

Putative role of epithelial rests of Malassez in alleviation of traumatic occlusion[☆]

M.B. Helal^{a,*}, M. Abd-Elmotelb^a, N.H. Sarhan^b, N.B. Nagy^a

^a Oral Biology Department, Faculty of Dentistry, Tanta University, Tanta, Egypt

^b Histology and Cell Biology, Faculty of Medicine, Tanta University, Tanta, Egypt

Received 21 April 2015; revised 16 August 2015; accepted 19 August 2015

Available online 23 October 2015

Abstract

Background: Epithelial cell rests of Malassez (ERM) are odontogenic epithelial cells located throughout life within the periodontal matrix. It has long been thought to be a functionless remnant, though recent studies suggested their role in periodontium regeneration and homeostasis.

Aim of study: The aim of the present study was to investigate the role of ERM in alleviating the deteriorating effect of an induced primary acute traumatic occlusion in rats.

Material and methods: Twenty four, 7 month old male rats were used in this study. Animals were randomly divided into two equal groups, a control and traumatic occlusion groups. Rats in the latter group had the occlusal surfaces of their right maxillary first molars unilaterally raised 1–2 mm with our innovative 7/8 nickel chrome stainless steel crowns that had free distal surfaces. Three rats, from each group, were euthanized at 1, 3, 6, and 9 weeks. Half of their right mandibular first molar-specimens were processed for light microscope (LM) and the other for transmission electron microscope (TEM).

Results: Rats in the control group revealed no difference in their normal ordinary periodontal ligaments (PDL) structure, by LM, at all times. Ultrastructurally, all samples revealed similarity in having closely approximated resting ERM cells. On the other hand, after one and three weeks of traumatic occlusion, there were mild progressive PDL disorganization, cementum resorption together with phagocytic and secretory ERM cells, respectively. Interestingly, at later stages on the 6th and 9th week, signs of repair and remodeling of PDL structure were manifested along with the establishment of closely juxtaposed clusters of ERM with apoptotic and secretory characters, respectively.

Conclusion: ERM cells appeared to have an important role within the periodontal apparatus. Their diverse ultrastructural features during periodontal deterioration and regeneration would suggest their role in alleviating the injury resulted from induced traumatic occlusion.

© 2015, Hosting by Elsevier B.V. on behalf of the Faculty of Dentistry, Tanta University.

Keywords: ERM; Cell rests of Malassez; Periodontium; PDL; Traumatic occlusion

[☆] This article is part of the MSC thesis of the first author, presented to faculty of Dentistry, Tanta University.

* Corresponding author.

E-mail addresses: Mai_badreldin@yahoo.com (M.B. Helal), mon_assem@hotmail.com (M. Abd-Elmotelb), nsarhan2006@hotmail.com (N.H. Sarhan), histodent@hotmail.com (N.B. Nagy).

Peer review under the responsibility of the Faculty of Dentistry, Tanta University.

1. Introduction

After complete crown formation, cells of inner and outer enamel epithelia develop Hertwig's epithelial root sheath (HERS) that has a crucial role in mapping and inducing radicular dentin formation. Remnants of HERS typically reside and make a unique integral component of periodontal ligament (PDL) known as epithelial rests of Malassez (ERM) [1]. Unlike the typical nature of epithelial tissue which is commonly defined as a sheet covering or lining body surfaces or cavities, ERM are sited away from the surface and completely interposed between the incessantly remodeled alveolar bone and the continuously deposited radicular cementum [2].

Histologically, ERM cells can be identified easily as small clumps of epithelial cells consisted of 10–20 cells, usually oval or round in shape within the PDL adjacent to the cementum surface. Occasionally, some of these cells were detected linked to cementoblasts or embedded in cementum and progressed to apoptosis [2]. Generally, ERM cells make a fenestrated network-like pattern that appeared in longitudinal section as a discrete cluster of epithelial cells [3,4]. Also, they were described to form a basketball hoop around the neck of the tooth [5]. In 3D reconstructions, ERM appeared as a series of interconnected helical threads running from dento-gingival attachment to the root apex [6]. Ultrastructurally, ERM in human PDL consisted of clusters of cells surrounded by a basal lamina. Characteristically, ERM had an irregular nucleus with condensed heterochromatin, tonofilaments, and poorly developed rough endoplasmic reticulum (RER). ERM cells were connected to each other by desmosomes and gap junctions. In addition, a primary cilium was often observed accompanied by a centriole in association with Golgi complex and rootlet-like structures suggesting that ERM are more than a vestigial structure [7].

Recent studies claimed that ERM cells could play a pivotal role in the development, maintenance and regeneration of periodontal ligament tissues [8–10]. Acellular cementum was claimed to be synthesized by HERS-derived cementoblasts [4]. Also, Fong et al. suggested that ERM played a critical role to resume the integral relation between tooth and its supporting structures [11]. In retained deciduous teeth with infra occlusion and over retention, ERM were found to inhibit ankylosis [8]. Moreover, ERM were found to be linked with periodontal regeneration and cementum repair [12,13].

Interestingly, Akimoto et al. claimed that HERS/ERM cells represented a heterogeneous population of

cells, though there has been controversy over their epithelial–mesenchymal transition (EMT) [14]. In human, this EMT could be induced by TGF- β 1 [15]. Porcine ERM could differentiate in vitro into ameloblast-like cells and generate enamel-like tissues in combination with dental pulp cells [16]. Also, human ERM were found to express amelogenin, ameloblastin, matrix metalloproteinase-20 (MMP-20), and kallikrein-4 (KLK-4) [17]. In addition, ERM contained a subpopulation of stem cells that could undergo EMT and differentiated into mesenchymal stem-like cells with multilineage potential. Using cell culture system, ERM formed bone, fat, cartilage, neural cells in vitro [18]. Also, when transplanted into immunocompromised mice, ERM generated bone, cementum-like and Sharpey's fiber-like structures [18].

Occlusal trauma was defined as injury to the periodontium resulting from occlusal forces that exceeds its reparative capacity. Historically, trauma from occlusion was classified as either primary or secondary. Primary occlusal trauma results from excessive occlusal force applied to teeth with healthy supporting tissues. While secondary occlusal trauma refers to changes which occur when occlusal forces are applied to teeth with inadequate supporting tissues [19]. More usefully, occlusal trauma was described as acute or chronic. While the latter develops from gradual changes in occlusion produced by tooth wear and drifting movement, acute trauma is due to an abrupt increase in occlusal load as a result of biting unexpectedly on a hard object Restorations and prosthetic appliances, which interfere with normal occlusion could cause acute trauma [20].

Since traumatic occlusion is known to disturb PDL hemostasis, the aim of the present study was to investigate the putative role of ERM in alleviation of the effect of an induced primary acute traumatic occlusion on rat periodontium.

2. Material and methods

2.1. Animals

Twenty four adult male rats, weighing 200–250 g were used in this study. They were kept under controlled temperature and lighting conditions with free access to standard food and water ad-libitum. Animals were randomly divided into two equal groups. Group-I (control), in which rats received no dental treatment. Group-II (traumatic occlusion), whose rats were subjected to primary acute traumatic occlusion. This was introduced through the usage of

Download English Version:

<https://daneshyari.com/en/article/3179605>

Download Persian Version:

<https://daneshyari.com/article/3179605>

[Daneshyari.com](https://daneshyari.com)