International Journal of Biomedical Investigation

Available Online: http://ijbi.edwiserinternational.com

Periodontal Disease

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Editorial

To establish the treatment or prevention of an illness will always be necessary the eradication of all etiological factors, and/or increase host local resistance and/or improve the overall resistance of the host against all etiologic factors, and/or promote a control of etiological factors at levels below their potential for aggression capable of being inactivate by the host's defensive system to induce health for a long period. To equate a treatment plan for a disease, it is extremely necessary to analyze how the etiological agents act to initiate the disease. The etiology of periodontal disease is associated with the interrelationship between the bacteria and the predisposing risk factors of local and/or general order, which are responsible for the disease status [1]. Bacteria always were considered as indispensable etiologic factors which are necessary to develop a periodontal disease; however, it is improbable that only bacteria are sufficient to initiate a periodontal disease. Then the diagnostic of the all etiological mediators of the periodontal disease is critical and difficult, due to the necessity of the association among a susceptible host, bacteria and the various etiological predisposing risk factors which can be inherent and specific for each individual. Despite this, the preponderance of all forms of periodontal diseases [2] are considered as microorganisms dependent and most of the procedures that focus on the treatment of periodontal disease are based on bacterial control. Bacteria and their harmful products induce a defensive inflammatory reaction that inactivates and destroys the bacteria, but release products such as enzymes and toxic metabolites of the bacteria and cells involved in the inflammatory reaction that are destroyed during the defensive process, which induce periodontal tissue damage by lytic activities [3].

Consequently, the main features of the periodontal disease are: gingival inflammation, junctional epithelium ulceration, damage of connective tissue and alveolar bone, producing apical migration of the junctional epithelium, which determines the formation of a periodontal pocket around the teeth. Then periodontal disease is a defensive mechanism which avoids penetration of bacteria into the periodontal tissues, to prevent infections and septicemia. Nevertheless, as a secondary effect this protective mechanism induces the development of periodontal pockets, the main pathognomonic indication of a periodontal disease: ulceration and increase in the depth of gingival sulcus, thus, generating a favourable anaerobic environment to be infected, as a result of the repeated contamination by the numerous species or the combination of the species as exogenous anaerobic and facultative bacteria which are considered as periodontopathogenic bacteria. These putative periodontal pathogens and their products as endotoxins could induce considerable pathological modifications, fundamentally in root surface which are exposed to the contaminated periodontal pocket.

Contaminated periodontal pocket similarly may be a source of infection because of the contact of the bacteria with the vascularized connective tissue through the ulcerated epithelium of the periodontal pocket. Contaminated periodontal pocket as infectious focus could be associated to the several systemic disorders, possibly managed by anachoresis, a process associated with dissemination of the microorganisms or/and toxics products into blood stream, assisting or producing infection in the various vital organs. Although all destruction of the periodontal tissues during the evolution of the periodontal disease has analogous features, the developmental origin triggered by the association between the bacteria and the various predisposing risk factors, may be different, and intrinsic to everyone, conditioning on the host's susceptibility, which does not constantly present an equal vulnerability to the innumerable local and/or general predisposing risk factors. The periodontal disease may induce generalized or localized destruction depending on the association of the opportunist bacteria and numerous intrinsic predisposing risk factors. General predisposing risk factors may be systemic factors which interfere in

the defensive inflammatory process against bacteria acting in the periodontal tissues around all teeth.

Local predisposing risk factor may be associated in bacterial retention increasing its quality and quantity and/or in the promotion of a mechanical vulnerability in the periodontal tissues favouring the action of bacteria, the predisposing risk factor may statistically intensify the incidence of a disease, but it does not promote the disease. Periodontal diseases possess multifactorial etiology and all of them present successive, rapid, destructive, acute cycles, always followed by long, quiescent, reparative, chronic cycles. Indication of the reparative phase of periodontal disease is possible being verified in histological analyses of untreated periodontal pockets as cementum and fiber bundle of gingival connective tissue attachment, separating the bottom of the periodontal pocket from the destructed alveolar bone which habitually presents a repaired cortical bone at a variety of levels isolating the cancellous bone from contaminated periodontal pocket environment. Consequently, after each consecutive destructive acute phase of periodontal disease, always occur an expansion in the quality and quantity of bacteria and the etiological predisposing risk factors.

Despite this, the transitory, short, destructive but protective acute phase always ceases being always followed by a long chronic quiescent reparative phase. This fact reveals that, during the periodontal disease progression, bacteria and etiological predisposing risk factors improve their qualities and quantities but are not capable to sustain the destructive phase activity during all the evolutionary cycle of periodontal disease. Possibly, the periodontal disease arises and develops when at a given time, a specific transitory fragility, inherent for each individual, helps involved etiological agents in initiating the destructive acute phase starting the progression of the periodontal disease. Because of this relationship between individuals and the numerous possible etiological factors of periodontal disease that may vary for each of them, it is very difficult to establish which etiological factors are acting to develop the disease in a particular individual and in a determined moment. If the etiological factors of periodontal disease were the same for all individuals and if the disease did not progress through destructive periodic acute outbreaks interspersed with chronic reparative outbreaks it would be simpler to establish an effective treatment plan.

The unique etiological factors that will always be present to cause periodontal disease for all individuals are bacteria. Perhaps for this reason periodontal disease treatment is based on the control of bacteria and their products. However, is this concept of treatment able to treat periodontal disease effectively? And the other multifactors of periodontal disease that in association with bacteria trigger the disease should not be focused? Periodontal disease is still a challenge to be deciphered.

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Article info

Received 09 February 2018
Revised 20 February 2018
Published 22 February 2018

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