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Histopathology of furcation lesions associated with pulp degeneration in primary molars

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Abstract

The purpose of this report is to describe the histopathology of radiolucent lesions associated with pulp necrosis in primary molars.

Twenty-one carious, previously untreated, primary molars with radiolucent lesions were extracted with forceps under local anesthesia. If a lesion remained attached to the root, the specimen was transferred to 10% neutral buffered formalin. The sockets were gently curretted and the tissue transferred to formalin fixative. Specimens were processed, stained with hematoxylin and eosin and viewed under a light microscope.

Most specimens contained a mixed response including granulomatous inflammation, chronic proliferative inflammation, acute inflammation, and epithelium. Granulomatous inflammation was the predominant response. The cellular population varied as to the relative amounts of lymphocytes, plasma cells, monocytes, macrophages, and polymorphonuclear leukocytes. Odontogenic epithelium was observed in 10 of the 21 specimens.

Radiolucent lesions associated with nonvital primary molars may be classified as furcation granulomas, granulomas with epithelium suggesting potential for cystic transformation or furcation cysts.

A radiolucent lesion in the root furcation is a classic radiographic sign of pulp necrosis in a primary molar (Winter 1962; Moss and Addelston 1965). In contrast, lesions associated with pulp necrosis in a permanent molar usually appear as a periapical radiolucency (Lalonde and Lueke 1968). These lesions may be diagnosed as either a granuloma or a cyst and histological examination is required to establish a final diagnosis.[1] Granulomatous inflammation is a consistent feature of the periapical radiolucent lesions associated with permanent teeth (Weiner et al. 1982) and consists of a classical fasicular or swirling pattern of mononuclear series cells surrounded by lymphocytes and fibroblasts (McKinney 1981). Compared to the extensive information describing periapical lesions in permanent teeth, limited information is available concerning the histopathology of lesions associated with pulp degeneration in primary teeth. Pulp pathology in primary teeth may cause pain and infection and adversely affect the developing successional tooth. Enamel hypoplasia, cessation of root development, positional alterations and arrested tooth development have been reported.[2] Knowledge of the histopathology of these lesions could provide additional insight for improving the treatment rationale. The purpose of this report is to describe the histopathology of radiolucent furcation lesions associated with pulp degeneration in primary molars.

Materials and Method

Specimens were obtained during extraction of 21 primary molars from 17 healthy children, eight females and nine males between four and 12 years of age. The extracted teeth included 10 maxillary first primary molars, four maxillary second primary molars, five mandibular first primary molars, and two mandibular secondary primary molars. All the teeth had carious pulp exposures and displayed a radiolucent lesion in the root furcation characteristic of pulp degeneration (Fig 1, page 280).

In a few instances, the radiolucent lesion appeared to extend beyond the root furcation and encompass a portion of the remaining root structure. None of the teeth had received any previous pulp therapy although several teeth had been previously restored. None were considered suitable candidates for conservative pulp treatment. All the teeth were extracted in the usual manner with elevators and forceps under local anesthesia. When a lesion remained attached to the root structure after extraction, it was detached and transferred to

[1] Block et al. 1976; Lalonde and Lueke 1968; Langeland et al. 1977; Weiner et al. 1982.

[2] Binns and Escobar 1967; Brook and Winter 1975; Messer et al. 1980.

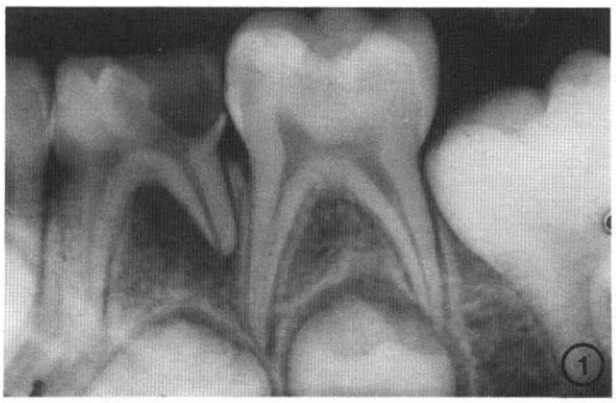

FIG 1. Mandibular first primary molar with radiolucent furcation lesion characteristic of pulp pathology.

