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Original research article

Evaluation of dentinal tubules of dentin of the roots of necrotic teeth by means of scanning electron microscope

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ARTICLE INFO

Article history:
Received 8 July 2016
Received in revised form
15 September 2016
Accepted 16 November 2016
Available online xxx

Keywords:
Dentin
Dentinal tubules
Necrotic teeth
SEM

ABSTRACT

Introduction: Dentin is porous due to existence of tubules, containing cell knobs and nerve fibers. It forms integral part of the tooth.

Aim: The aim of the study was measurement of the width of dentinal tubules of the roots of necrotic teeth with chronic periapical inflammation after endodontic treatment as well as differences related to age and gender.

Material and methods: The study comprised 53 teeth (30 molars, 23 premolars) extracted at the Oral Surgery Department, Medical University of Lublin. The teeth were divided into two groups: 40 teeth extracted because of periapical lesions, 15 of which had been treated endodontically before, and the control group of 13 healthy teeth extracted for orthodontic reasons.

Scanning electron microscope was used to evaluate the construction of dentinal tubules. The diameter of dentinal tubules was measured close to the cementum and near the dentinocemental junction.

Results and discussion: The study demonstrated larger diameter of dentinal tubules in necrotic teeth roots compared to the width of tubules of vital teeth. In the control group the mean tubule diameter near the cement was 1.38 μm, and nearby the root canal 3.10 μm. In the group of devitalised teeth the average diameter measured in the same position amounted to 3.68 μm and 4.89 μm.

Conclusions: The width of dentinal tubules of necrotic teeth was significantly higher compared to the teeth with vital pulp. Endodontic treatment does not change the width of dentinal tubules. The width of dentinal tubules does not depend on age and gender.

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http://dx.doi.org/10.1016/j.poamed.2016.11.016

Please cite this article in press as: Jakiel J, et al. Evaluation of dentinal tubules of dentin of the roots of necrotic teeth by means of scanning electron microscope, *Pol Ann Med.* (2016), http://dx.doi.org/10.1016/j.poamed.2016.11.016

1. Introduction

Dentin is a hard, mineralized connective tissue that provides the bulk of the tooth.[1–3] It is located between the chamber filled with dental pulp and tooth enamel at the crown and between the cementum and the pulp of the tooth root. Dentin thickness ranges from 3.0–3.5 mm in the coronal section tapering toward the root apex to 0.8–1.3 mm.[2] Dentin construction resembles bone construction, however, it is much harder. It is characterized by high flexibility and elasticity, which determines its resistance to fracture.[1,2,4]

A characteristic feature of dentin is high porosity due to channels passing through the entire thickness thereof. The diameter of the tubules tapers toward the periphery; tubules adjacent to the pulp have a diameter of 2.0–3.0 μm and taper to about 0.6–0.8 μm close to the dentinocemental junction. Also the number of the tubules changes depending on the distance from the pulp chamber. Close to the chamber there are approximately 48 000 tubules/mm^2, in the middle approximately 37 000 tubules/mm^2, and close to the dentinocemental junction around 22 000/mm^2.[1,4,5]

Dentin–pulp complex and interdependence between the two tissues are claimed to be an integral part of the tooth. Pulp and dentin are embryologically, histologically and functionally the same tissue and therefore are considered as a complex. Both tissues derive from mesenchymal dental papilla (*papilla dentis*). Pulpo-dentin complex is one structure due to pulp cell knobs and nerve fibers placed within dentinal tubules. Dentin sensitivity to stimuli depends on the pulp and pulp reactions depend on changes in dentin. The interdependence of the two tissues is observed in inflammatory processes of the pulp.[1,2,6,7]

