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Periodontal Disease: The Diagnose

Jose Ricardo Kina[1]* and Eunice Fumico Umeda Kina[2]

[1]*Retired Associate Professor of Periodontics, São Paulo State University Julio de Mesquita Filho Araçatuba, São Paulo, Brazil*

[2]*Private Practice, Sao Paulo State University, Brazi[1]*

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***Corresponding author:** Jose Ricardo Kina, Department of Periodontics, São Paulo State University Julio de Mesquita Filho Araçatuba, São Paulo, Brazil

Short Communication

Periodontal disease one of the most common and prevalent diseases of the oral cavity has been a challenge in its etiology, diagnosis and treatment. The etiology of periodontal disease is multifactorial, and it is considered that biofilm plays an essential role in its development and evolution [1]. Due periodontal disease development be dependent of the bacteria and various predisposing risk factors that are nonspecific for each individual and also in the same individual are unspecific since at a determined moment may trigger periodontal disease and at another moment may not have the same pathological capacity to trigger periodontal disease makes periodontal disease a difficult challenge to be solved [2]. Manly when the diagnose of the etiologic factors that initiate periodontal disease needs to be performed since to treat any disease is always extremely necessary to eliminate or to establish a control in all etiologic factors which are responsible for disease activity [2,3]. Then if the main factors that will be always necessary to initiate periodontal disease are bacteria is logic to establish a control in bacteria development [1]. However, establishing a control in bacteria development may be enough to control periodontal disease? This approach is always unpredictable because only bacteria are not sufficient to initiate periodontitis [1]. The predisposing risk factors always will be necessary to help bacteria developing periodontal disease [2,3]. If to treat a disease is necessary to know all etiologic factors and their inherent pathological mechanisms which may be associated with disease establishment and progression [4-6], are the methods applied up to know to establish a periodontal disease diagnostic reliable? The methods applied to diagnose periodontal disease are based in clinical and radiographic parameters which do not seem sufficient to diagnosis periodontal disease mainly its acute destructive phase always interleaved by a chronic quiescent phase. The clinical diagnosis methods are based in probing deep, probing deep bleeding, attachment levels measures, and tooth mobility. The accuracy of these clinical diagnostic methods may show false-positive results, inducing evaluations and interpretations that may compromise the diagnosis, bringing therapeutic implications such as therapeutic planning and therapeutic follow-up. The periodontal diagnostic procedures should be useful to inform what type of periodontal disease is present, where it is located, the severity of the periodontal disease, and if it is active or inactive [5,7].

Unfortunately, up to now the current clinical diagnosis methods applied only may inform that in a determinate moment in the past the individual developed periodontal disease. For example, probing deep test is the measure from the gingival margin until the bottom of the normal sulcus with the probe passing through the fragile junctional epithelium reaching the gingival connective tissue fibers insertion. The result of this measurement is around 2mm, meaning, 0.5mm of the depth of the gingival sulcus and 1.5mm of the junctional epithelium extension, which is fragile and allows the passage of the probe through it. When the individual present untreated periodontal disease the probing deep tests result in the measure from the gingival margin until the bottom of the periodontal pocket with the probe passing through ulcerated junctional epithelium that migrate to apical, until reaching repaired gingival connective tissue fibers insertion above destructed alveolar bone. The result of this measure is the deep of the periodontal pocket. When the individual has gingival recession, a peculiar periodontal disease that induct loss of the periodontal bone, periodontal ligament, migration of the gingival margin to apical without development of the periodontal pocket, the probing deep test result almost always in the measure from the gingival margin until the bottom of an apparent normal sulcus with the probe passing through the junctional epithelium reaching the repaired gingival connective tissue fibers insertion. Then the probing deep test in this situation can generate an unreal measure that may be understood as health periodontal tissue but losing periodontal tissues always should be interpreted that the individual developed periodontal disease at some point in the past.