10% neutral buffered formalin for fixation. All extraction sockets were gently curretted and the contents transferred to 10% formalin. The tissue specimens were processed for routine paraffin embedding and cut as 5 μm serial sections. The sections were stained with hematoxylin and eosin (H&E) and examined under a light microscope to differentiate cell type and general features of the lesion.

Results

Microscopic evaluation of the furcation lesions revealed a mixed cellular response which included granulomatous inflammation, chronic proliferative inflammation, acute inflammation, and epithelium.

Acute inflammation was identified by the presence of polymorphonuclear leukocytes and chronic proliferative inflammation by the presence of lymphocytes, monocytes, macrophages, and plasma cells (Fig 2).

Granulomatous inflammation was observed in almost all the specimens and is characterized by the presence of mononuclear phagocytic cells, monocytes, and macrophages in an orderly fasicular or circular streaming pattern, often surrounding a central nidus of amorphous eosinophilic material (Fig 3).

These fasciles often were surrounded by outer rimming of lymphocytes and fibroblasts. Foreign body type giant cells were present in some sections (Figs 2, 4).

Variation was observed between sections in the relative amounts of lymphocytes, plasma cells, monocytes, macrophages, and polymorphonuclear leukocytes. Chronic proliferative inflammation was the other predominent inflammatory pattern often seen adjacent to or amid the granulomatous inflammatory component. Fibroblasts were found in all specimens and tended to be localized peripherally to the granulomatous inflammatory fasciles (Fig 3) or scattered among the chronic proliferative reaction (Fig 2). Lesser numbers of plasma cells, lymphocytes, and macrophages were observed scattered throughout the rest of the microscopic field (Fig 2). Acute inflammation, represented by the presence of polymorphonuclear neutrophilic leukocytes, was evident in some specimens (Fig 2). The neutrophils

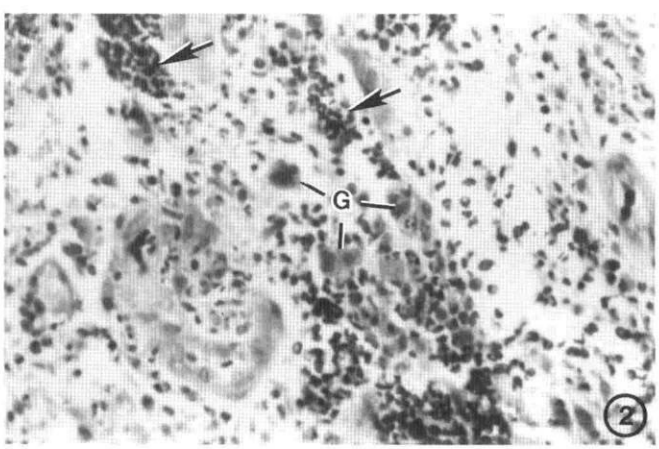

FIG 2. Section displaying mixed response of acute and chronic proliferative inflammation. Arrows indicate polymorphonuclear leukocytes of acute inflammation. Giant phagocytic cells also are present (G) [25 x 1.25 x 3.2 x].

