

Ligneous Periodontitis

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Abstract

Ligneous periodontal disease is a rare condition characterized by membranous gingival enlargement. Ligneous mucosal disease leads to rapid tooth loss by increased bone resorption. In the literature, it has been shown that different therapies for ligneous lesions have usually resulted in limited success. In this review, it was aimed to evaluate the pathogenesis of ligneous lesions based on the current literature knowledge.

Keywords: *Ligneous Periodontitis; Gingival Hyperplasia; Plasminogen Deficiency; Periodontal Treatment*

The term "ligneous periodontitis", earlier known as "amyloidaceous ulcerated gingival hyperplasia" or "destructive membranous periodontal disease", was first used by Günhan, *et al.* [1]. Destructive membranous periodontal disease, or ligneous periodontitis (LP) is a progressive entity which is a part of a systemic disease caused by plasminogen deficiency (PLGD) and fibrin deposition [1,2]. It is an analog lesion of ligneous conjunctivitis and it is a rare condition characterized by gingival enlargement, gingival swelling and aggressive periodontal tissue destruction [3-5]. Hyperplastic generalized gingival lesions have also been reported in association with a form of chronic conjunctivitis known as ligneous conjunctivitis, with ligneous eyelid lesions associated with the development of a fibrin-rich, woody-like pseudomembranous layer [1,6,7]. Scully, *et al.* [8] reported that gingival overgrowth with ulceration appears to be a complication of PLGD it also appears to be related to ligneous conjunctivitis in some cases. PLGD and impaired degradation of fibrin lead to extensive fibrin accumulation, and this constitutes the typical clinical characteristics of LP and/or ligneous conjunctivitis [9,10].

Studies have demonstrated that pseudomembrane formation can also develop on the mucosa of the oral cavity, the laryngo-tracheo-bronchial tree, the nasopharynx, the middle ear, the kidney, the peritoneum, the mastoid system and the female genital tract (ligneous vulvovaginitis or cervicitis) [3,11,12]. Initially, most patients suffer from conjunctival or gingival symptoms because these sites are easily exposed to external factors and injuries [13]. It has been suggested that in some predisposed cases, poor oral hygiene, local factors such as infections, irritations, spicy food, minor trauma, hypersensitivity and surgical procedures may contribute to the formation of new ligneous lesions [12,14].

Plasmin is a serine protease released into the systemic circulation as a precursor enzyme known as plasminogen, which is primarily synthesized in the liver [15,16]. Plasminogen is converted into plasmin by two types of plasminogen activators: tissue plasminogen activator and urokinase plasminogen activator. Inhibition of this activation process is controlled by plasminogen activator inhibitor-1 [15,17]. This leads to efficient lysis of fibrin clots in the blood stream and has an important role in extracellular matrix proteolysis, cell migration, angiogenesis, embryogenesis, and the activation of growth factors and matrix metalloproteinases, which promote cellular adhesion and wound healing [18]. Type-I PLGD (hypoplasminogenemia), which is the type most often associated with ligneous lesions, demonstrates autosomal-recessive inheritance which should be confirmed by molecular genetic analysis. It has been reported that the prevalence of type-1 PLGD is $\approx 0.3\%$ to 0.4% of the general population [18,19]. However, the theoretically predicted prevalence of these

individuals with homozygous or compound-heterozygous mutations has been observed to be in the range of 1.6 per 1 million people in Europe [19]. In patients with PLGD, the wound-healing capability seems to be halted at the stage of granulation tissue formation. Therefore, in affected areas, ligneous lesions are typically rich in fibrin due to the lack of proteolytic capacity [20]. Previous histopathological studies have suggested that the histological features of ligneous lesions are characterized by excessive fibrin exudation, surface ulceration, hyperkeratosis, acanthosis, epithelial downward proliferation, polymorphonuclear leukocyte-rich exudate with intraepithelial edema and subepithelial nodular amorphous, homogeneous, amyloid-like, eosinophilic material accumulation that does not stain with Congo red stain [1,21].

In the literature, several periodontal and surgical treatment alternatives for ligneous lesions have been tried but have been only partially successful, and therapy has usually resulted in limited success and a loss of teeth. Periodontal treatment of gingival lesions was performed with oral hygiene care, scaling and root planing, subgingival curettage, chlorhexidine rinsing, gingivectomy and systemic tetracycline therapy but these attempts were failed and resulted in the rapid regrowth of pseudomembranes and eventual tooth loss [1,3,11,12,20]. In addition to these treatment protocols, systemic and topical corticosteroids, low-dose doxycycline and warfarin were prescribed for LP treatment but these protocols had limited clinical benefit [21,22].

Conclusion

Ligneous periodontitis is a difficult condition to manage. Although it still has no certain treatment options, intensive periodontal health care is essential for LP patients. Further investigations are needed focusing on the development of an effective therapy of LP.

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