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The physiologic sclerotic dentin: A literature-based hypothesis

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ABSTRACT

Despite the many hypotheses trying to explain how the physiologic sclerotic dentin is formed, there has been so far no convincing explanation for all of its observations. In this review, we tried to make a hypothesis based on the facts published to date. We found that the apoptosis of odontoblasts, which takes place after the formation of the apical constriction, may be the key-factor for the development of physiologic sclerotic dentin, because the resulting apoptotic bodies cannot be eliminated through phagocytosis and become trapped within the dentinal tubules due to the continuous formation of secondary dentin. The apoptotic bodies suffer later from a secondary or apoptotic necrosis leading to the release of the internal contents of pyrophosphate and hydrogen phosphate. Pyrophosphate can dehydrate the dentin and hydrogen phosphate can demineralize it, leading to the release of Ca^{2+} ions which then contribute to the intratubular mineralization.

Introduction

The physiologic sclerotic dentin is identified through the accumulation of mineral deposits within the lumen of the dentinal tubules. It begins after 3–4 years from the complete eruption of teeth, first at the apical part of the root then moves coronally, and at the external end of the tubules near the cementum then moves toward the pulp. Although it increases with age, it is not the result of aging process itself because the tubules where the physiologic sclerotic dentin starts to accumulate (i.e. near the apex of the teeth) are the youngest [1].

The characteristic feature of sclerotic dentin is that it reduces the amount of scattered light. Therefore, it has a transparent appearance to transmitted light [1,2]. Since the physiologic sclerotic dentin is located more at the mesial and distal areas of the roots than at the buccal or lingual areas, it appears on the horizontal sections as a butterfly shape (Fig. 1a and b). Moreover, the line where the sclerotic dentin meets the normal dentin looks serrated on the vertical sections (Fig. 2a and b) [1].

When transmission electron microscopy was used, the absence of odontoblasts and predentin was evident where the sclerotic dentin was formed [3–5]. Therefore, it was concluded that the odontoblasts, despite their role in forming the peritubular dentin, do not secret the sclerotic dentin [6]. This conclusion was also supported by the following facts:

- The difference in the appositional manner between the peritubular dentin (clear forming front) and the sclerotic dentin (diffused deposits) [7].
- One of the adaptive changes in dental pulp with age is the reduction of the cellular content including the odontoblasts [8]. Nevertheless, the physiologic sclerotic dentin continues to form even in an increasing rate [1].

Moreover, the role of pulpal extracellular fluid in the mineralization process of sclerotic dentin remains a matter of question. Despite the fact that the extracellular fluid has a concentration of calcium ions that can reach 3 mM [9], which is considered sufficient to induce the mineralization of predentin and demineralized dentin [10], it cannot passively pass through the dentinal tubules, because the odontoblast with its process fills the tubule lumen completely at least in the predentin and the adjacent dentinal layer [6]. Even if there is a discontinuation in this seal of the dentinal tubule in such a way that allows the movement of pulpal fluid, the mineralization will take place first at the pulpal side of the tubule and will be a self-limiting process (i.e. as the calcification at the pulpal side of the tubule increases, it hinders the later flow of the pulpal fluid so that the intratubular mineralization will stop at a certain depth in the tubule). Considering all of the above, the question that may now arise is: which factor is responsible for forming the physiologic sclerotic dentin?

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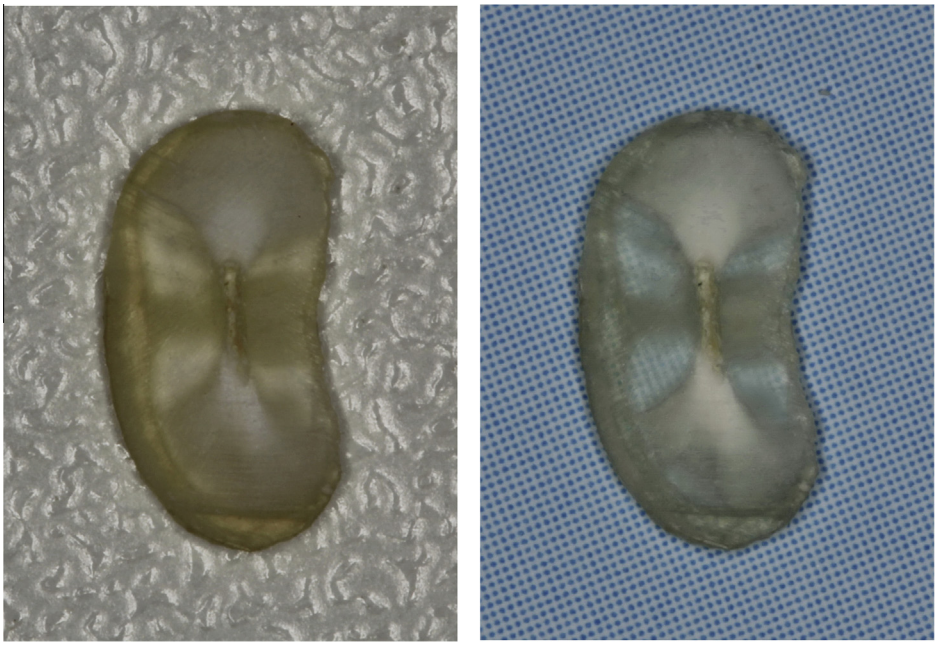

