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Original Article *C.E. Article # 13-393 (0.5 credit)*

Natural and induced radiation caries: A SEM study

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Abstract: Radiation caries is a well-known indirect side effect of head and neck radiotherapy. The initiation and progression of radiation caries were studied using an *in situ* model. The morphology of induced carious lesions was compared with that of natural radiation caries. Both natural and induced radiation caries showed the same patterns of decay; widespread areas with porosity of enamel, crater formation with exposure of subsurface enamel, preferential dissolution of prisms with hollowing out of prism cores, loss of large parts of surface enamel and loss of full enamel coverage exposing the underlying dentin. In irradiated patients most enamel slabs were severely demineralized within 6 weeks whereas in control subject the slabs showed no significant demineralization after 12 weeks. The model may offer an excellent opportunity to study preventive fluoride regimens because of the rapid induction of radiation caries and its similarity with natural lesions. (*Am J Dent* 1993; 6: 130-136).

Clinical significance: General practitioners should realize that radiation caries is a process with a rapid onset and extremely rapid progression that needs treatment as soon as it is diagnosed. The development in time and not so much its morphology make the radiation caries process unique.

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Introduction

Radiation caries, a highly destructive form of dental caries, is a well-known indirect side effect of radiation treatment for malignant tumors in the head and neck region.[1-3] Irradiation-induced hyposalivation is considered to be the most important etiological factor. Reduced salivary flow and changed salivary composition result in a loss of the protective properties of saliva, a decrease of the salivary pH and buffering capacity, changed nonimmune and immune antibacterial systems, and a shift in oral flora towards cariogenic microorganisms. Hyposalivation is accompanied by a change in the pattern of food consumption to frequent, nondetergent, high-carbohydrate meals.[2,4-11] These factors contribute to an enormous increase in the caries challenge in irradiated patients. In recent studies it was shown that X-irradiation decreased the enamel acid solubility.[12-14] This implies that irradiated enamel is not more susceptible to demineralization than non-irradiated dental enamel, and that irradiation-induced hyposalivation is the main causative factor of radiation caries.

As soon as 3 months after radiotherapy, radiation caries may become clinically evident, whether or not the teeth were included in the field of irradiation.[15-16] Radiation caries progresses so rapidly that a healthy dentition can be totally destroyed within 1 year.[16] Generalized superficial defects that initially affect the smooth surfaces of the teeth are frequently observed. These lesions may result in complete destruction of the coronal enamel and dentin. Decay localized at the incisal or occlusal edges of the teeth is often noticed with these lesions. A second common type of lesion is localized at the cervical regions of the teeth. Progression of these lesions may lead to amputation of the crowns of the teeth, particularly the incisors. Occasionally, a brown-black discoloration of the entire tooth crown is observed with abrasion of the incisal and occlusal edges.[2,3]

The morphological characteristics of radiation caries have been described.[2,3,17,18] No details of the morphologic development of radiation caries as a function of time, however, were given in these studies. In this paper the morphological features of natural and induced radiation caries are described. The initiation and progression of this type of caries were studied using an *in situ* model for the induction of xerostomia-related dental caries.

Materials and Methods

NATURAL RADIATION CARIES

Patients - Eleven permanent molars and incisors, extracted from six patients who had undergone head and neck irradiation (50-70 Gy, 2 Gy/day, 5 days/week), were used. The mean age of the subjects was 63.4 years (range 40-70 years) and all the salivary glands had been included in the treatment portals. Because of the relatively low salivary flow rate, the degree of hyposalivation could be estimated only as the amount of oral fluid present in the oral cavity. This was measured by wiping the oral cavity with a water-adsorbant gauze, which was weighed before and after saliva collection. The test was performed on three different days at approximately the same time of day, and the subjects were not allowed to take food or beverages for 2 hours before the test.[19] The teeth that were extracted were all caries free pre-irradiation and had developed radiation caries during a period of non-compliance with preventive measures. They were divided into two categories based on the extent of decay of the crowns: (1) Crowns with superficial smooth surface caries and/or slight decay at the cervical regions (n= 4); (2) Severely decayed crowns with exposure of dentin (n= 7).

