

Assessment of the impact of some local risk factors on periodontal health - case report

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Abstract: *The constant interaction between the subgingival bacterial biofilm and the host immune response leads to the clinical onset of a variety of signs and symptoms of periodontal disease. However, the disease can manifest differently from individual to individual, in terms of severity and rate of progression. This can be explained by the predisposing activity of certain local risk factors. These local risk factors can facilitate bacterial biofilm accumulation, complicate good oral hygiene methods, increase the mechanical stress on teeth or inflict physical damage to the periodontal tissues. In all, their combined action can have a significant predisposing and facilitating effect on the onset and evolution of periodontal disease. If left unmodified and uncorrected, such local risk factors can contribute decisively to the triggering of severe and rapidly progressive forms of periodontal disease, as depicted in the exhibited case report.*

Key Word: *Periodontal Disease, Local Risk Factor*

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I. Introduction

Periodontal disease (PD) is an inflammatory disease that affects the supporting tissues of the teeth being caused by the subgingival accumulation of bacteria, taking the form of bacterial biofilm attached to the tooth surface or floats within the gingival sulcus. [1] The clinical manifestations and progression of periodontal disease may vary from individual to individual, being primarily influenced by the microbial composition of the subgingival biofilm and the type and intensity of the host immune response, and secondarily, by a series of local and systemic risk/predisposing factors. [2] Thus, the clinical features of periodontal disease can range from a slight inflammation of the marginal gingiva (gingivitis) to the severe resorption of the alveolar bone and consequent loss of teeth (periodontitis). [2]

In general, local risk factors act as bacterial biofilm retentors or prevent the patient from efficiently and thoroughly removing plaque deposits. [3,4] These local risk factors include morphological abnormalities, such as crowded teeth and malocclusions, dental treatments, such as orthodontic and prosthodontic appliances and mineralized biofilm deposits (dental calculus). [5] However, certain local risk factors act as direct influencers on the biological and mechanical status of teeth, as they induce significant mechanical stress. These stressful circumstances are represented by the violation of the biological width by the subgingival margins of dental crowns and by the generating of exceeding, over-threshold, occlusal forces. [6] Such supraliminal occlusal forces can be generated either by patient's malocclusions or by improperly designed prosthetic appliances. In any situation, the adaptive capabilities of the tooth and its supporting tissues (periodontal ligament and alveolar bone) are over-whelmed, resulting in their increasing damage and consequent extraction. [7] Fortunately, most local risk factors are modifiable and correctable so as to offer the practitioner a favorable status for perfect plaque control, resolution of inflammation and regeneration of periodontal tissues. [5]

The purpose of this case-study is to illustrate a series of local risk factors that contributed to the onset of severe periodontitis and to offer a good understanding of the pathogenic processes that lead to the exhibited clinical manifestations.

II. Case Report



Figure 1 – Intraoral view of the patient's periodontal and dental status

A 46-old male patient was referred by his general dentist to the Department of Periodontology of University of Medicine and Pharmacy of Craiova, Craiova, Romania, for periodontal assessment, diagnosis and treatment. The chief-complaint of the patient was, quote, “significant gingival bleeding when eating and brushing and an intense sensation of gingival burning”. The anamnesis of the patient revealed that, at the moment of examination, the patient was not suffering from any systemic disease, nor that was he under regular medication of any type. However, the patient was a smoker (consuming an average of 10 cigarettes per day) and enjoyed a fat-, sugar- and carbohydrates-rich diet. The patient declared that from his knowledge, no close members of his family suffered from periodontitis. The dental history of the patient revealed that, about one year ago, he underwent a bi-maxillary oral rehabilitation procedure with fixed prosthodontics. This therapy was needed so as to bridge the edentulous areas of the dental arches, which suffered dental extractions at various moments in the past. The patient recalls that all dental extractions were performed by a dentist for extensive dental destruction and that no teeth became mobile and loose by themselves. After approximately 4 weeks of the dental bridges' definitive cementation, the patient started to feel a significant gingival discomfort. Unfortunately, the patient had to leave the country for work-related reasons, and returned after one year. The patient declared no known allergies to any food or drug.

Upon performing the intra-oral examination and a panoramic radiograph, the following clinical signs and symptoms were identified and a diagnosis was reached.

i) On the maxillary dental arch:

The color of the marginal gingiva (and inter-dental papillae), adjacent to the full-arch dental bridge, was bright-red, whereas the color of attached gingiva was close to normal (coral pink). The gingival papilla between the 1.1 and 2.1 teeth was increased in volume and expressed an edematous appearance. Other gingival papillae appeared slightly retracted. The attached gingiva lost its “stippling” texture and appeared smooth and soft, with little elasticity. Using an exploratory probe, the subgingival margins of the dental crowns were assessed, resulting that all of the crowns had over-extended subgingival margins, reaching the junctional gingival epithelium, positioned at the base of the gingival sulcus. Moreover, the dental bridge in the second quadrant had become deluted and was easily removed during the examination.

ii) On the mandibular dental arch:

All gingival tissues (marginal gingiva, attached gingiva, gingival papillae) exhibited a dark-red, almost cyanotic color, extending the entire dental arch. The frontal gingival papillae (between the mandibular incisors) were severely affected by recession (about 3-4 mm from the tooth cervical margin). On the contrary, the gingiva adjacent to the mandibular canines appeared to have an increased volume, a McCall gingival rolled festoon being observed on both teeth. Significant plaque and calculus deposits were observed on all aspects of the frontal mandibular teeth (between 3.3 and 4.3). The gingival tissues had smooth and soft texture and their regular, undulated, shape was severely damaged by inflammation. The central mandibular incisors had shifted against each-other, creating important gaps between them and the lateral incisors. As described by the patient, this had significantly affected his normal speech and voice tonality. The dental bridge in the third quadrant exhibited mobility on the distal abutment, as so did the dental bridge in the fourth quadrant on the mesial one. However, the two bridges were not deluted.

