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Can Systemic Risk Factors Significantly Impact The Periodontal Status Of An Elderly Patient? A Case Report

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Abstract: *When periodontal subgingival bacteria enter the gingival tissues, an inflammatory reaction is triggered, aimed to restrain and to undo the microbiological challenge. The efficiency of this host immune response is paramount for determining the type of evolution and rate of progression of periodontal disease. At this moment, the bacterial challenge may seem to meet a certain counterattack and consequent elimination, but the host immune response is predisposed to the influence of multiple systemic factors that can alter its efficacy and intensity. Thus, the regular immune mechanisms may be inefficient against periodontal pathogenic challenge, and consequently such systemic elements are considered to be risk factors for the onset, evolution and progression of periodontal disease. These systemic risk factors include age, systemic conditions, medication, hormonal changes, smoking, diet and stress. As reflected by this case report, the influence of systemic risk factors can be significant on the periodontal status of a patient, particularly, an elderly one.*

Key Word*: Periodontal Disease, Systemic Risk Factors, Systemic Disease, Periodontal Status*

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I. Introduction

As subgingival bacterial biofilm deposits are left undisrupted, periodontal pathogenic bacteria begin to enter the gingival tissues through micro-ulcerative lesions within the non-keratinized internal and jonctional gingival epithelia. [1] This event triggers host immune inflammatory reactions, mediated by numerous pro-inflammatory cytokines and enacted by various defensive cells, such as lymphocytes, neutrophils and macrophages. [1] The initial stage of gingival inflammation may lack clinical signs, but as the bacterial challenge continues and the inflammatory reaction intensifies, significant gingivitis symptoms will occur. [2] In an individual with a good and stable immune response and, if there is good plaque control, this can be achievable and the PD's evolution may be reversed. [3] However, the type and intensity of the host immune response can be highly influenced by general factors, such as age, smoking, systemic disease, medication and even stress. [4] As these elements act as perturbations of the host's immune response efficiency and physiological mechanisms, they are also considered systemic risk factors that predispose the patient to PD's increased severity and rapid evolution rate. [4]

Certain systemic risk factors are embedded in the patient's genetic code and constitution, such as gender and age, and thus are unmodifiable, but have a major impact on the individual characteristics of the host immune response and the quality of periodontal structures, which are genetically determined. [5] Other systemic risk factors may occur spontaneously or are a consequence of the aging process, such being the case of general diseases. [6] Intensive scientific research has proven that there is a significantly influencing relationship between PD and diabetes mellitus, cardiovascular diseases and rheumatoid arthritis, some of these links being bi-directional. [6, 7] These correlations have been reunited under the concept of "periodontal medicine". [7] Other systemic diseases, such as leukemia and anemia, directly modify the defensive capabilities of the body, as do hormonal changes, which can also influence the physiology of the periodontal structures (e.g. bone metabolism in osteoporosis). [8] Not only disease, but also medication is a frequent systemic risk factor for PD onset, mainly drugs which are administrated for elevated blood tension (Calcium-channel-blockers), epilepsy (Phenytoin) and immuno-suppressors (Cyclosporine). [9] Fortunately, other systemic risk factors may be easier to control and adjust. These include smoking, nutritional misbalances and stress. [10]

The aim of this case report is to illustrate the impact that systemic risk factors can have on the clinical periodontal status of a patient and to discuss the mechanisms which may underlie the complex interactions between the pathogenic processes generated by these factors and consequent PD's signs and symptoms.

II. Case Report

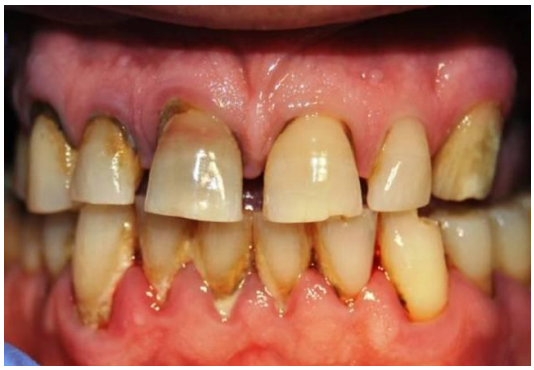

Figure 1 – Initial visit: intraoral view of the patient's periodontal and dental status, after removal of second quadrant bridge

A 63-year-old male patient addressed the Department of Periodontology of the University of Medicine and Pharmacy of Craiova, Craiova, Romania, his chief complaint being, quote, "high teeth sensibility when drinking cold beverages or eating sour foods and high mobility of upper left dental bridge". The anamnesis of the patient revealed that he was suffering from elevated blood pressure and for this reason he had been under constant medication with Calcium-channel-blockers (Nifedipine) for about 10 months. Besides the medication needed for the elevated blood pressure, the patient was not taking any other drugs. The patient also declared that he was a heavy regular smoker, consuming more than 20 cigarettes per day. Besides smoking, the patient is also a regular light alcohol consumer and enjoys a fat-rich diet. To the patient's knowledge no close relatives suffered from periodontal disease. Regarding his dental history, the patient declared that in the past, he had done regular visits to his dentist for various dental complaints and treatments. This included dental extractions of the maxillary bilateral molars, due to pulp complications of carious lesions, and consequent fitting of dental bridges in the resulting edentulous areas. The patient also suffered a frontal trauma to the maxillary incisors, which required a root canal treatment on the 1.1 tooth, with a stable outcome. Unfortunately, five years ago the patient had to move abroad for work purposes and ceased regular visits to his dentist. The patient declared that he suffered from no food or drug allergies.