Scanning electron microscopy - The teeth were washed in running tap water to remove surface debris. Thereafter the teeth were fixed in a 2% (w/v) buffered (0.1 M sodium cacodylate, pH 7.4) glutaraldehyde solution at 20°C for 16

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hours, washed with 0.1 M sodium cacodylate buffer solution (pH 7.4) for 10 minutes to remove excess glutaraldehyde and post-fixed in a 1% (w/v) OsO_4 in cacodylate buffer solution for 8 hours at 4°C. After fixation, the teeth were washed in the same buffer solution to remove the non-bound OsO_4 and then in distilled water to remove the buffer. Subsequently, the teeth were dehydrated in a graded series of ethanol up to 100% ethanol. The ethanol was exchanged for iso-amyl acetate in a critical point apparatus and dried with liquid CO_2 (C_T 33°C, C_P 72 atm). The prepared teeth were glued on aluminum stubs with fast curing epoxy resin, coated with gold (approximately 15 nm) in a sputter coater[a] and examined in a JEOL 35C SEM[b] operated at 15 or 25 kV.

INDUCED RADIATION CARIES

Patients and control subjects - Seven irradiated edentulous patients, four men and three women (mean age 67.3 years, range 55-73 years) and six non-irradiated edentulous subjects, two men and four women (mean age 56.2 years, range 43-67 years), participated in this part of the study. The irradiated patients all suffered from xerostomia. They had received an average radiation dose of 55 Gy (range 50-66 Gy) at a level of 2 Gy/day, 5 days/week to the head and neck area. All salivary glands were included in the treatment portals and irradiation treatment was completed at least 1 year before starting the experiment. All patients and control subjects wore full dentures. The salivary secretion was estimated with the wiping method.

Modification of dentures - Both the left and right molars of the lower denture of a patient or control subject were replaced by a metal sample holder.[20] Each holder contained six enamel slabs (three slabs buccally and three lingually), which could be removed and replaced by unscrewing the occlusal part of the holder. About 9 mm^2 of each slab was exposed to the oral environment and the surface of each slab was about 0.5 mm below the outer surface of the sample holder.

Enamel slab preparation - The labial surfaces of caries-free human mandibular permanent incisors and canines were partially ground flat on 1,200-grit silicon carbide paper on a polishing machine,[c] polished on a Kent Mark II polisher[d] using Hypress diamond compounds (1 μm; Engis) and cut in rectangular blocks (3 x 4 x 1.5 mm) by means of a water-cooled diamond saw.[e] All enamel slabs were embedded in a cold-curing polymethyl methacrylate (Rapid Repair[f]). Care was taken to keep the polished enamel surfaces free from acrylic resin. Finally, the slabs were ultrasonically cleaned in tap water for 10 minutes.

Hardness measurements. Microhardness measurements perpendicular to the enamel surface were performed with a Leitz Durimet miniload hardness tester fitted with a Knoop diamond.[g] A load of 100 g was applied for 20 seconds. Five indentations were made in a definite pattern in the central area of each enamel slab.

Scanning electron microscopy - The enamel slabs were washed under running tap water and wiped with tissue to remove surface debris, dried in air, and subsequently glued on aluminum stubs with fast-curing epoxy resin. Specimens were not fixed with glutaraldehyde and $Os0_4$ and critical point dried, as was performed with the the natural radiation caries samples, because the presence of the bacteria was not an important part of the study of induced radiation caries. When transverse examinations were required, the slabs were also fractured. A thin Au layer (approximately 15 nm) was sputtered on the slabs. Scanning electron micrographs were taken with the SEM.

Experimental set-up - In irradiated patients the experiments extended over a 6-week period; beyond this period most enamel slabs were so severely affected that the enamel was lost. At weekly intervals an enamel slab was removed randomly from the dentures of each patient for SEM evaluation. The control experiments extended over a 12-week period. An enamel slab was removed for SEM 3, 6, and 12 weeks after the start of the experiment. In both patients and control subjects, hardness measurements were performed on enamel slabs at the times mentioned above.

All subjects were not allowed to clean the enamel slabs other than under running tap water. The remaining parts of their dentures were brushed with toothpaste containing no fluoride (Prodent Non-Fluoride[h]). The participants were instructed to keep their dentures in tap water (0.1 mg/L F^-) during the night.

Results

All irradiated patients suffered from moderate to severe xerostomia with the amount of saliva in the oral cavities being less than 450 mg (Table 1). The control subjects had amounts of saliva greater than 1.5 g in their oral cavity, which is considered normal.[19]

NATURAL RADIATION CARIES

Characteristic morphological features of natural radiation caries are depicted in SEM micrographs from a representative maxillary molar (Fig. 1), incisor (Fig. 2), and cuspid (Fig. 3). At most carious sites, the teeth were covered with a dense plaque accumulation, consisting of spheroidal, spherical, rod-shaped, and intertwining filamentous bacteria.

