for normality, Fisher's exact tests and Spearman's correlation coefficient using a commercial statistical software package (SAS 8.01, procedures MEANS, FREQ and CORR), values of $P<0.05$ being regarded as significant.

Results

Population of dogs and teeth included in the study

Nine small and medium-sized dogs (5 males and 4 females) aged 5 to 12 years (mean 8.8 years) were finally included in the study. Six dogs had had no previous dental treatment, 3 others had had maximally 2 previous dental treatments in their life, last at least 3 years ago. Statistical correlation was positive between age and number of previous dental treatments.

45 teeth were extracted during the study because of advanced periodontal disease (Figure 1) with left mandibular first molar being overrepresented (11.1 %).

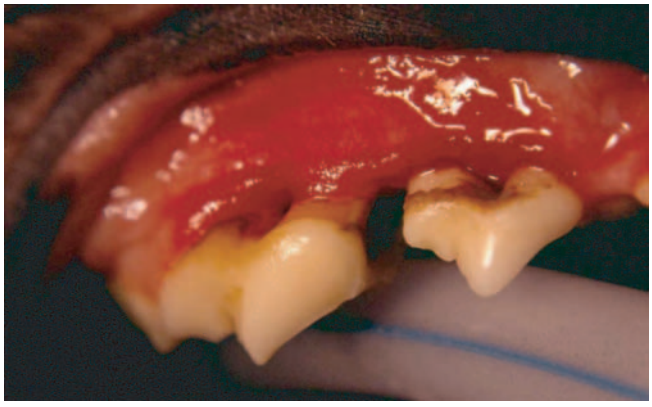

Figure 1: Advanced periodontal disease in a 10-year-old male cocker spaniel. Furcation exposure (F3)

Final histological examination was done in 22 for the evaluation appropriate teeth. 23 teeth were excluded from the study as the pulp tissue was too small or severely damaged during processing, which would make evaluation of the pulp unreliable. Main observations regarding the population of dogs, affected teeth and affected periodontal tissues and histological evaluation of the pulp are presented in Table 1.

Periodontal tissues affection

Only 18.2 % of the teeth had normal probing depth and all of the teeth showed some degree of mobility with 63.7 % showing severe or extreme mobility. The majority of teeth (88.9 %) had furcation exposure (Figure 1).

Table 1: Characteristics of population of dogs, affected teeth, affected periodontal tissues and histological condition of the pulp. Control teeth are not included.

DOG	AGE	PREVIOUS TREATMENTS	TOOTH	PERIODONTAL INVOLVEMENT			HISTOLOGIC EVALUATION OF THE PULP
				PD	M	F	
1	12	2	right mandibular canine	D	4	/	total necrosis
			left mandibular first molar	B	4	3	chronic partial pulpitis
			left maxillary canine	C	3	/	total necrosis
			right mandibular fourth premolar	A	4	2	total necrosis
			right mandibular first molar	B	4	3	chronic total pulpitis
2	10	1	left mandibular first molar	B	3	3	total necrosis
			right maxillary third incisor	B	3	/	altered uninflamed
3	8	0	left mandibular canine tooth	B	3	/	altered uninflamed
			left maxillary fourth premolar	B	3	3	total necrosis
			right mandibular first molar	B	3	3	total necrosis
			left mandibular fourth premolar	A	3	3	acute pulpitis
			left mandibular first molar	B	3	3	acute pulpitis
4	9	0	right maxillary first molar	B	3	3	chronic total pulpitis
5	5	0	right maxillary first molar	D	3	2	total necrosis
6	7	0	right maxillary third premolar	B	2	3	transitional stage
7	8	0	left maxillary second premolar	B	2	3	transitional stage
			right maxillary second premolar	B	2	3	altered uninflamed
8	10	0	left maxillary third premolar	A	1	3	total necrosis
			left mandibular first molar	A	1	3	total necrosis
9	10	2	right maxillary fourth premolar	B	1	3	transitional stage
			right mandibular first molar	B	1	3	transitional stage
			left mandibular first molar	B	1	3	chronic partial pulpitis

