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External Apical Resorption - Radiographic and SEM Analysis on Teeth with Chronic Apical Periodontitis

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Abstract: ***Studies have suggested that the permanent dentition is protected against physiological resorptive processes, but pathological resorption has been found in cases of trauma, orthodontic treatment, expansion of tumor or cystic formations, or has been largely the result of inflammatory processes in the pulp tissue, etc. The process of tooth resorption involves an elaborate interaction among inflammatory cells, resorbing cells and hard tissue structures. Frequently, this pathologic condition is difficult to predict, diagnose and treat.Key stages in the treatment of chronic apical periodontitis (CAP) are the assessment of the status of periapical zone, condition of the tissues, effective decontamination and subsequent sealing of the root canal space.***

Keywords: apical lesions, chronic apical periodontitis, external apical resorption, foraminalresorption, periforaminal resorption

1. Introduction

Typical chronic apical periodontitis is generally accompanied by periapical bone destruction, which is radiographically observed as periapical radiolucency around the apex. Pathological events in apical periodontitis are mainly characterized by bone destruction which is mediated by osteoclasts [7]. The periapical inflammation is briefly mentioned in the dental literature as a possible cause of radicular external resorption. The severity of resorption is proportional to the duration of the periapical inflammation. Histological studies had shown that the external resorption of cement and dentin is due to an activity of the granulation tissue in the area of the chronic inflammation [3,18,21].

External apical root resorption may be evident histologically but not visible radiographically until it is advanced. If working length is determined without considering this resorption, overinstrumentation or overfilling may occur [12].

The process of tooth resorption involves an elaborate interaction among inflammatory cells, resorbing cells, and hard tissue structures. Frequently, this pathologic condition is difficult to predict, diagnose and treat [4].

Studies by Delzangles (1989), in which the apical surfaces of teeth extracted with apical lesions were examined by means of SEM, have demonstrated that teeth bearing granulomas presented centralized, systematic and extensive apical resorption in the central foramen [5]. The resorption usually occurred in the cementum, however when the lysis was the most remarkable, the dentin was also injured. The author reports that the hard dental tissue surrounding the cyst has shown little or no resorption. In the cysts, when the resorption was present, the zones in it were dispersed and resembled regions of various sizes which could beconnected or not. The areas of resorption were not systematically centralized in the foramina, but were extended over the radicular surface. The occasional distribution of thin zones pathologically attacked caused by the cysts was a great contrast compared with the regularity of the due sites of the granulomas [5,21].

The first report of dental tissue resorption, published in 1530, was made by Michael Blum and this is probably the first scientific report on this process, insufficiently studied even to the present [11].

Root resorption is a biological phenomenon, characterized by processes of cement and/or dentine depletion, resulting from the physiological or pathological activity of resorptive cells, called odontoclasts (a subclass of the osteoclasts) [6, 8]. Studies have suggested that the permanent dentition is protected against physiological resorptive processes, but pathological resorption has been found in cases of trauma, orthodontic treatment, expansion of tumor or cystic formations, or has been largely the result of inflammatory processes in the pulp tissue, etc. [10]. In internal root resorption, normal or necrotic pulp tissue, transformed into granulation tissue with giant multinuclear cells resorbing the dentinal wall in the absence of the odontoblast layer and predentine, has been histologically demonstrated [9, 17].

Resorption is defined as a condition associated with either a physiologic or a pathologic process resulting in a loss of dentine, cementum, and/or bone. Andreasen and Andreasen defined the process further as being of three types; surface resorption, that is a self-limiting process, usually involving small areas followed by spontaneous repair from adjacent parts of the periodontal ligament in the form of new cementum; inflammatory resorption, where the initial root resorption has reached the dentinal tubules of an infected necrotic pulp or an infected leukocytic zone; and replacement resorption, where bone replaces the resorbed tooth material that leads to ankylosis [2]. Root resorption subsequent to orthodontic treatment is considered as surface resorption or transient inflammatory resorption, because replacement resorption is rarely seen following tooth movement. Morphologically and radiographically it may

present as a slightly blunted or round apex to a grossly resorbed apex [2]. Regarding root resorption in this context various authors came out with dissimilar views based on their studies as follows: **i.**Endodontically treated teeth undergoes more resorption than vital teeth; **ii.** Endodontically treated teeth undergoes less resorption than vital teeth; **iii.** Endodontically treated teeth and vital teeth undergo similar resorption.