The distal surface of a maxillary molar is shown in Figs. 1A, 1B. The neighboring tooth was removed before irradiation. The crown of the molar is relatively intact, but signs of superficial smooth surface decay can be seen on almost the entire distal surface, in particular at the areas A and B. Large portions of the enamel are lost at the cervical area of the crown (Fig. 1A, area D.)

Fig. 1B details areas A and B of Fig. 1A showing a step-like pattern (a) partly covered by patches of plaque (b); (c) indicates a shallow, nearly rectangular area, with arrows showing a larger accumulation of plaque. The step-like pattern represents either perikymata at the enamel surface or slightly worn-off enamel surface following prism planes

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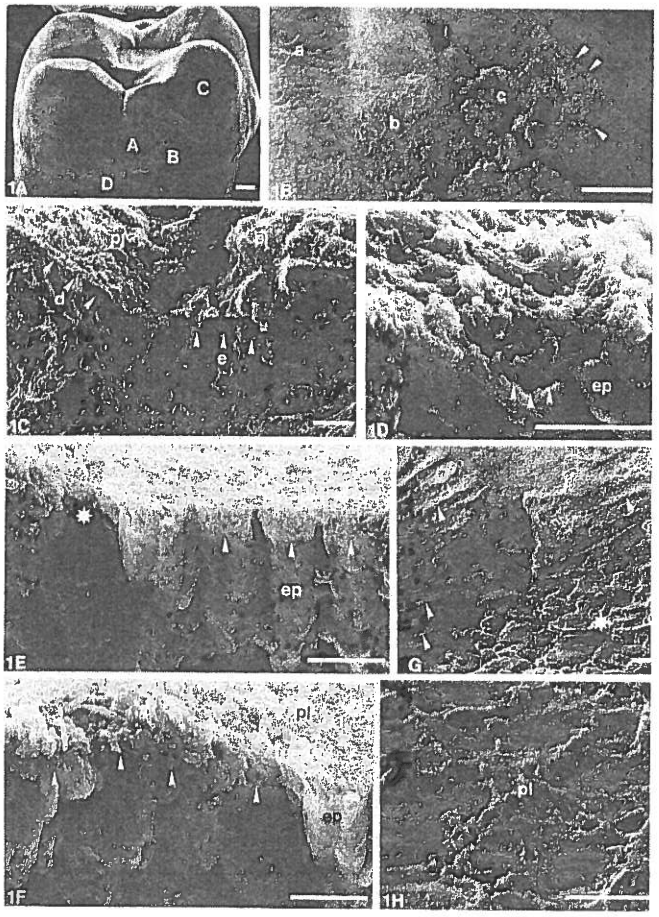

Fig. 1. Maxillary molar. A. Distal surface of a maxillary molar with severe enamel demineralization at (B) and (D) (Bar=1 mm). B. Detail of areas (A,B) of Fig. 1A. Areas (a,b) are covered with patches of plaque and show slight demineralization. Larger accumulation of plaque and more severe surface demineralization is observed at (c) (Bar=1 mm). C. Cross-fracture through area (c) of Fig. 2B showing locations with minor (d) and more pronounced demineralization (e). Note the heavy accumulation of plaque (pl) (Bar 100 μm). D. Detail of Fig. 1C (e) showing pronounced demineralization of the enamel (arrows) and plaque deposition (pl) (Bar=10 μm). E. Detail of Fig. 1C (d) with enamel prisms (ep) covered with plaque (pl). The border between the layer of plaque and prisms is indicated by a change in contrast (arrows). At (*) the prism-pattern has been disturbed due to demineralization. (Bar=10 μm). F. Detail of area (*) of Fig. 1E (Bar=10 μm). G. Low magnification of Fig. 1B (a) showing surface softening as a step-like pattern (arrows), partially covered (*) with patches of plaque (Bar=100 μm). H. Detail of plaque patches (pl) of Fig. 1G. (Bar=100 μm).