Legend: D (probing depth) : A: >0 -2 mm, B: 3 – 5 mm, C: 6 – 8 mm, D: >8 mm,
M (mobility): : physiological mobility, 1: detectably increased mobility, 2: clearly visible mobility, 3: severe mobility, 4: extreme mobility,
F (furcation involvement): 0: none furcation involvement, 1: lateral exposure, 2: incomplete penetration, 3: complete penetration

Histologic evaluation of the pulp

Findings in control teeth:

Figure 2 illustrates what was considered "normal" pulp in aged animals. In all pulps observed blood vessels were mildly (graded 1 to 2) dilated, but capillary congestion was seen only in the crown part of one root; in this case increased number of inflammatory cells were also present in blood vessels (Figure 3). No chronic apical periodontitis was seen in any case.

Findings in periodontally affected teeth:

Intact uninflamed pulp was not observed. Altered uninflamed pulp was diagnosed in 13.6 % of the teeth. Pulpitis was diagnosed in 27.3 % of cases with additional 18.2 % of the teeth showing increased numbers of PMN cells in blood vessels with margination in some parts of the blood vessels, where the pulp was diagnosed as being in transitional stage. Necrosis was found in 40.9 % of cases and in 44.4 % of these cases chronic apical periodontitis was confirmed (only in one case both roots were affected).

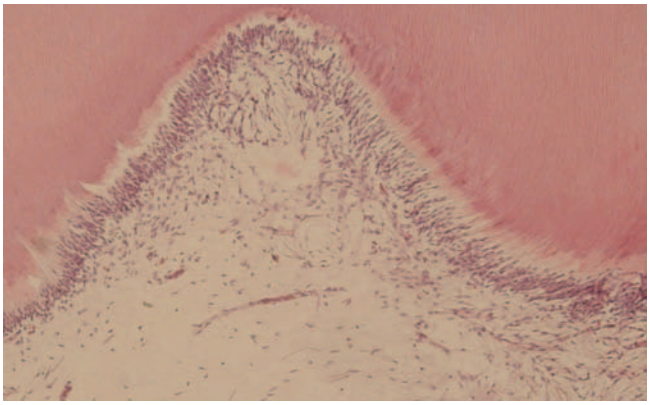

Figure 2: "Normal" pulp tissue of aged dogs. (Light microscope. Mag. 100X, haematoxylin and eosin)

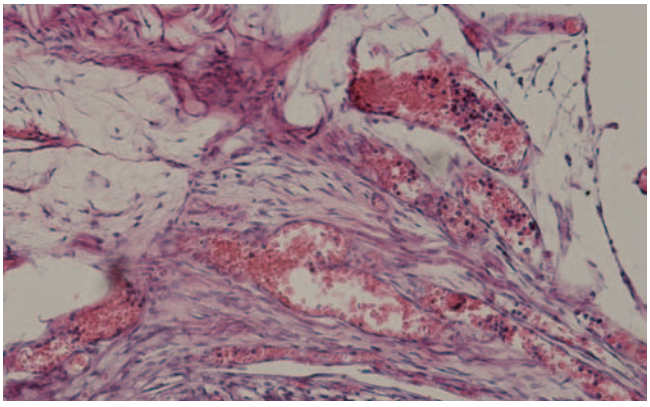

Figure 3: Blood vessels congestion and PMN cells present in the blood vessels in one control tooth. (Light microscope. Mag. 200X, haematoxylin and eosin)

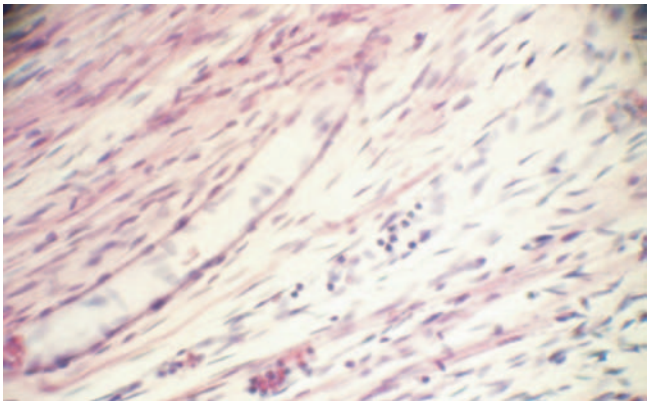

Figure 4: Acute pulpitis. Increased number of PMN cells in blood vessels. PMN cells margination. (Light microscope. Mag. 250X, haematoxylin and eosin)