The anamnesis was followed by an intra-oral examination, accompanied by a panoramic radiograph. The patient also issued his informed written consent. This enabled the detection of clinical signs and symptoms of PD and to reach a clear diagnosis:

i) On the maxillary dental arch:

The marginal gingiva adjacent to the teeth 1.1, 1.2 and 1.3 exhibited a cyanotic change of color and was affected by a slight apical recession (about 1-2 mms apical migration). This area was also rich in thick deposits of dental calculus, covered with soft bacterial biofilm, contributing directly to the pathological changes of the marginal gingiva. The gingival inflammatory sings were less obvious in the second quadrant, the gingival tissues expressing normal color and a good stippling texture. However, the maxillary incisors showed considerable inter-dental spaces, with no contact point. This led to a flattening of the inter-dental papillae's shape. When asked about the position of the maxillary incisors and the space between them, the patient declared that they erupted in this manner, but that in recent years, the central diastema seemed to get wider. The prosthodontic appliance in the second quadrant exhibited stage three distal mobility and was removed after the initial examination and the 2.4 abutment extracted. The dental bridge in the first quadrant was slightly mobile (stage one), but still showed a good marginal adaptation.

ii) On the mandibular dental arch:

The marginal gingiva exhibited a generalized reddish color, while the attached gingiva was more close to the normal, coral pink, color. The gingival papillae and the marginal gingiva adjacent to the frontal mandibular group expressed an increased volume, with a McCall gingival rolled festoon showing on the 3.3 and 4.3 teeth. Despite this increased volume, moderate gingival retractions could also be observed on several teeth: 3.1, 4.1, 4.2 and 4.3. The gingival margin was also apically displaced on the bilateral posterior teeth, but showed less obvious inflammatory signs. Thick bacterial biofilm and calculus deposits were observed within the gingival sulcus of the frontal mandibular teeth and on their inter-proximal and lingual aspects. An important clinical sign, which increased awareness were the white, cream-like, deposits found in the gingival sulcus of 3.1 and 4.1. All teeth on the mandibular arch showed significant sings of occlusal attrition, in some of them (3.3, 3.4, 3.5, 3.6, 4.3, 4.4, 4.5 and 4.7) the dentine tissue being exposed, an obvious cause of the patient's declared

increased tooth sensitivity. On the 4.6 tooth, a large, fractured coronal obturation could be observed, which allowed the formation of a secondary carious lesion on the occlusal surface of this tooth.

iii) Occlusion

From the first glance at the patient's intra-oral status it was clear that this was a bruxer patient. Multiple signs contributed to this conclusion: the severe generalized attrition of the occlusal and incisal margins of teeth leading to characteristic "polished-like" appearance of dental surfaces, the receding gingival margin on the lateral areas of the mandibular arch and the fractured incisal margins of 2.1 and 3.3.

iv) Periodontal probing

The periodontal probing assessment revealed that multiple deep periodontal pockets (≥ 7mms) were present on the vestibular and palatal aspect of the maxillary teeth which served as abutments for the fixed prosthetics. The deepest periodontal pocket was recorded for the 2.4 tooth, being as deep as 11mms on the disto-palatal probing site. The maxillary central incisors also exhibited periodontal pockets of up to 7 mms depth. For the mandibular teeth, the periodontal probing status was more favorable, as isolated sites of 5-6 mms probing depth were found around the central mandibular incisors and the bilateral first molars. However, despite the more reduced probing depths, the mandibular probing sites expressed significantly more bleeding on probing than the maxillary ones (55% mandibular bleeding on probing index compared to 26% maxillary). Overall, the bleeding on probing index was reduced in comparison to the probing depths, probably due to decreased gingival blood flow induced by the heavy smoking. Plaque index was determined at 68%.

v) Radiological evaluation

The panoramic evaluation of the patient reflected significant generalized horizontal alveolar bone resorption. Nevertheless, the maxilla was significantly more affected than the mandible, with almost 70% resorption around the abutment teeth, 60% bone loss at the central incisors, as compared 30% in the lower ones. The situation of the 2.4 tooth is worth mentioning, as it is shown by the radiograph, its vertical bone resorption extended all the way to its apical region, probably leading to the formation of a mixed endodontic-periodontal lesion. The severe stage three mobility of this tooth required the removal of the bridge and the abutment's extraction. In other sites, particularly between the mandibular incisors and bicuspids, the loss of alveolar bone lamina-dura can be observed, signaling a loss of minerals that affects the bone in these areas.