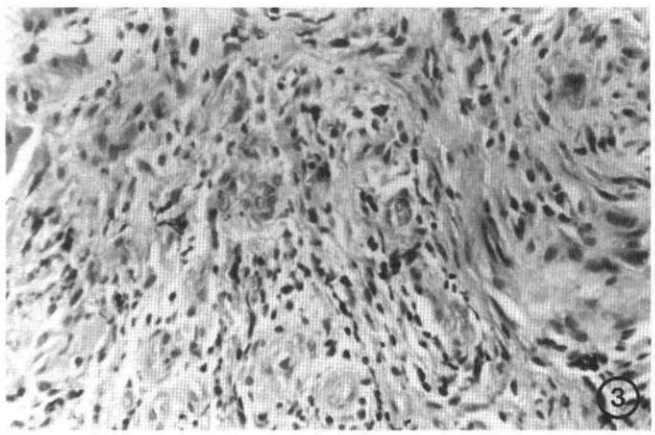

FIG 3. Orderly swirling or fascile pattern of mononuclear phagocytic cells typical of granulomatous inflammation (25 x 1.25 x 3.2 x).

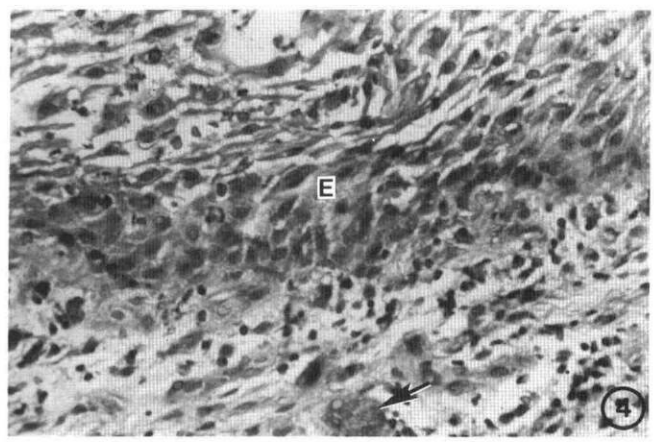

FIG 4. Epithelium (E) with spongiosis and exocytosis. Also note the chronic proliferative inflammatory cells and a giant cell (arrow) [25 x 1.25 x 3.2 x).

were scattered in variable numbers, or found in microfoci, throughout the sections as a component of the chronic proliferative and granulomatous inflammatory tissues. Granulation tissue was not observed.

Epithelium was detected in 10 of the 21 specimens. Epithelium was observed in both granulomatous and chronic proliferative inflammatory areas and frequently demonstrated exocytosis and spongiosis (Figs 4-6).

The individual surgical pathology specimens were signed out as furcation granulomas, furcation granulomas with epithelium, or furcation cysts.

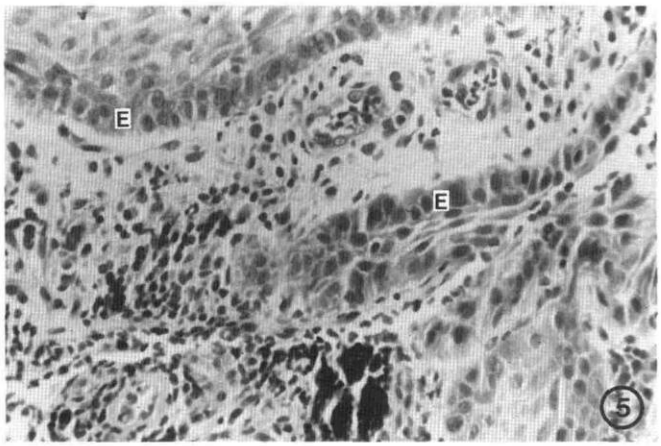

FIG 5. Epithelium of odontogenic cyst wall (E) with chronic inflammatory cells (25 x 1.25 x 3.2 x).

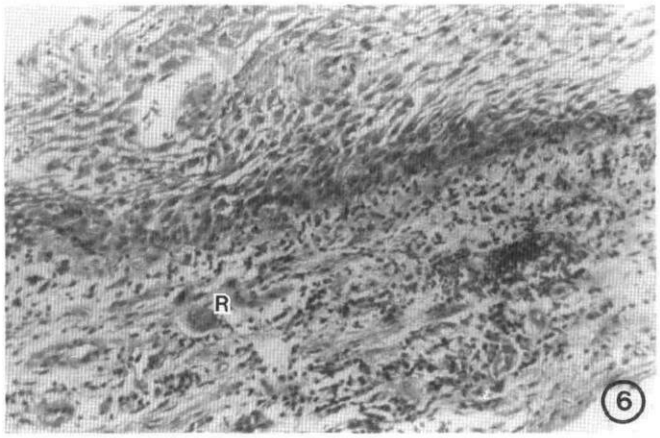

FIG 6. Epithelium in a furcation granuloma with odontogenic cell rests (R) and chronic inflammatory cells (10 x 1.6 x 3.2 x).