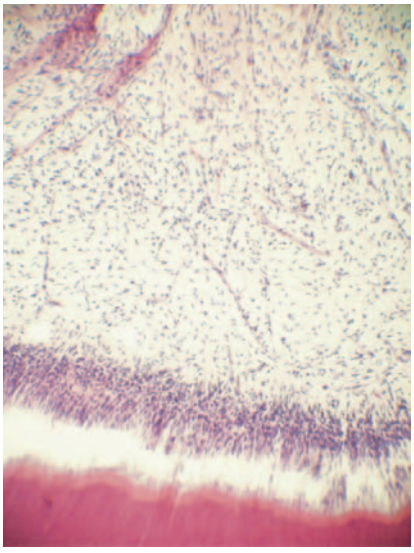

Figure 5: Congested capilaries in crown portion of the pulp in acute pulpitis. (Light microscope. Mag. 100X, haematoxylin and eosin)

Acute pulpitis was confirmed in 9.1 % of the teeth, these teeth having increased numbers of PMN cells in the blood vessels. Margination of PMN cells was observed (Figure 4) and blood vessels were dilated and congested (Figure 5). PMN cells were the main cells involved in acute pulpitis but were not observed outside the blood vessels.

Chronic pulpitis was diagnosed in 18.2 % of cases. In half (9.1 %) of these cases plasma cells, the main cells observed in the cases of chronic pulpitis, were located in the crown portion of the teeth examined. However, some PMN cells were also observed in blood vessels. In these cases pulpitis was determined as chronic partial pulpitis. (Figure 6)

In 9.1 % of cases chronic total pulpitis (Figure 7) was diagnosed as plasma cells were scatered through the pulp and PMN cell number in blood vessels was increased.

The degree of pulpitis varied among the tooth roots in some cases of multirooted teeth.

Some degree of dilatation of blood vessels was observed in all the vital teeth (13 teeth) and in some cases hyalin and/or fibrin was seen in the blood vessels (Figure 8).

There was no statistical correlation found between frequency of previous dental treatments and histologic findings within dental pulp. However, a small number of specimens limited the reliability of statistical tests for correlation between periodontal tissues affection and histologic findings within the dental pulp

Discussion

Inflammation of pulp tissue as well as healing is comparable to that of connective tissue else-

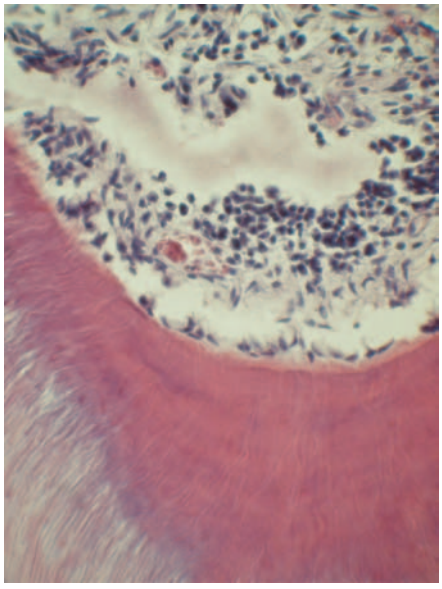

Figure 6: Plasma cells in crown portion of the pulp. Chronic partial (crown) pulpitis. Reparative dentine apposition. (Light microscope. Mag. 250X, haematoxylin and eosin)