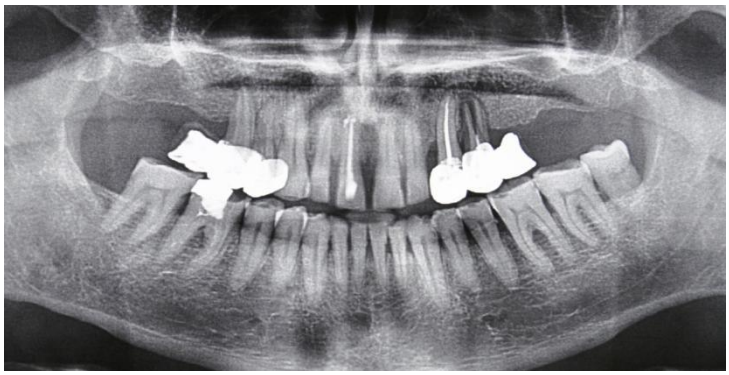

Figure 2 – Panoramic radiographic evaluation of the patient (initial)

vi) Diagnosis

According to the 2017 Classification of Periodontal Diseases, [11] issued by the European Federation of Periodontology, the patient was diagnosed with Generalized Periodontitis Stage 3, Grade B. This diagnosis reflects the periodontal status of the patient, consisting of generalized severe periodontitis with a moderate rate of progression. Adding to the unfavorable periodontal status are the predisposing risk factors consisting of systemic ones, including age, systemic conditions and their respective medication and smoking, as well as local ones, mainly the significant occlusal disorder (bruxism).

III. Discussion

Since most local risk factors are modifiable and can be reduced or eliminated, the majority of the systemic ones are not easily approachable and often require a multi-disciplinary medical team. This is justified by the genetic determinism of certain systemic risk factors or by the general implications on the homeostasis that they trigger. [12]

One of the first systemic risk factors that the patient was confronted-with is his actual age. It is considered that after 35-40 years of age, patient are more predisposed to chronic periodontal conditions, because, given their prolonged evolution, these diseases become manifest. [13] In addition, in elderly patients the collagen synthesis decreases, so the periodontal tissues, which are rich in collagen, are gradually deprived of

this element. Thus, the periodontal structures become physically weak, unable to regenerate after bacterial challenging events. In a similar manner acts the down-regulation of the host immune response in the elderly patients, which further enhances the bacterial damage on the periodontal tissues. [14] Taking these principles into consideration, it is clear that our patient was at high risk of periodontal disease onset and of gradual degeneration of the periodontal tissues.

As patients get older, the probability of developing significant systemic diseases increases, the most frequent ones being diabetes mellitus and cardiovascular conditions. Unsurprisingly, these major systemic diseases have been the first to be studied in connection with periodontal disease and, consequently, have been proven to express mutual bi-directional influencing relationships. [15] Elevated blood pressure is one of most common forms of cardiovascular conditions and is frequently treated by administration of Calcium-channel-blockers. [16] This is the case of our patient, as he had been under treatment with Nifedipine for about 10 months. Such drugs are known to cause gingival overgrowth, which is directly proportionate with the quantity and duration of the administered medication. [16] In the early stages of the gingival changes, the gingiva is elastic, but gradually softens, as it was observed during the examination. Another observed significant clinical were the white, soft deposits found within the gingival sulci, which are characteristic for patients suffering from diabetes mellitus. [17] Given the patients age, he was referred to a general practitioner for complete diabetic screening. Consequently, the patient was diagnosed with incipient type II diabetes and started the indicated treatment. It is known that in diabetic patients, the risk of PD onset is at least three times higher than in non-diabetic individuals. [17]

Smoking is another systemic risk factor which was well-represented in our patient, as he used to smoke at least 20 cigarettes per day. As nicotine forms deposits inside blood vessels, their lumen reduces in diameter, allowing fewer and fewer blood to reach the gingival tissues. [18] This deprives the tissues of valuable oxygen, reducing the oxidative potential and healing capabilities. In addition, the host immune response is also down-regulated by the impaired neutrophils' chemotaxis and the disrupted lymphocyte's behavior. [18] As our patient enjoyed a fat-rich diet, it can be argued that a nutritional misbalance may have also systemically predisposed the onset of periodontal disease. It is known that vitamin A, B, C and D deficiencies can decrease the gingival tissue's defensive capabilities and their resistance to the bacterial challenge, particularly in patients with diabetes mellitus. [19, 20] Moreover, patients with a fat and carbohydrate-rich diet are prone to the development of cellular insulin resistance, which has a significant negative impact on the general homeostasis and host immune response. [21]

IV. Conclusion

In conclusion, we believe that this case accurately reflects the pathogenic mechanisms and clinical manifestations that systemic risk factors can inflict into a patient, particularly an elderly one, in terms of periodontal disease predisposition, onset and evolution. A significant message is that dental practitioners/periodontists should pay close attention when examining such patients, as they can help discover and, eventually, refer for diagnosis systemic underlying conditions.

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All authors had equal contribution to that of the first author.

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