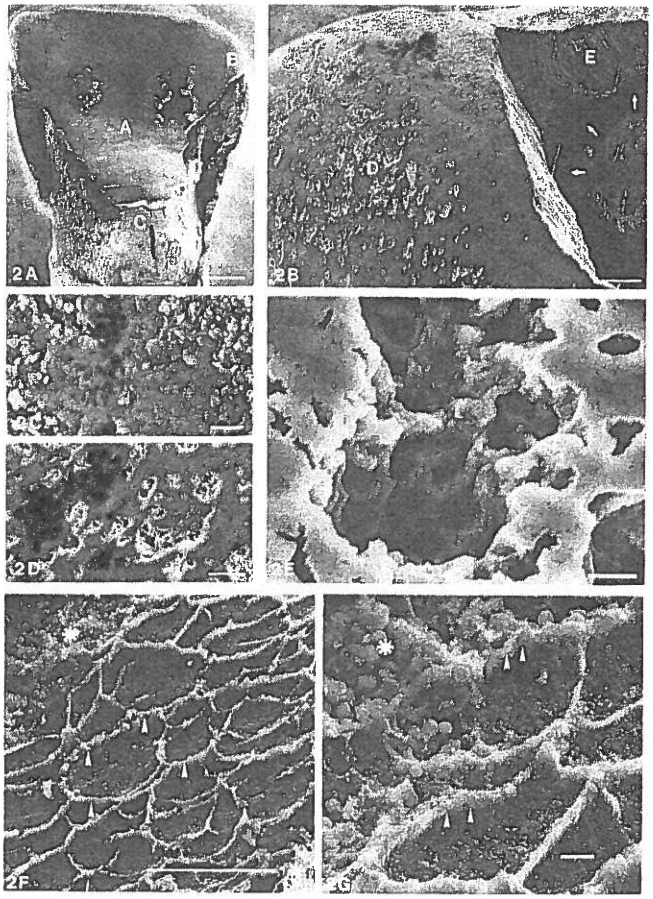

Fig. 2. Maxillary incisor. A. Palatal surface of a severely decayed maxillary incisor with extensive cervical caries, loss of enamel from cervical and proximal areas, enamel fractures (B), and exposure of dentin. (Bar=1 mm). B. Detail of the incisal edge showing demineralization (D) and fractured enamel (E); gap (arrows) due to shrinkage (Bar= 100 μm.) C. Detail of demineralization at (A), (Bar=10 μm). D. Higher magnification of (D) with an irregular pattern of porosity and crater formation (Bar=30 μm). E. Detail of (A) with crater formation and exposure of subsurface enamel (Bar=5 μm). F. Exposed dentin at (C) with characteristic precipitation of calcium phosphate salts (arrows) and bacteria (*) at border of dentin and lost enamel (Bar=100 μm). G. Detail of Fig. 2F with bacterial growth (*) and deposition of calcium phosphate at the dentin surface (Bar=10 μm).

inwards. This pattern is shown at a higher magnification in Fig. 1G (arrows). The patches of plaque (*) are shown in more detail in Fig. 1H.

Fig. 1C shows a cross-section through the area (c) of Fig. 1B. A part of the covering plaque layer has been peeled off. The enamel seems reasonably intact at area (d), while in the middle part (area e), with a large accumulation of plaque, enamel demineralization has occurred. This is shown in more detail in Fig. 1D. Details of the fracture sites (d) and (e) are shown in Figs. 1E and 1F. Obviously demineralization has occurred at the left side (*), where the regular prism pattern is disturbed. A higher magnification of this area is shown in Fig. 1F indicating surface softening/demineralization. At the right site, a regular prism-pattern can be observed (Fig. 1E: ep). The plaque layer (pl) above the partly softened enamel prisms is clearly visible by the change in contrast (Figs.1E, 1F).

Fig. 2A shows a severely decayed maxillary incisor with extensive cervical caries. Most enamel is lost from the cervical and proximal areas. It is a characteristic finding that in the more advanced cases of radiation decay, fractures occur within the enamel and large parts of the tooth crowns become denuded of enamel resulting in exposure of the underlying dentin. The gap between enamel and dentin shown in the SEM micrographs (Fig. 2B, arrows) is an artifact caused by differences in shrinkage of enamel and dentin during preparation of the specimens for SEM. The palatal