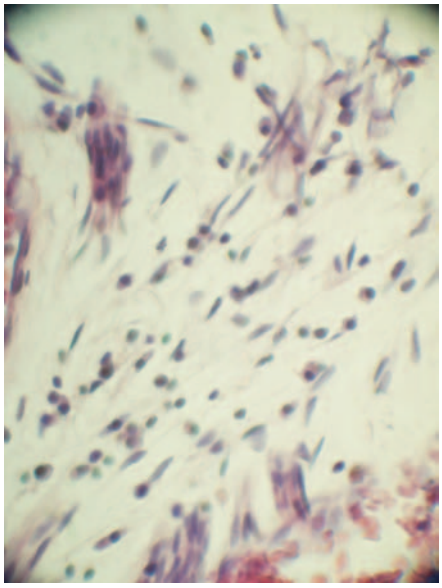

Figure 7: Plasma cells scatered through the pulp in chronic total pulpitis. (Light microscope. Mag. 400X, haematoxylin and eosin)

where (36, 37), despite its location in low-compliance pulp chamber (36, 38). The two key components in pulpal inflammation are microcirculation and sensory nerve activity, which effects the pulpal blood flow (38, 39). Bacteria and their products are the main etiological factors for dental pulp inflammation (37), additionally host

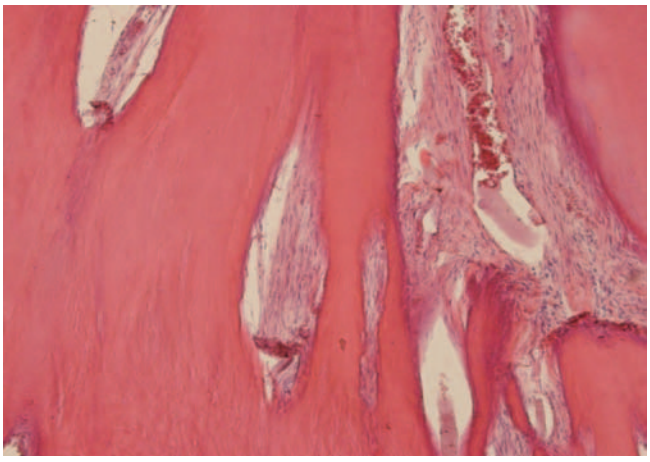

Figure 8: Dilatation and congestion of blood vessels, hyalin accumulation in blood vessels in apical delta. (Light microscope. Mag. 100X, haematoxylin and eosin)

response may contribute to the destruction of pulpal and periapical tissues (4, 40, 41).

The greatest difficulty from a clinical point of view is to assess changes and vitality of the pulp (36, 42). If a cause of pulpitis is quickly removed, the pulp tissue can heal, otherwise the pulpitis becomes irreversible with subsequent necrosis and spreading to periapical tissues (4, 31).

Pulpitis as a consequence of periodontal disease was observed in some studies (4, 25 - 27) in human dentistry. However, pulpitis was not confirmed in periodontally affected teeth of dogs (43) and it is also less likely to occur in dogs compared to humans (15, 28 - 30), if accessory and lateral canals are supposed to be the main entrance for bacteria form infected periodontal ligament to the pulp (4, 17). Despite that, pulpitis was confirmed in our study in 27.3 % of cases.

In acute pulpitis PMN cells were the most prominent cells observed, in severe cases margination – sticking of PMN cells to the endothelial lining, was seen, indicative of chemotactic agents present in the pulp tissue (4, 40, 44). PMN cells play an important role in the defence against bacteria in pulpal tissue (45). They contain antibacterial and enzymatic basic proteins that digest the irritant (4, 40) but these substances and the oxidative burst during PMN cells activity can itself cause greater tissue damage than microorganisms (40). However, acute inflammation occurs soon after the injury but persist for a short period - up to a week, then going into a chronic stage, if it is not resolved, and plasma-cells become responsible for humoral immunity (4).

Partial chronic pulpitis in our study was always present in crown portion of the teeth, which could be due to bacteria invading the pulp through a lat-

eral canal in the furcation area of the tooth. Lateral canals in this area are observed in human teeth but not confirmed in dogs (28). Partial chronic pulpitis in these cases, however, is more likely to be a consequence of greater forces acting on these teeth (4) as in both cases it was mandibular left first molar toot that was affected. The finding could also be due to wearing of the crown enamel exposing dentinal tubules (31) as in one case reparative dentin was seen in pulp chamber. Chronic pulpitis, however, is a consequence of a long-acting but moderate irritation (4).