Fig. 1. (a) and (b) The distribution of physiologic sclerotic dentin on the horizontal root section (butterfly shape).

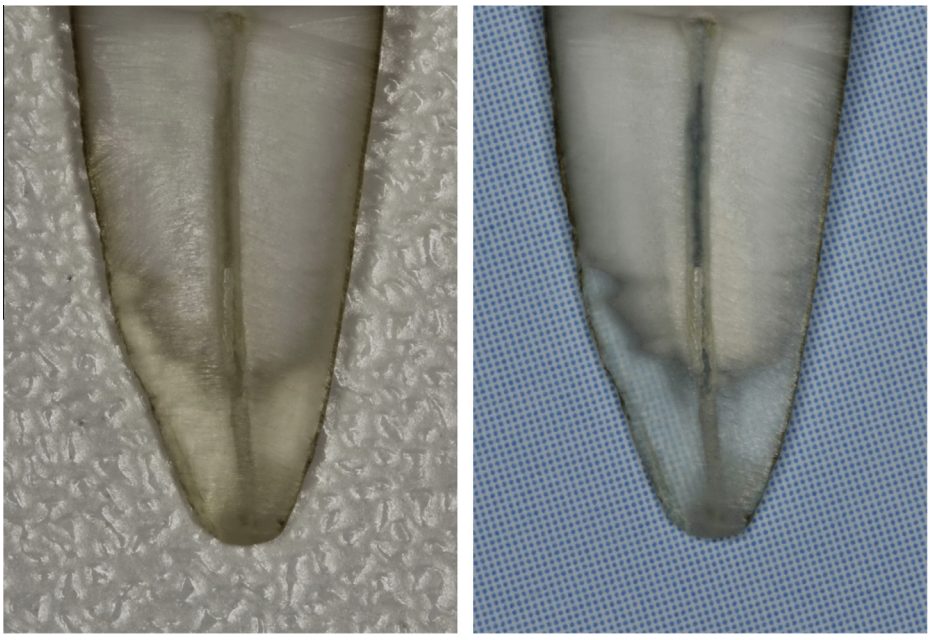

Fig. 2. (a) and (b) The distribution of physiologic sclerotic dentin on the vertical root section (serrated line).

The pulp has relatively high normal tissue pressure and low tissue compliance. Moreover, it is located within the rigid dentinal walls of the tooth, so that any increase in pulpal tissue volume will increase the tissue pressure [11]. This increase in tissue pressure can remain located within the part of the pulp where it was raised without spreading to involve the whole pulpal tissue [12,13].

The increase in pulpal tissue volume can be either true (A direct increase in tissue volume due to increased blood flow and increased capillary filtration in case of pulp inflammation) or relative (An indirect increase in tissue volume due to the reduction in the available space inside the tooth and the physiologic compression of the pulp by the continuous formation of secondary dentin).

The increase in pulpal tissue pressure due to the physiologic compression can be given as follows [14]:

$$\Delta P = B^{*}(-\Delta V/Vi)$$

where ΔP, the increase in pulpal tissue pressure; B, the bulk modulus for pulpal tissue; Vi, the initial volume of pulpal tissue; ΔV, the difference in pulpal tissue volume due to the physiologic compression (The new volume – The initial volume). It is equal to the volume of the formed secondary dentin but it is in this case a negative value. For this reason it was modified by adding a minus.

Considering the fact that the regular secondary dentin forms at a daily rate of 0.8 μm [15], the pulpal tissue pressure will increase the most where the pulp has the smallest volume (i.e. at the apical