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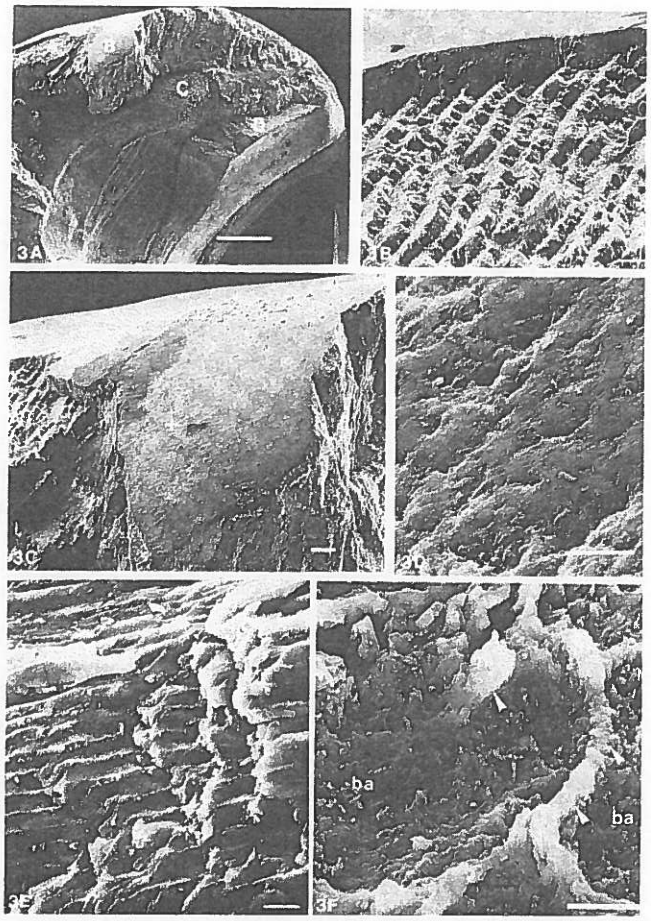

Fig. 3. Maxillary cuspid. A. Severely decayed maxillary cuspid with major parts of labial, incisal, palatal, and mesial surfaces denuded of enamel (Bar=1 mm). B. Higher magnification of Fig. 3A (A), showing a sound subsurface layer after fracturing. The prisms are oriented in planes reaching the very surface of striae of Retzius (Bar=10 μm). C. Higher magnification of labial smooth surface at (B) showing demineralization (Bar=100 μm). D. Higher magnification of (B) with porosity of enamel and crater formation (Bar=10 μm). E. Detail of (A) showing characteristic hollowing out of prism cores with remnants of interprismatic substance (Bar=10 μm). F. Higher magnification of fractured enamel at (D) showing a longitudinal view with evidence of preferential prism dissolution (Bar=10 μm). G. Higher magnification of exposed dentin at (C) with characteristic precipitation of calcium phosphate salts (arrows) (Bar=10 μm).

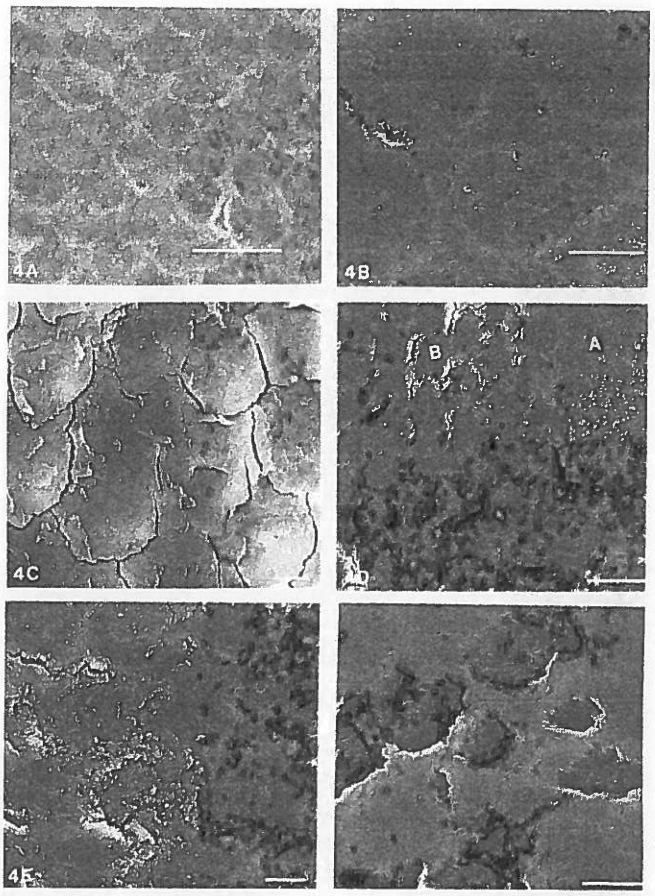

Fig. 4. Early stages of induced radiation caries. A. Accentuation of arcade-formed prismatic structure (Bar=10 μm). B. Detail of accentuated arcade-formed prismatic structure (Bar=3 μm). C. Development of splits and microfractures along prismatic arcades (Bar=3 μm). D. Starting crater formation from third week onward (Bar=10 μm). E. Higher magnification of crater formation at (B) of Fig. 4D (Bar=3 μm). F. Higher magnification of craters from another slab showing exposure of deeper enamel layers (Bar=10 μm).