The degree of pulpitis varied between the roots of some multirooted teeth, as has been described by Langeland et al. for humans (26). This is most likely the result of inflammation extending through the pulp from the primary in one root to the other, there being a reduced stimulus away from the primary site. In the same manner chronic apical periodontitis observed only in one root of multirooted teeth can be explained.

Total necrosis of the pulp could be the result of bacteria and/or their metabolites entering the pulp, as well as of reduced nutrition of the pulp due to blood vessel damage (4, 46). Blood vessels in dogs are very fine as they need to pass through apical delta foramina, most of which are under 100 µm in width (28 - 30), comparing to humans, where the apical foramen is reported to measure between 180 and 290 µm (47). The integrity of these blood vessels is easily damaged and blood supply to the dental pulp reduced. Therefore, the high incidence (40.9 %) of pulp necrosis is likely to be related to the high incidence (63.7 %) of increased tooth mobility in advanced periodontitis in dogs. Additionally, necrosis could also be the result of lasting increases in pulp tissue pressure, which is greater in acute inflammation compared to chronic (4). Necrosis as a consequence of advanced periodontal disease is also reported by other investigators (4, 27).

Chronic apical periodontitis occurs as a consequence of bacterial infection of the dental pulp (31, 48) and was only seen in cases of total pulp necrosis. It is the attempt of the body to limit the spreading of destructive processes from the pulp to periapical tissues (48).

Some degree of dilatation of blood vessels was observed in all pulps of control and periodontally affected teeth. This dilatation could be indicative of hyperaemia (49) or a sign of a tooth being orthodontically stressed (50), but it could also be found in intact pulps or it is just fictive due to greater proportion of collagen fibers (4). Congested blood vessels were more obvious in cases with pulpitis.

As worn teeth were not excluded from the study it could be, that in the case of acute pulpitis in control teeth, dentine tubules recently provided diffusion channels for noxious substances which diffused inward toward the pulp (4, 31). However, no trauma to this tooth was reported in the anamnesis and as other teeth from the same animal showed no similar changes it is also less likely that there was an unevidenced inflammation affecting general health of the animal. Additionaly, we found no reports on inflammation of dental pulp tissue in aged teeth.

Hyalin and/or fibrin was seen in the blood vessels, always in connection to the congestion of blood vessels and we presume that it is a consequence of the congestion. No simmilar changes were observed in control teeth.

Histologic observations of lateral canals are not exact (25), however, pulp tissue in all observed lateral canals in our study was not inflamed even if pulpitis was diagnosed. It could therefore be, that lateral canals are not the most important entrance for bacteria and/or their products to the dental pulp. The idea is also suggested by Bergenholtz and Lindhe (25).

No correlation was detected between periodontal tissue affection and histological evaluation of the pulp as »mild« pulp changes were observed in some cases where the periodontal tissue affection was severe; similar observations are reported by several investigators (4, 22, 25) for human teeth. Bergenholtz and Lindhe (25) suggest that pulp tissue alterations are more likely to occur with the prolonged duration of periodontal disease rather than to the severity of the disease. Verstraete (51) suggests that periodontal disease that progresses to involve the exposure of lateral canals, cement and open dentinal tubules on exposed root surfaces, or the apex can result in endodontic lesion. However, most authors (18, 26, 32, 43) agree that the complete disintegration of the pulp tissue occurs only if the periodontal disease is so severe that apical foramina are exposed to bacteria from periodontal pockets extending to the apex of the tooth. Indeed, pulp necrosis in our study was always found in cases, where probing depth was graded severe (classified as C or D).

Conclusions

The finding of obvious pulpitis in a significant proportion of cases, despite the low number of lateral canals in teeth of dogs comparing to humans, was unexpected. If this pulpitis is related to bacterial contamination from infected periodontal ligament, then lateral canals are not the

most important route af access to dental pulp in dogs affected with periodontal disease. Further study is required to isolate and determine the possibility of pulpitis being the result of aging and attrition of the teeth.