There are biological mediators that are involved in the initiation and progression of the apical resorption in CAP. Matrix metal proteinases (MMPs), which are endogenous Zn-dependent catabolic enzymes, are responsible for the degradation of collagen and proteoglycans. Their influence and importance to the pathogenesis of CAP are thoroughly studied and clearly defined [1]. Furthermore, the concentration of IgG antibodies has been shown to be almost five times higher for the diseases of the periapical zone than in the non-inflamed oral mucosa. Cytokines IL-1α, IL-1β, TNF-α, prostaglandins, mainly PGE2 and PGI2, and endotoxins are key mediators of the inflammatory process, and also enhance the resorptive processes in the radicular hard dental tissues. Neutrophils are the major source of PGE2 and are present in the initial stage of CAP [14].

The aim of this article is to compare and analyze theradiographic image with electron microscopic results for the presence of apical resorptive areas in teeth with chronic apical periodontitis.

2. Method and Material

The apices of 39 teeth (n = 39) were examined. The teeth were radiographed before extraction according to the digital parallelperiapical technique. The patients were informed for this survey and sign the Informed consent. After extraction, teeth were placed in vials containing a 10% formalin solution and identified.The specimens were dehydrated in increasing concentrations of alcohol (70%, 90% and 99%) for 5 hours each. After resection, the apices were fixed to stands for SEM-examination. The prepared samples were vacuum coated with gold dust in an argon medium by using JEOL JFC- 1200 fine coater and examined with a scanning electron microscope (JEOL JSM-5510 SEM) at the corresponding magnifications (x50, x100),operating at 10 kV.

The scanning electron microscopy images were classified into four levels (0, 1, 2 and 3) - Group I-IV,according to their periforaminal external apical resorption (EAR) - area that circumscribes the apical foramen, and foraminal EAR - area that limits the profile or apical foramen perimeter.This classification was made according to modified criteria based on those described by Vier and Figueiredo [22]:

- absence of resorption-*level 0*- Group I;
- resorption affecting up to 1/4 of the examined area-*level 1*- Group II;
- resorption affecting more than 1/4 of the examined area-*level 2*- Group III;
- resorption affecting more than ½ of the examined area-*level 3*- Group IV.

The digital, parallel, periapicalradiographs were analyzed by two observers (A.G.and D.Y.). The evaluation of the status of periapical tissue was made according to periapical index (PAI) of Ørstavik (score I, II, III, IV and V) [15]. The sign of resorption in apical zones of the roots were analyzed according to the criteria for classifying external apical resorption in radiographs previously described by Ferlini Filho used by Vier-Pelisser F. [22]: 0=absent: root without alteration; 1=superficial: one or more cup-like radiolucencies less than 1 mm deep; 2=deep: superficial profile except extending more than 1 mm deep.Kappa's coefficient was also used to analyze the correlation between external apical resorption diagnosed radiographically and by SEM. The correlation was established between the following measurements: radiographic external apical resorption and periforaminal external apical resorption, radiographic external apical resorption and foraminal external apical resorption, and radiographic external apical resorption and external apical resorption, in the final SEM.

3. Results

The results were grouped and present in Tables 1 and 2. The cases with PAI3 were 48,7%, cases with PAI4 were 33,3% and cases with PAI5 were 18%. Periforaminal resorptionwas observedby scanning electron microscopy in 53.8 % of samples and intraforaminal resorption in 76,9% of samples (Fig.1,2). This calculation was made regardless of level of extension of the analysed samples (apices). The absence of apical resorption was observed only in 2.56% of the specimens (only in one sample)(Fig.3).The specimens in which apical resorption affected more than 1/4 of the examined area (Group III) were found in 20.14% of the cases.The specimens in which apical resorption affected more than 1/2 of the examined area (group IV) were found in 53.8% of the cases(Tabl.3). The comparison between periapical index (PAI) and SEM level of resorption was doneaccording to the criteria for classifying external apical resorption in radiographs (Table 4; Fig.4).