Table 1. Amount of oral fluid (mg) present in the oral cavity of irradiated and control subjects.

Experiment	Mean	Range	N
Natural radiation caries	320	30- 400	6
Induced radiation caries	410	50- 450	7
Controls	2000	1800-3000	6

(area A on Fig. 2A, Fig. 2C) and incisal (area D on Fig. 2B, Fig. 2D) surfaces show evidence of demineralization. The enamel in these regions shows large areas with irregular patterns of surface destruction with crater formation. Exposure of subsurface enamel in such craters with evidence of hollowing out of prism cores is depicted at a higher magnification in Fig. 2E.

Figs. 2F and 2G represent an area of exposed dentin at the cervical region (area C on Fig. 2A). In most of the cases it looked as if the enamel mineral had dissolved from the same areas and had reprecipitated in adjacent areas (arrows). Bacteria are found at the border with the fractured enamel and at ridges of precipitated calcium phosphate.

Fig 3A depicts a severely decayed maxillary cuspid with major parts of the labial, incisal, palatal, and mesial surfaces denuded of enamel. Both exposure and decay of dentin can be observed. At location A, a large area of surface enamel has been lost (Fig. 3A). Typical prism-planes meeting the very surface of the enamel at the striae of Retzius can be observed. The pattern itself is not an example of demineralization, but a result of fracturing due to softened surface enamel. A detail of area (B) of Fig. 3A is shown in Fig. 3C. The porous character indicates demineralization/softening of the enamel. A detail of this area is shown in Fig. 3D. Some bacteria are seen and a wide-spread crater formation is clearly visible.

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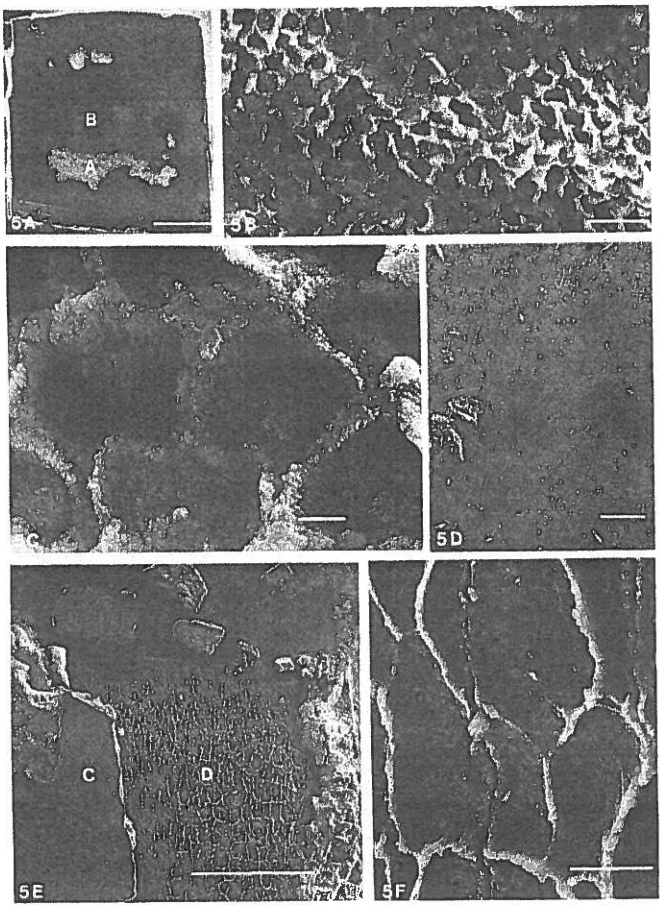

Fig. 5. Advanced stages of induced radiation caries. A. Overview of enamel slab after 3 weeks with loss of large parts of surface enamel (Bar=1 mm). B. Detail of (A), subsurface enamel showing a characteristic prismatic pattern of demineralization with preferential dissolution of prisms and remnants of interprismatic substance (Bar=30 mm). C. Volcano aspect of hollowed out prism cores, detail of Fig. 5B (Bar=3 μm). D. Higher magnification of (B) with generalized porosity and starting crater formation (Bar=3 μm). E. Overview of enamel slab after 6 weeks with evidence of loss of full enamel coverage at (D) and relatively intact surface enamel at (C) (Bar=1 μm). F. Higher magnification of exposed dentin at (B) with characteristic precipitation of calcium phosphate salts (Bar=30 μm).