Periodontal disease is known to affect distant tissues, in similar manner it could also affect pulp tissue.

The severity of destruction of periodontal ligament does not seem to be related to pathological alterations within the dental pulp tissue. Therefore, the possibility of pulpitis or pulp necrosis in periodontally involved teeth should be considered when planning periodontal treatment

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References

1. Harvey CE. Periodontal disease in dogs: etiopathogenesis, prevalence, and significance. Vet Clin North Am Small Anim Pract 1998; 28: 1111-28.

2. DeBowes LJ. The effects of dental disease on systemic disease. Vet Clin North Am Small Anim Pract 1998; 28: 1057-62.

3. Kinane DF, Lindhe J. Pathogenesis of periodontitis. In: Lindhe J, Karring T, Lang NP, eds. Clinical periodontology and implant dentistry. 3rd ed. Copenhagen: Munksgaard, 1997: 189-222.

4. Seltzer S, Bender IB. The dental pulp: biologic considerations in dental procedures. 3rd ed. St. Louis: Ishiyaku EuroAmerica, 1990: 360-78.

5. Scannapieco FA. Systemic effects of periodontal diseases. Dent Clin North Am 2005; 49: 533-50.

6. Bretz WA, Weyant RJ, Corby PM, et al. Systemic inflammatory markers, periodontal diseases, and periodontal infections in an elderly population. J Am Geriatr Soc 2005; 53: 1532-7.

7. Felice P, Pelliccioni GA, Checchi L. Periodontal disease as a risk factor in pregnancy. Minerva Stomatol 2005; 54: 255-64.

8. Franek E, Blach A, Witula A, et al. Association between chronic periodontal disease and left ventricular hypertrophy in kidney transplant recipients. Transplantation 2005; 80: 3-5.

9. Pavlica Z, Petelin M, Nemec A, Erzen D, Skaleric U. Measurement of total antioxidant capacity in gingival crevicular fluid and serum in dogs with periodontal disease. Am J Vet Res 2004; 65: 1584-8.

10. Ebersole JL, Cappeli D, Mathys EC, et al. Periodontitis in humans and non-human primates: oral.systemic linkage inducing acute phase proteins. Ann Periodontol 2002; 7: 102-11.

11. Riche EL, Bogges KA, Lieff S, et al. Periodontal disease increases the risk of preterm delivery among preeclamptic women. Ann Periodontol 2002; 7: 95-101.

12. Hajishengallis G, Sharma A, Russel MW, Genco RJ. Interactions of oral pathogens with toll-like receptors: possible role in atherosclerosis. Ann Periodontol 2002; 7: 72-8.

13. DeBowes LJ, Mosier D, Logan E, Harvey CE, Lowry S, Richardson DC. Association of periodontal disease and histologic lesions in multiple organs from 45 dogs. J Vet Dent 1996; 2: 57-60.

14. Peters LB, Wesselink PR, Buijs JF, van Winkelhoff AJ. Viable bacteria in root dentinal tubules of teeth with apical periodontitis. J Endod 2001; 27: 76-81.

15. Bergenholtz G, Hasselgren G. Endodontics and periodontics. In: Lindhe J, Karring T, Lang NP, eds. Clinical periodontology and implant dentistry. 3rd ed. Copenhagen: Munksgaard, 1997: 296-323.

16. Janson L, Ehnevid H, Lindskog S, Blomlöf L. Relationship between periapical and periodontal status: a clinical retrospective study. J Clin Periodontol 1993; 20: 117-23.

17. Rossman LE. Realtionship between pulpal and periodontal diseases. In: Genco RJ, Goldman HM, Cohen DW, eds. Contemporary periodontics. St. Louis: Mosby, 1990: 605-18.

18. Whyman RA. Endodontic-periodontic lesions. Part I: Prevalence, aetiology, and diagnosis. N Z Dent J 1988; 84: 74-7.

19. Bergenholtz G. Pathogenic mechanisms in pulpal disease. J Endod 1990; 16: 98-101.

20. Adriaens PA, DeBoever JA, Loesche WJ. Bacterial invasion in root cementum and radicular dentin of periodontally diseased teeth in humans: a reservoir of periodontopathic bacteria. J Periodontol 1988; 59: 222-30.