iii) Occlusion

The static occlusion assessment showed that the patient had a significant end-to-end bite, the patient stating that this aspect had been present since the eruption of the teeth, generating traumatic contacts between the incisal edges of the upper and the lower incisors and cuspids. This chronic dental occlusion trauma was exacerbated by working-side interferences (cuspid and double-bicuspid guidance) on the lateral teeth during the lateral movement of the mandible (as observed during dynamic occlusion tests). The applying of the fixed dental bridges did not correct any of these issues.

iv) Periodontal probing

Upon performing full-mouth periodontal probing, multiple deep periodontal pockets (periodontal pocket depth ≥ 7 mm) were identified, on teeth positioned on all quadrants. The most severely affected teeth were 1.8, 1.7, 3.8, 3.1 and 4.4, with probing depths of at least 8 mm and stage 3 mobility. The bleeding on probing index was 85%, while the plaque index was 53%. Furcation defects were also identified on 1.8, 1.7, 3.8, on these teeth the furcation being completely exposed.

v) Radiological evaluation

After completion of the clinical examination, a panoramic radiograph was performed, so to asses to severity of alveolar bone resorption. Horizontal alveolar bone resorption was generalized, in various degrees of severity. The most severely affected teeth (1.8, 1.7, 3.8, 3.1 and 4.4) had alveolar bone loss extending to the apical third of their roots. This raised the possibility of mixed endodontic-periodontal lesions, causing retrograde pulpitis in affected teeth with no previous root-canal treatment. Vertical alveolar bone resorption was also observed in certain teeth (3.1, 4.4), together with significant proximal carious lesions (as those between 1.8 and 1.7). The panoramic radiograph also clarified the fact that, in most abutment teeth, the subgingival margin of the dental crowns was too close to the alveolar bone crest.



Figure 2 – Panoramic radiographic evaluation of the patient, after removal of dental bridge in the second quadrant

vi) Diagnosis

According to the 2017 Classification of Periodontal Diseases, [8] issued by the European Federation of Periodontology, it was established that the patient suffered from Generalized Periodontitis, Stage 4, Grade C. This translates to a generalized, severe periodontitis, with a rapid rate of progression. The current periodontal status of this smoker patient was unstable, with importantly active local predisposing/risk factors: calculus deposits, occlusal trauma and vicious dental contacts and dental crowns which invade the biological width.

III. Discussion

After setting a certain periodontal diagnostic and identifying the local risk factors that acted as predisposing elements in the pathogenesis process of periodontal disease, it is important to discuss the mechanisms that these factors induce on the periodontium.

The risk of periodontal diseases in smokers can be at least as twice as high than in non-smokers [9], and on this background, the local factors could have a much more negative impact on the periodontium.

Occlusal trauma can have multiple causes, ranging from the abnormal development of the dental arches and tooth eruption, to edentulous areas which have not been fitted with dental prosthesis or that have been incorrectly constructed dental bridges upon. [10] In this case, the patient exhibited several edentulous areas, which were given late dental bridges, allowing the abutment teeth to migrate and shift their axes. Consequently, the shifted teeth axes lead to excessive mechanical forces to be applied on the abutment teeth, when biting down or chewing. It has been shown that, a misbalance of pressure and apposition forces leads to alveolar bone significant resorption, tooth mobility and eventually, tooth loss. [11] This pathogenic process was accompanied by rich subgingival plaque deposits, which caused the formation of periodontal pockets and gingival attachment loss. In addition, the patient also exhibited static traumatic occlusion (end-to-end) that generated similar excessive mechanical forces, contributing to the severe gingival retractions in the mandibular incisors. To increase the severity of the situation, the dental bridges which the patient received failed to correct the pre-existing occlusal trauma, on the contrary adding and increasing the mechanical stress that the teeth were subjected to and significantly contributing to the onset and evolution of very severe periodontitis.

As shown by the examination of the dental crown's subgingival margins and the radiologic image, it was clear that the biologic width was invaded on the abutment teeth. The marginal gingiva surrounding the frontal maxillary teeth shows clear signs of acute inflammation. According to the literature, a minimum of three mms should exist between the subgingival margin of a dental crown and the alveolar bone crest. [6] If placed incorrectly, meaning too deep inside the gingival sulcus, the crown margins will act as bacterial plaque retentors and violate the biological width (the junctional gingival epithelium and underlying connective tissue, extending from the base of the gingival sulcus to the alveolar bone crest). [12] Consequently, the gingival margin will become inflamed, retract and the alveolar bone crest will lose its mineral composition and eventually, reduce its height, as seen on the radiologic panoramic image. [12]

The patient also exhibited important calculus deposits, particularly on the lingual and interproximal aspects of the mandibular incisors. Calculus acts as a considerable risk factor for periodontal disease onset, because its outer surface will always be covered with fresh, unmineralised bacterial biofilm, therefore acting as an ideal plaque retentor. [13, 14] This motivates the thorough removal of subgingival and supragingival calculus deposits as a key stage in periodontal therapy protocols. [15]

IV. Conclusion

To conclude, it can be said that, unfortunately, the patient was confronted with a periodontal storm. The end-to-end malocclusion was not orthodontically redressed and the occlusal mechanical misbalance was further exacerbated by the inadequate fixed-dental bridges. To add to the unfavorable situation, this smoker patient also showed very poor plaque control with important calculus deposits. This case illustrates that local risk factors can significantly contribute to the development of severe forms of periodontal disease.

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