In Fig. 3E a fracture through the enamel at location (D) of Fig. 3A with longitudinal prisms and evidence of preferential dissolution of prism cores with remnants of interprismatic substance are shown. The typical reprecipitation of mineral material as ridges onto the exposed dentin (comparable with Fig. 2F) and bacteria (ba) are shown in Fig. 3F.

INDUCED RADIATION CARIES

SEM of the enamel slabs from irradiated patients all showed characteristic patterns of demineralization and decay. The SEM micrographs from representative enamel slabs obtained from different patients are depicted in Figs. 4 and 5.

During the first weeks (early stages) most enamel slabs showed an accentuation of an arcade-formed prismatic structure (Figs. 4A, 4B, Fig. 4D area A). In many slabs microfractures developed along these prismatics arcades (Fig. 4C, Fig. 4D area B). From the third week onward most of the samples showed a porous enamel surface and starting crater formation (Figs. 4E, 4F). Although plaque accumulation was greater at the peripheral parts of the slabs, the carious destruction was extensive in both the central and peripheral parts of the slabs.

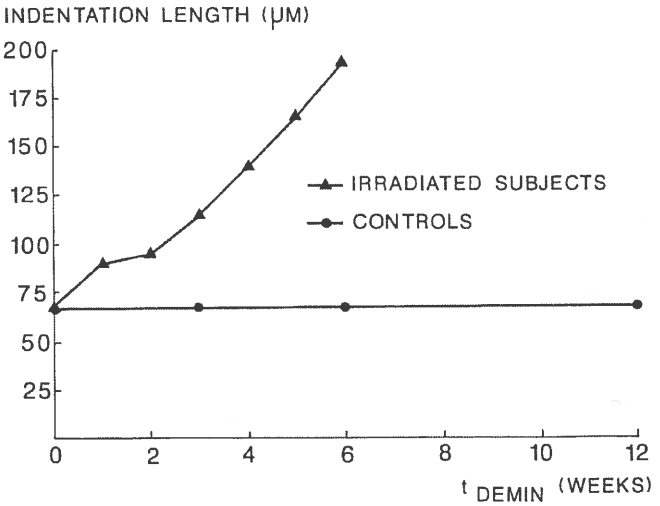

Fig. 6. Indentation length (μm) of the enamel slabs in irradiated patients (n = 7) and control subjects (n = 6) as a function of time of demineralization (weeks).

After 3 to 6 weeks (advanced stages) most enamel slabs were severely damaged and demineralized. In many slabs large parts of the surface enamel were lost exposing the deeper enamel layers (area A on Fig. 5A). At higher magnification a characteristic pattern of demineralization, namely preferential dissolution of the prism cores with remnants of interprismatic substance, was observed. The result is a keyhole like appearance (Fig 5B, 5C). The appearance of this keyhole structure is not specific for radiation caries, because it is observed with natural caries as well. In contrast to natural caries, however, it is induced in a short period of time in these enamel slabs (within 3 weeks after radiation). Moreover, one should have expected an attack at the borders of the enamel slab where accumulation of bacteria takes place. Fig. 5D represents a detail of area B on Fig. 5A, where generalized porosity with starting crater information is depicted. In some cases the enamel has even peeled off exposing the dentin (Fig. 5E). The pictures suggest that the tooth mineral was dissolved from some areas and was reprecipitated in adjacent areas (Figs. 5E, 5F).

Most control enamel slabs showed no demineralization during the 12-week experimental period when viewed in the SEM. At most, local areas with slight porosity of the enamel surface were observed.

Hardness measurements - The changes in indentation length, I (μm), of the enamel slabs placed in the oral cavities of irradiated patients (0-6 weeks) and control subjects (0-12 weeks) are shown in Fig. 6. In irradiated patients,

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about one-third of the enamel slabs was damaged so severely by the progressive caries process after the third week that further measurements were not reliable. For that reason median values are presented instead of mean values, and no standard deviations are given. The hardness measurements indicated a significant demineralization of the samples in irradiated patients within 2 weeks. In control samples no increase in indentation length was observed.