When the individual undergoes periodontal disease treatment, the periodontal pocket heals through a long junctional epithelium. The probing deep test in this case is the measure from the gingival margin until the bottom of the normal sulcus with the probe passing through the repaired long junctional epithelium reaching the gingival connective tissue fibers insertion repaired above destructed periodontal alveolar bone. The result of this measure comprise around 0,5mm of the repaired gingival sulcus added to the measure of a long junctional epithelium that for being long, produce an altered measure entitled false periodontal pocket. Then the probing deep test in this situation can generate an unreal measure of the depth of

the repaired gingival sulcus that returns to 2mm deep after healing but due to the fragility of the repaired long junctional epithelium may create a false positive measure which may be interpreted as indicative of the presence of periodontal pocket. The probing deep bleeding is a diagnostic test that is used by some clinicians as criterion to diagnose gingival inflammation, to monitor periodontal disease activity but the test is not trustful since the probing deep blinding depends of the probe type and the exerted force of the operator to perform the probing depth. The tooth mobility may be associated to the various diseases and is not always associated with periodontal disease mainly if the tooth does not present periodontal pocket development. Maybe the most trustful clinical diagnostic test for periodontal disease is periodontal attachment levels measures which have as fixed parameter, the cemento-enamel junction. The periodontal attachment levels measures comprise the extension from the cemento-enamel junction until the bottom of the gingival sulcus or of the periodontal pocket or of the false periodontal pocket reaching the gingival connective tissue fibers insertion situated immediately above alveolar periodontal bone. As periodontal attachment test is based on cemento-enamel junction localization a fixed parameter, this test permits reproducibility and the fixed reference point provides the possibility to compare the periodontal disease evolution. However, the main interpretation of this test is that at a determined time point the individual developed periodontal disease which destructed periodontal ligament [5]. In some situation the main difficulty to apply this test is to define accurately the cemento-enamel junction localization. Although the clinical usefulness of these tests to diagnose periodontal disease is questionable, its clinical applications are still routinely applied to attempt to diagnose periodontal disease.

Several diagnostic tests for periodontal disease have been proposed as: genetic tests for susceptibility to periodontitis, host response tests for diagnosing periodontal disease, microbiological tests to diagnose periodontal disease, blood tests to diagnose periodontal disease, host response markers to revel periodontal disease, gingival crevicular fluid assays, markers of cell death, markers of the acute inflammatory response, serum diagnostic for periodontal disease, salivary diagnostic for periodontal disease [7]. All diagnostic tests have their value but a specific test to predict periodontal disease remains unfeasible and is still a challenge. The periodontal disease etiology is multifactorial were bacteria play an essential role but without association with predisposing risk factors, bacteria alone probably are unable to develop periodontal disease [1-6]. In addition periodontal disease etiologic factors are unspecific for each individual being able to trigger destruction in an individual but not in other individuals and can sometimes cause destruction in one place but not in other places of the same individual. The periodontal disease evolution occurs through of the rapid acute destructive period always interleaved by a long chronic quiescent period even without treatment and without elimination or control of the etiological factors that trigger its acute destructive phase [2,5]. Although each acute destructive period induces an increase in the etiological factors that trigger the activity of the disease, periodontal disease is always able to enter in the quiescent chronic period [5]. Moreover, periodontal disease is a singular defensive mechanism that as sequel may induct periodontal tissue destruction [2,4,5]. Understand the etiology of periodontal disease and the mechanisms of how these etiological factors act to promote the destruction of periodontal tissues, which exponentially increases the quality and quantity of its etiological factors, but still fails to maintain active the destructive phase of periodontal disease should be the key to establish a diagnostic test for periodontal disease [5,7]. However, it would be possible to establish a diagnostic test for a disease that in reality should not be considered a disease but rather a complex defensive reaction that as a sequel induces periodontal tissues destruction almost always permanently due to complexity of the periodontal tissues around the teeth mainly the junctional epithelium [8,9].

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