Pulp damaging factors include attrition, erosion, abrasion, recession, tooth decay, as well as mechanical, chemical and thermal factors. In case of minor damage to the tooth higher concentration of transforming growth factor β (TGF-β) in the pulp chamber occurs. TGF stimulates the odontoblasts lying around the damaged tissues and therefore an increase in the secretion of building factors leading to the formation of reparative dentin appears. When the pathogen exceeds the ability of the tooth to defend, inflammation of the pulp develops. Inflammatory response begins at the approximation of pathogens at a distance of 0.5 mm from the pulp chamber.[8] Inflammation is accompanied by pH decrease of the interstitial fluid in the pulp and dentinal tubules. Upon destruction of odontoblastic layer, TGF-β stimulates odontoblast precursor cells present in the pulp to proliferation and chemotaxis. The cells take the place of the destroyed odontoblasts and start to accumulate tertiary dentin.[9]

Pulp inflammation in the beginning is limited only to the action of toxic agent, but after 72 h a spread of inflammation throughout the pulp and destruction of the odontoblastic layer appears. In response to the action of bacterial toxins vasoconstriction and increased tissue fluid flow in the outer layers of the dentinal tubules begin. Increased tissue perfusion reduces toxicity and removes bacteria from the tissues. At the same time cellular and immune response are activated. Odontoblasts begin to produce numerous growth factors such as bone morphogenetic protein 2 (BMP-2), fibroblast growth factor (FGF), epidermal growth factor (EGF), insulin-like growth factor (IGF), TGF-β which participate in mineralization of dentin matrix. During pulp inflammation concentration of the inflammatory mediators as histamine, serotonin, and prostaglandins increases. Inflammatory markers have a tendency to diffuse through the tubules into saliva surrounding the tooth.[10]

After vasoconstriction, vasodilation occurs, leading to the accumulation of red blood cells in the central part of blood vessels and leukocyte migration and adhesion to the walls of the vessels. The next step is formation of cracks in vascular endothelium, leading to an increase in filtration of plasma with lymphocytes, macrophages and plasma cells into surrounding tissue. Increase in the volume of fluid in the tooth chamber contributes to swelling of the pulp and increased pressure in the tissue causing irritation of nerve endings, which in consequence causes pain. Swelling also contributes to venous clamp and impossibility to drain excess of fluid, which leads to further increase in pressure and, consequently, to necrosis of the pulp because of tissue hypoxia. Pressure increase in the chamber is associated with irritation of nerve endings, causing on the one hand increased capillary flow, on the other hand activation of neurokinin increasing pressure in the vessels. During pulp inflammation hemorrhage, destruction of odontoblasts and inflammatory infiltration of lymphoid cells occur.[7,10,11]

Studies have shown that the presence of bacteria in dentinal tubules reduces tubular fluid, contributing to growth of bacteria and increased concentration of toxic substances that lead to tissue destruction.[3]

When the inflammatory process is long-lasting, a tooth-like granulation tissue will appear within the chamber. The tissue is characterized by a large number of blood vessels, cells and collagen fibers. The primary role of granulation tissue is to repair damaged tissue.

Untreated inflammation of the pulp leads to apical inflammation. The pathological process spreads via blood vessels. Initially the vessels unclench, their permeability increases and cells and inflammatory mediators pass into periapical space.[10,11]

2. Aim

The aim of this study was measurement of the width of the root dentinal tubules of necrotic teeth with chronic inflammation of periapical tissues, after endodontic treatment as well as differences related to age and gender.

3. Material and methods

The study material comprised 53 teeth (30 molars, 23 premolars) extracted in patients at the Oral Surgery Department, Medical University of Lublin. The teeth were extracted in patients reporting no general disease.

The teeth were divided into two groups. The examination group (Group W) consisted of 40 teeth extracted because of periapical inflammatory lesions (17 gained from females, 23 from males). Patients included in the group aged between 20

Please cite this article in press as: Jakiel J, et al. Evaluation of dentinal tubules of dentin of the roots of necrotic teeth by means of scanning electron microscope, *Pol Ann Med.* (2016), http://dx.doi.org/10.1016/j.poamed.2016.11.016