Discussion

Early radiation caries lesions seem to be similar to incipient normal caries lesions in permanent teeth.[2,18,21,22] The prismatic pattern of demineralization and the irregular pattern of destruction have also been observed in normal incipient carious lesions.[21] Striking differences between normal and radiation caries are the rapid onset and progression of radiation caries, its widespread occurrence on enamel surfaces, the loss of large enamel parts, and the fact that radiation caries is most commonly found on tooth surfaces that are relatively immune to dental caries. As mentioned in the introduction, the main etiological factor for radiation caries is irradiation-induced hyposalivation. The quantitative and qualitative changes of whole saliva,[8] and the concomitant shift in the oral flora towards cariogenic microorganisms produce a cariogenic environment that is aggravated by the changed pattern of food consumption. Direct effects of ionizing irradiation on dental enamel do not contribute significantly to the demineralization process.[12-14] In radiation caries the characteristic demineralization of caries-immune, self-cleansing areas, is also related to the altered oral environment produced by irradiation-induced changes in salivary flow, composition and consistency of saliva that gives rise to accumulation of highly acidogenic dental plaque on these surfaces.[3] The aggressiveness of the oral environment was clearly demonstrated in the model by the difference in progression between caries induced in irradiated *versus* non-irradiated subjects.

To induce radiation caries lesions in a relatively standardized and reproducible way, the surface layer of the enamel slabs was removed in the model. Previous studies carried out in our laboratory had shown that the enamel fluoride concentrations in adjacent sites on unground surfaces varied significantly, whereas removal of the surface enamel by grinding resulted in enamel fluoride concentrations in adjacent sites that were not significantly different.[23] The occurrence of dissolution of prism sheaths in some surface areas of extracted teeth instead of the mostly observed interprismatic demineralization, may be related to local differences in the fluoride content of the surface layer,[24] and was probably seen less in the model because of removal of the surface layer during slab preparation. Edentulous subjects were chosen because data obtained in these subjects are not influenced by variables such as oral hygiene or other preventive measures which an irradiated dentulous subject has to apply in order to preserve his dentition (topical fluoride applications). Considering the oral flora, only minor differences in its composition exist between dentulous and denture-wearing edentulous xerostomic patients.[25]

From the rapid, standardized and reproducible induction of radiation caries in the *in situ* model, the sequence of the stages in the development of radiation caries can be followed. The initial step in the demineralization process is the occurrence of porosity on widespread areas of enamel. This is followed by crater formation with exposure of subsurface enamel, preferential dissolution and hollowing out of prism cores, loss of large parts of surface enamel, and loss of full enamel coverage exposing the underlying dentin. The same morphological features were also observed in the extracted teeth with natural radiation caries. In these teeth, however, the more advanced stages predominated.

In a previous investigation[13] comparison of results obtained with scanning longitudinal microradiography (LMR), scanning optical monitoring (OM), and hardness measurements showed a significant increase in indentation length before any significant change in mineral composition could be observed with LMR and OM. SEM observations only showed accentuation of prisms at that point in time. Although hardness measurements are not reliable on severely damaged enamel surfaces, they can be utilized as an indicator of the initial softening of the enamel slabs and were added to the model to characterize the onset of the caries process.

In this study, *in situ* induced radiation caries resembled natural radiation caries. In all patients, the induced radiation caries showed the same typical patterns of decay and the same sequence of events. The *in situ* model may offer an outstanding opportunity to study preventive fluoride regimens in preserving the natural dentition in subjects who have undergone head and neck radiotherapy.

a. Balzers Union, Balzers, Liechtenstein.
b. JEOL, Tokyo, Japan.
c. Buehler Ltd., Lake Bluff, IL, USA.
d. Engis, Maidstone, England.
e. Horico, Berlin, Germany.
f. DeTrey, Weisbaden, Germany.
g. Leitz, Wetzlar, Germany.
h. Kortman Intradal, Veenendaal, The Netherlands.

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CE Questions - Natural and induced radiation caries: A SEM study.

1. In irradiated patients, most enamel samples were severely demineralized within:

A. 1 week
B. 2 weeks
C. 3 weeks
D. 6 weeks
E. 12 weeks

2. The most important etiological factor of radiation caries is:

A. Time
B. Hyposalivation
C. Lack of preventive care
D. None of the above

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