Discussion

These observations demonstrate that radiolucent furcation lesions associated with pulp degeneration in a primary molar are mixed and dynamic. Various inflammatory reactions were seen in the same biopsy specimen with granulomatous inflammation as the predominant type. Epithelium was observed in 10 of the 21 specimens, suggesting that these lesions either are odontogenic cysts or have cystic potential. The presence of epithelium was not associated with any particular type of inflammatory reaction. Potential sources of epithelium include remnents of the dental lamina, odontogenic epithelium, or epithelium introduced from the oral cavity.

The histological features observed are essentially the same as those reported for periapical radiolucent lesions associated with permanent teeth.[3] Therefore, the radiolucent furcation lesion associated with pulp pathology in a primary molar is essentially analogous to the periapical lesion associated with permanent teeth. These findings support the recommendation that pulp therapy for primary molars with furcation lesions should be directed toward complete removal of the diseased pulp and obliteration of the root canal space with a biocompatible material, or extraction of the involved tooth.

A limitation of this report is that to avoid possible damage to the developing premolar, the curretting was accomplished very gently. Therefore, the peripheral and deep areas of the lesion may not have been completely included in the biopsy specimens. This may account for the lack of granulation tissue because a dental granuloma would be most amenable to repair by granulation tissue in the peripheral regions.

Radiolucent lesions also are observed with failure of pulp therapy in primary molars. A recent report describes rapidly enlarging cystic lesions following failure of pulpotomy treatment of several primary molars with formocresol or phenol-containing drugs (Grundy and Adkins 1984). Since pulp therapy introduces various medicaments into the tooth, it is possible that the irritant differs following pulpotomy failure leading to a different tissue reponse than observed with these nonpulp-treated teeth. However, the observation of epithelium in 10 of the 21 cases in this report clearly demonstrates the potential for lesions associated with pulp pathology in primary teeth to be cystic. Thus, when root canal therapy is chosen, careful postoperative radiographic evaluation is essential to make sure radiolucent lesions resolve without cyst formation.

Conclusion

The observations in this report demonstrate that radiolucent furcation lesions associated with pulp pathology in primary molars may be classified as furcation granulomas, granulomas with epithelium suggesting the potential for cystic transformation, or furcation cysts. Histological examination, which should be carried out on all soft tissue lesions, is required for a definitive diagnosis.

[3] Block et al 1976; Lalonde and Lueke 1968; Langeland et al. 1977; Weiner et al. 1982.

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AMA proposes AIDS testing

The American Medical Association (AMA) recently adopted the following recommendations to curb the spread of the AIDS virus. They proposed confidential testing for AIDS, with appropriate counseling, on three levels: mandatory; routine, but with patient cosnent; and at the encouragement of doctors.

Mandatory testing

- Donors of blood or blood products, organs, tissues, semen, or ova
- Immigrants to the United States
- Military recruits
- Inmates in state or federal prisons

Routine voluntary testing

- Patients at clinics that treat sexually transmitted diseases
- Patients at drug abuse clinics
- In areas of high incidence of AIDS or for individuals who engage in high-risk behavior; pregnant women in the first trimester of pregnancy; people seeking family planning services; and patients undergoing surgical or invasive procedures (If voluntary policy doesn't work, hospital and medical staff should consider mandatory testing.)

Testing encouraged by a doctor

- Voluntary testing based on a doctor's medical judgment of a person's medical history or health, such as having a rare form of pneumonia