21. Torabinejad M, Kiger RD. A histologic evaluation of dental pulp tissue of a patient with periodontal disease. Oral Surg Oral Med Oral Pathol 1985; 59: 198-200.

22. Czarnecki RT, Schilder H. A histological evaluation of the human pulp in teeth with varying degrees of periodontal disease. J Endod 1979; 5: 242-53.

23. Hiatt WH. Pulpal periodontal disease. J Periodontol 1977; 48: 598-609.

24. Bergenholtz G, Lindhe J. Effect of soluble plaque factors on inflammatory reactions in the dental pulp. Scand J Dent Res 1975; 83: 153-8.

25. Bergenholtz G, Lindhe J. Effect of experimentally induced marginal periodontitis and periodontal scaling on the dental pulp. J Clin Periodontol 1978; 5: 59-73.

26. Langeland K, Rodrigues H, Dowden W.

Periodontal disease, bacteria, and pulpal histopathology. Oral Surg 1974; 37: 257-70.

27. Rubach WC, Mitchell DF. Periodontal disease, accessory canals and pulp pathosis. J Periodontol 1965; 36: 34-8.

28. Hernandez SZ, Negro VB, Maresca BM. Morphologic features of the root canal system of the maxillary fourth premolar and the mandibular first molar in dogs. J Vet Dent 2001; 18: 9-13.

29. Gioso MA, Knobl T, Venturini MAFA, Correa HL. Non-apical root canal ramifications in the teeth of dogs. J Vet Dent 1997; 14: 89-90.

30. Gamm DJ, Howard PE, Walia H, Nencka DJ. Prevalence and morphologic features of apical deltas in the canine teeth of dogs. J Am Vet Med Assoc 1993; 202: 63-70.

31. Love RM, Jenkinson HF. Invasion of dentinal tubules by oral bacteria. Crit Rev Oral Biol Med 2002; 13: 171-83.

32. Manfra Marretta S, Schloss AJ, Klippert LS. Classification and prognostic factors of endodontic-periodontic lesions in the dog. J Vet Dent 1992; 2: 27-30.

33. Benenati FW, Roane JB, Waldrop TC. The perio-pulpal connection: an analysis of the periodontic-endodontic lesion. Gen Dent 1981; 29: 515-20.

34. Rateitschak KH, Rateitschak EM, Wolf HF, Hassel TM. Periodontology. In: Rateitschak KH, Wolf HF, eds. Color atlas of dental medicine. 2nd ed. Stuttgart: Georg Thieme Verlag, 1989: 89-147.

35. Holmstrom SE, Frost-Fitch P, Eisner ER. Veterinary dental techniques for the small animal practitioner. 3rd ed. Philadelphia: Saunders, Elsevier, 2004: 291-338.

36. Heyeraas KJ, Sveen OB, Mjor IA. Pulp-dentin biology in restorative dentistry. Part 3: Pulpal inflammation and its sequelae. Quintessence Int 2001; 32: 611-25.

37. Warfinge J. Dental pulp inflammation; experimental studies in human and monkey teeth. Swed Dent J Suppl 1986; 39: 1-36.

38. Maltos KLM, Menezes GB, Caliari MV, et al. Vascular and cellular responses to pro-inflammatory stimuli in rat dental pulp. Arch Oral Biol 2004; 49: 443-50.

39. Kim S. Neurovascular interactions in the dental pulp in health and inflammation. J Endod 1990; 16: 48-53.

40. Seely AJE, Pascual JL, Christou NV. Cell membrane expression (connectivity) regulates neutrophil delivery, function and clearance. Crit Care 2003; 7: 291-307.

41. Chu SC, Tsai CH, Yang SF, et al. Induction of vascular endothelial growth factor gene expression by proinflammatory cytokines in human pulp and gingival fibroblasts. J Endod 2004; 30: 704-7.