Table 1: The assessment of periapical status on digital parallel radiographs by PAI (according to Ørstavik)

Periapical index (PAI)- according to Ørstavik		
1. 3	14. 3	27. 3
2. 4	15. 3	28. 3
3. 3	16. 4	29. 4
4. 4	17. 4	30. 5
5. 4	18. 3	31. 3
6. 3	19. 4	32. 4
7. 3	20. 5	33. 3
8. 3	21. 3	34. 3
9. 5	22. 4	35. 3
10. 5	23. 4	36. 4
11. 3	24. 3	37. 5
12. 3	25. 4	38. 4
13. 3	26. 5	39. 5

Table 2: Distribution of external apical resorption diagnosed in periapical radiographs according to the three criteria

Criteria	*n**	%
0=Absent	1	2.56
1=Superficially	26	67
2=Severe	12	30.44
Total	39	100

*n = number of samples

Table 3: The distribution of the level of resorption according to modified criteria based on those described by Vier & Figueiredo

Group/Level	*n**	*%*
Group I-0	1	2.56
Group II-1	8	20.5
Group III-2	9	20.14
Group IV-3	21	53.8
Total	39	100

*n = number of samples

Table 4: The comparison between periapical index (PAI) and SEM level of resorption

Comparison between periapical index (PAI) and SEM level of resorption		
1. 3/0	**14.** 3/2	**27.** 3/3
2. 4/1	**15.** 3/3	**28.** 3/1
3. 3/0	**16.** 4/2	**29.** 4/3
4. 4/2	**17.** 4/3	**30.** 5/3
5. 4/3	**18.** 3/1	**31.** 3/3
6. 3/2	**19.** 4/1	**32.** 4/3
7. 3/2	**20.** 5/2	**33.** 3/2
8. 3/3	**21.** 3/0	**34.** 3/0
9. 5/3	**22.** 4/2	**35.** 3/2
10. 5/3	**23.** 4/2	**36.** 4/1
11. 3/1	**24.** 3/0	**37.** 5/3
12. 3/0	**25.** 4/3	**38.** 4/3
13. 3/2	**26.** 5/3	**39.** 5/2

