

# Pulp and apical tissue response to deep caries in immature teeth: A histologic and histobacteriologic study



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## ARTICLE INFO

### Article history:

Received 21 July 2016

Received in revised form 3 September 2016

Accepted 9 October 2016

### Keywords:

Dental caries

Immature teeth

Odontoblasts

Radicular dentin

Apical papilla

Hertwig's epithelial root sheath

## ABSTRACT

Descriptions of the pathologic changes in the pulp and associated apical structures of human immature teeth in response to deep caries are lacking in the literature.

**Objectives:** This article describes the histologic events associated with the radicular pulp and the apical tissues of human immature teeth following pulp inflammation and necrosis.

**Methods:** Twelve immature teeth with destructive caries lesions were obtained from 8 patients. Two intact immature teeth served as controls. Teeth were extracted for reasons not related to this study and immediately processed for histopathologic and histobacteriologic analyses. Serial sections were examined for the pulp conditions and classified as reversible or irreversible pulp inflammation, or pulp necrosis. Other histologic parameters were also evaluated.

**Results:** In the 3 cases with reversible pulp inflammation, tissue in the pulp chamber showed mild to moderate inflammation and tertiary dentin formation related to tubules involved in the caries process. Overall, the radicular pulp tissue, apical papilla and Hertwig's epithelial root sheath (HERS) exhibited characteristics of normality. In the 3 cases with irreversible pulp inflammation, the pulps were exposed and severe inflammation occurred in the pulp chamber, with minor areas of necrosis and infection. Large areas of the canal walls were free from odontoblasts and lined by an atubular mineralized tissue. The apical papilla showed extremely reduced cellularity or lack of cells and HERS was discontinuous or absent. In the 6 cases with pulp necrosis, the coronal and radicular pulp tissue was necrotic and colonized by bacterial biofilms. The apical papilla could not be discerned, except for one case. HERS was absent in the necrotic cases.

**Conclusion:** While immature teeth with reversible pulpitis showed histologic features almost similar to normal teeth in the canal and in the apical region, those with irreversible pulpitis and necrosis exhibited significant alterations not only in the radicular pulp but also in the apical tissues, including the apical papilla and HERS.

**Clinical significance:** Alterations in the radicular pulp and apical tissues help explain the outcome of current regenerative/repairative therapies and should be taken into account when devising more predictable therapeutic protocols for teeth with incomplete root formation.

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## 1. Introduction

Immature teeth are characterized by open apices with thin dentin walls. In early stages of root formation, the proliferating end of the Hertwig's epithelial root sheath (HERS), the union of inner and external enamel epithelium, bends at a near 45° angle. This structure is referred to as epithelial diaphragm and is believed to

be responsible for root dentin formation through signaling to undifferentiated mesenchymal cells present in the dental papilla [1]. As the differentiated odontoblasts lay down the primary dentin, the dental papilla becomes encased within the dentin structure. In advanced stages of root development the volume of dental papilla is considerably reduced, restricted to only the apical portion of the root. This structure has been named apical papilla [2]. The apical papilla harbors stem cells that are believed to be capable of differentiating into primary odontoblasts for formation of root dentin [3]. In histologic sections stained with hematoxylin and eosin, the apical papilla appears as an eosinophilic tissue with

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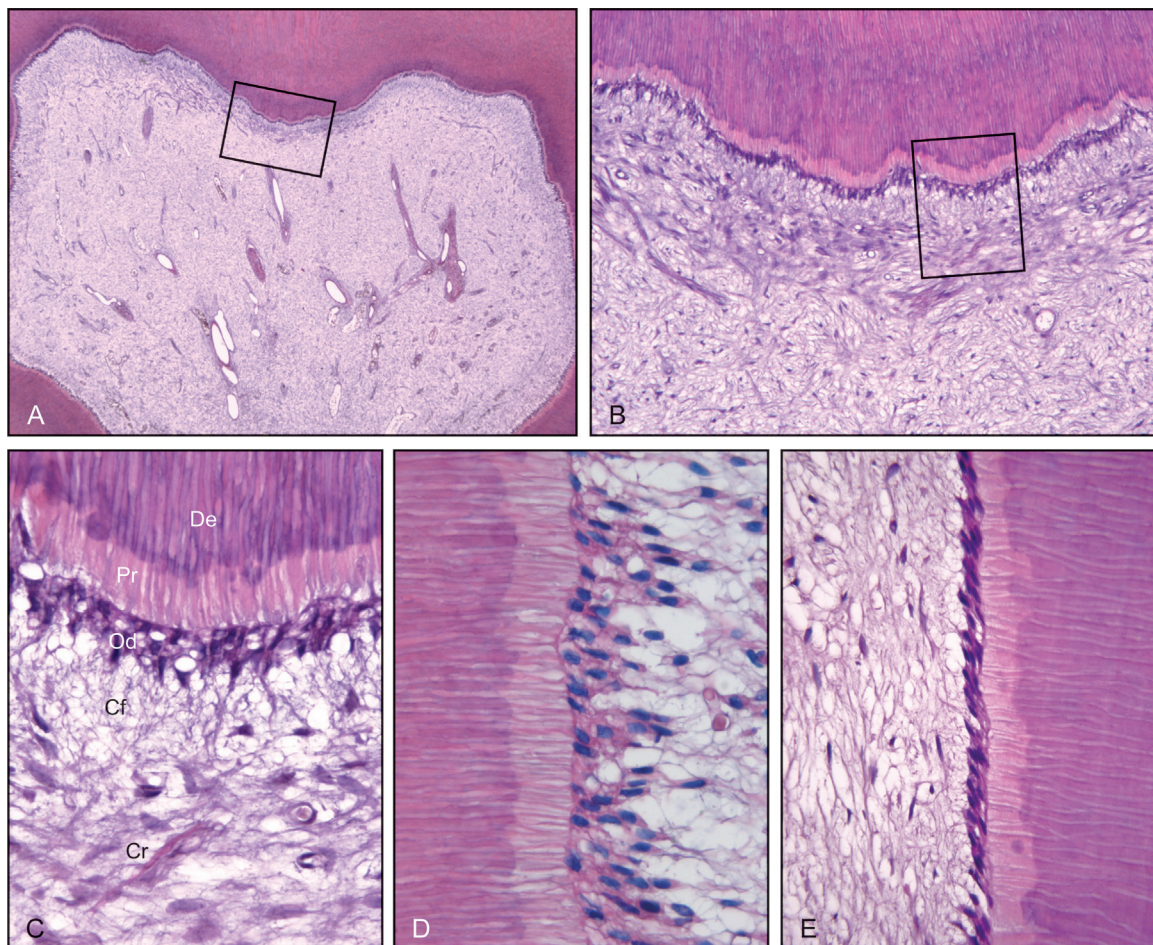
**Table 1**  
Teeth with immature roots and different clinical and histopathological diagnosis. Case description.