42. Pierce A. Pulpal injury: pathology, diagnosis and periodontal reactions. Aust Endod J 1998; 24: 60-5.

43. Okuda A, Murakami H, Tsutsui Y, Asari M. Comparative histopathologic findings of class I endo-perio and class II perio-endo lesions in dogs. In: Pavlica, ed. Proceedings of the 11th European Veterinary Dentistry Congress. Granada: European Veterinary Dentistry Society, 2002: 31-2.

44. Sawa Y, Yoshida S, Shibata KI, Suzuki M, Mukaida A. Vascular endothelium of human dental pulp expresses diverse adhesion molecules for leukocyte emigration. Tissue Cell 1998; 30: 281-91.

45. Nakamura K, Yamasaki M, Nishigaki N, et al. Effect of methotrexate-induced neutropenia on pulpal inflammation in rats. J Endod 2002; 28: 287-90.

46. Bergenholtz G, Nyman S. Endodontic complications following periodontal and prosthetic treatment of patients with advanced periodontal disease. J Periodontol 1984; 55: 63-8.

47. Marroquin BB, El-Sayed MA, Willershausen-Zonnchen B. Morphology of the physiological foramen: I. Maxillary and mandibular molars. J Endod 2004; 30: 321-8.

48. Marton IJ, Kiss C. Protective and destructive immune reactions in apical periodontitis. Oral Microbiol Immunol 2000; 15: 139-50.

49. Heyeraas KJ, Kvinnsland I. Tissue pressure and blood flow in pulpal inflammation. Proc Finn Dent Soc 1992; 88(Suppl 1): 393-401.

50. Wong VS, Freer TJ, Joseph BK, Daley TJ. Tooth movement and vascularity of the dental pulp: a pilot study. Aust Orthod J 1999; 15: 246-50.

51. Verstraete FJM. Self-assessment colour review of veterinary dentistry. London: Manson Publishing, 1999. 100-12.

VNETJE IN NEKROZA ZOBNE PULPE KOT POSLEDICA PARODONTALNE BOLEZNI PRI PSIH

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Povzetek: Bolezni pulpe vplivajo na zdravje obzobnih tkiv, mnenja o vplivu parodontalne bolezni na zobno pulpo pa so deljena. Nekaj raziskovalcev meni, da je vnetje zobne pulpe lahko posledica parodontalne bolezni. Če ob tem verjamemo, da so lateralni kanali glavno vstopno mesto za bakterije in/ali njihove presnovke iz obolele pozobnice v zobno pulpo, je pri psih vnetje pulpe zaradi parodontalne bolezni veliko manj verjetno kot pri ljudeh. Zobje pri psu imajo namreč veliko manj stranskih ramifikacij pulpinega kanala, razen tistih v apikalni delti.

Pulpitis in nekrozo zobne pulpe pri psih smo ugotavljali s histopatološkim pregledom pulp zob, izdrtih zaradi napredovale parodontalne bolezni. Skupno je bilo pregledanih 22 zob, izdrtih zaradi parodontalne bolezni, za primerjavo pa smo uporabili 5 klinično zdravih zob, odvzetih pri psih s klinično zdravimi obzobnimi tkivi. Vnetje zobne pulpe smo diagnosticirali pri 27.3 % zaradi parodontalne bolezni prizadetih zob, povečano število vnetnih celic pa smo poleg tega ugotovili še v pulpah 18.2 % zob. Nekroza pulpe je bila potrjena v 40.9 % primerov. Pri 44.4 % zob, kjer je bila potrjena nekroza, smo ugotovili kronični apikalni parodontitis.

V nasprotju s pričakovanji smo ugotovili akutni in kronični vnetni odziv v zobni pulpi zob, izdrtih zaradi napredovale parodontalne bolezni. Potrebne bi bile nadaljnje preiskave, da se utemelji patogeneza vnetja zobne pulpe. Kljub temu pa je pri načrtovanju zdravljenja prizadetih obzobnih tkiv smiselno upoštevati tudi možnost, da se pri zobeh, prizadetih zaradi parodontalne bolezni, lahko pojavljata vnetje in nekroza pulpe.

Ključne besede: zobozdravstvo – veterinarsko; parodontalne bolezni; zobna pulpa, bolezni – patologija; psi