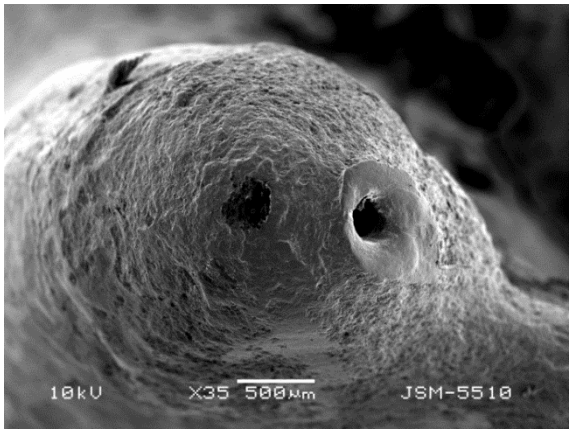

Figure 1: Periforaminal resorption

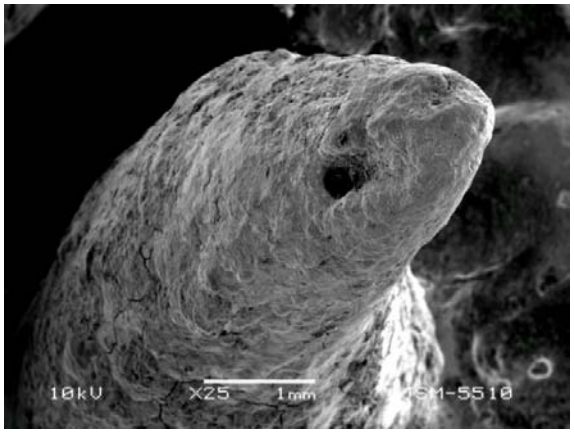

Figure 2: Intraforaminal resorption

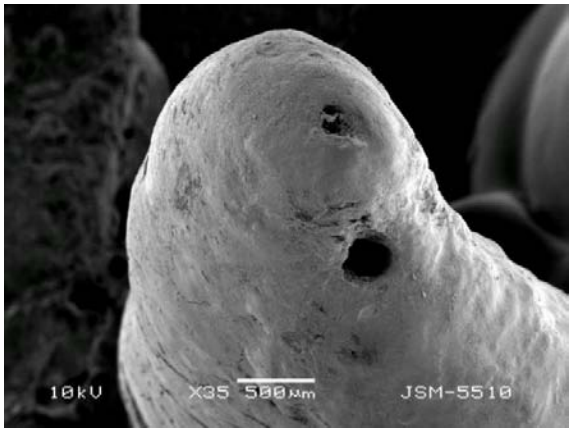

Figure 3: The sample without resorption

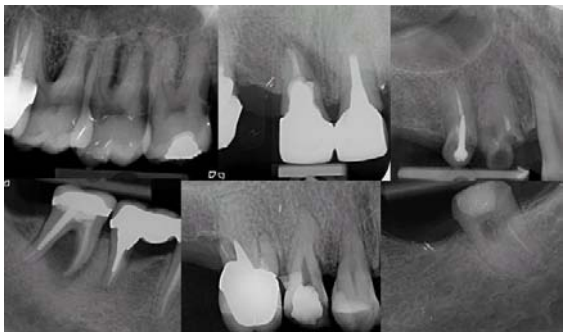

Figure 4: Some of the digital parallel periapical radiographs

In all comparisons of the variances with Fishers F-test, the value of the F ratio fell within the critical value at the 1% level of significance ($p < 0.01$). Student's t-test was used to determine whether there were significant differences between the means of the five four groups at the level of significance ($p < 0.05$).

4. Discussion

One important issue that must be considered in the histopathological description of cyst and granuloma is the fact that both constitute a continuous and variable aspect of the same phenomenon – the inflammation. The literature have illustrated, that in cases of periapical lesions resulting from a pulpal infection and consequent necrosis, inflammatory tissue around the apex may cause resorption of dentin and apical cementum [5, 19].

The fact that the apical resorption or the formation of resorption gaps would constitute in niches which would facilitate the deposing and accumulation of bacteria thus resulting in the formation of periapical bacterial biofilm. On the other hand, it might happen that, in the first place, the physical presence of the bacteria on the apical surface would occur. This situation, however, could culminate in apical resorption. The presence of activating factors for bone resorption in human peri-radicular lesions, such as components C3 of the complement, such as interleucina-1, and factor of tumor necrosis, once presenting the capacity of reabsorbing the bone, could also injure the dental surface [21, 23].

Areas of apical root resorption favor the extraradicular biofilm formation and contributefor the perpetuation of the periapical pathology [16].

The SEM morphological analysis evidenced anextensive area of dentin-cementumresorption, whichpossibly contained microorganisms unachievablefor the endodontic procedures of disinfection (Fig.5).

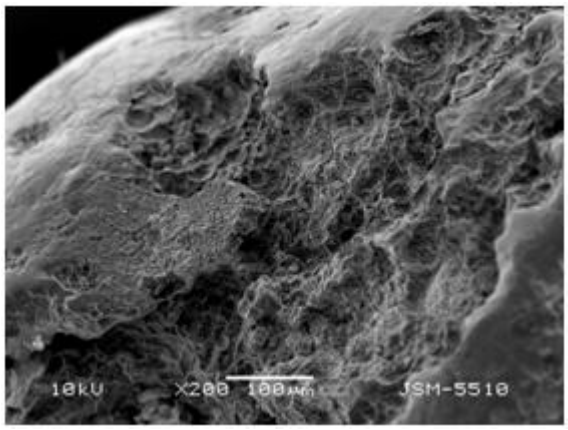

Figure 5: The apical resorption area (magnification x200).

According to Tronstadet al. [20], endodontictreatment failure may be attributed to the presenceof extraradicular infection and microorganisms,which are surrounded by an extracellular matrixand protected from the defense system of the hostand systemic antimicrobial therapies.

External resorption can be classified into 4 categories by the clinical and histologicmanifestations: external surface resorption, external inflammatory root resorption, ankylosis and replacement resorption. In this in vitro study we could not find correlation between radiographic images and SEM-sample.

5. Conclusions

When teeth with periapical lesions were examined for external apical resorption with both radiography and by SEM, in the limitation of this study, we can make the following conclusion: there was no correlation between the findings.

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