Sample	Age	Sex	Tooth	Pulp diagnosis	Periapical diagnosis	Histological pulp diagnosis	Odontoblast changes in canal	Tertiary dentin in canals	Changes in apical papilla	Changes in HERS
1	10	M	#14	Pulp necrosis	Symptomatic AP	Irreversible pulpitis	Yes	Yes	Yes	Yes
2	9	M	#30	Unknown	Symptomatic AP	Reversible pulpitis	Yes	Yes	No	No
3	10	M	#3	Irreversible pulpitis	Symptomatic AP	Irreversible pulpitis	Yes	Yes	Yes	Yes
4	13	F	#4	Reversible pulpitis	Normal apical tissue	Reversible pulpitis	Yes	Yes	Yes	No
5	10	M	#31	Pulp necrosis	Acute apical abscess	Pulp necrosis	Yes	Yes	Yes	Yes
6	9	F	#19	Pulp necrosis	Acute apical abscess	Pulp necrosis	Yes	Yes	Yes	Yes
7	9	M	#19	Unknown	Symptomatic apical periodontitis	Reversible pulpitis	Yes	Yes	Yes	No
8	8	M	#14	Previously initiated therapy	Unknown	Pulp necrosis	Yes	Yes	Yes	Yes
9	9	M	#3	Unknown. Indirect pulp capping	Symptomatic apical periodontitis	Irreversible pulpitis (pulp polyp present)	Yes	Yes	Yes	Yes
10	15	M	#31	Pulp necrosis	Asymptomatic apical periodontitis	Pulp necrosis	Yes	Yes	Yes	Yes
11	7	F	#19	Pulp necrosis	Unknown	Pulp necrosis	Yes	Yes	Yes	Yes
12	7	F	#30	Irreversible pulpitis	Normal apical tissue	Pulp necrosis	Yes	Yes	Yes	Yes

Note: 1: sample ID 1 and 3 were collected from the same patient; 2: sample ID 2, 7, and 8 were collected from the same patient; 3: sample ID 11 and 12 were collected from the same patient.

large cells, separated from the rest of the pulp by a cell-rich zone [2]. HERS can usually be observed layering the external apical surface, sometimes enveloping the thin apical dentin walls.

While tissue response to caries in fully formed teeth has been well documented [4–7], descriptions of the pathologic changes occurring in the pulp and associated immature apical structures, as



**Fig. 1.** Control teeth. (A) Mandibular third molar. Overview of the pulp chamber. Abundance of neurovascular structures (H&E, original magnification  $\times 16$ ). (B) Detail of the area demarcated by the rectangle in (A), depicting the normal morphology at the dentin-pulp interface in an immature tooth (original magnification  $\times 100$ ). (C) High power view of the area demarcated by the rectangle in (B). Dentin (De) with tubules running parallel to each other; predentin (Pr) with uniform thickness; palisading odontoblast layer (Od); cell-free zone (Weil's zone) (Cf); cell-rich zone (Cr) (original magnification  $\times 400$ ). (D) Distal canal. Transition from canal orifice to coronal third. High power view shows parallel dentinal tubules crossing uninterruptedly dentin and predentin. Undisturbed odontoblast layer (original magnification  $\times 400$ ). (E) Distal canal. Transition from middle to apical third. A single row of "spindle-shaped" odontoblasts is present. Tubules with regular course in predentin and dentin (original magnification  $\times 400$ ).

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