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Is localized aggressive periodontitis a distinct entity? Redefinition of a unique periodontal disease

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ABSTRACT

Aggressive periodontitis is a questionable disease with limited evidence supporting its incidence and mechanism. Till now, the genetic hypothesis is the most acceptable theory that supports its presence. According to literature, several unproven theories were conducted that could not provide definite explanation to its behavior. The available evidence only supported the ability to restore the destructed tissues rather than controlling the disease. The aim of the present research is trying to postulate a new hypothesis that may clarify the nature of the localized form of aggressive periodontitis.

INTRODUCTION

Aggressive periodontitis is a specific form of periodontitis manifested by massive destruction of the supporting structures. Based on the number of the involved teeth, the disease could be categorized either as localized or generalized (1). Clinical attachment loss, mobility, teeth loss, periodontal abscesses, external root resorption (loss of cementum) (2, 3) are common manifestations for both forms with variable degrees.

In the generalized aggressive periodontitis, greater percentage of clinical attachment loss commonly involves nearly the entire dentition (1). While in the localized form, significant attachment loss could be clearly identified by its relation to two permanent teeth. Permanent maxillary incisors, mandibular incisors and first molars are the most commonly affected teeth (4). Bilateral mirror image of arch-shaped resorption of alveolar bone can be detected and considered as a characteristic phenomenon of the localized form (2).

In contrast to generalized aggressive periodontitis, the localized form exhibits a steady nature (5). According to the literature, it is generally identified throughout childhood and adolescence periods in systemically healthy individuals (6, 7).

Two hypotheses have tried to explain the behavior of the localized form. The first theory suggested that aging may carry the localized form to proceed into the generalized one by spreading to the neighboring teeth (8, 9). It was found that the rate of bone destruction exceeds chronic periodontitis by 3 or 4 times. While the second hypothesis described it as a self-limiting disease (2).

According to literature, several studies tried to propose different theories to explain the mechanism of the localized form. In 1923, localized aggressive periodontitis was firstly described by Gottlieb as an atrophy of the alveolar ridge (10). Then, in 1928, it was suggested that early periodontitis was attributed to the inhibition of the continuous cementum formation (deep cementopathia or disease of eruption) which is considered as a foreign body that provokes the cell-mediated immunity (2, 3). On the other hand, in 1938 till 1947, the disease was portrayed as paradontosis or periodontosis (non-inflammatory degenerative disease). In 1942, Orban and Weinmann (11) proposed that the disease begins at the alveolar bone causing vascular resorption (spider monkey or lacunae resoprtion).

Afterwards, Baer (1971) (12) introduced the term

periodontitis to describe the rapid destruction of alveolar bone of permanent teeth without association with a local factor. The term localized juvenile (pre-pubertal) periodontitis was proposed by Lehner et al. (1974) (13) to describe the cell-mediated immunodeficiency-associated periodontitis which usually begins at the age of 4 years (during or after the age of deciduous teeth eruption) and extends to the pubertal period. (3). It mainly affects the permanent dentition that remains for longer period till being lost by the age of 15 years (2). In 1999, the American Academy of Periodontology changed the term into localized aggressive periodontitis. This term is as yet substantial till now (3).

The bacteria-based hypothesis (Nibali et al., 2009, 2015) was the main theory that clarified the mechanism of disease progression based on the presence of *Aggregatibacter actinomycetemcomitans* (3). Up till now, the latter theory is the most acceptable one.

PROPOSAL OF A NEW HYPOTHESIS

The proposed hypothesis suggests that localized aggressive periodontitis is one of the atopic diseases that may develop around the age of 3-5 years and reaches its peak of activity with the age of 8-12 years (puberty stage) followed by stoppage. Disease regression is attributed to maturation of the immune system. Unfortunately, the active phase of the disease is usually accompanied by destruction of the periodontal apparatus that relates to either deciduous and/ or permanent dentition. The development of oral soft tissue structures with limited bony support surrounding the affected teeth may cause malocclusion (drifting, over-eruption, proclination, etc.). Based on the protocol of the atopic diseases, the disease is self-limiting and usually reduces its rate in adolescence. The future manifestations could be regarded to the presence of interproximal retentive regions and bacterial flare up which usually occurs.

In order to examine the disturbed response of the immune system in young children, the proposed checklist is recommended to be fulfilled. Furthermore, performing RAST test (skin prick test) which is specific to the IgE antibodies is recommended before starting treatment. It is a safe and easy test to detect the hyper response of the immune system.

A checklist was developed (Table 1) to help clinical assessment, on the basis of the proposed hypothesis. If the total score for the checklist exceeds 7 points, the patient is categorized as at high risk. The parents should be advised with further specified investigations and meticulous medical follow up with the physician to control the systemic condition. Meticulous follow up at short intervals for periodontitis is also recommended in case of gingivitis, for at least one year to ensure controlling the condition and stopping the disease progression.

Checklist		Yes	No	Unclear
1	Is the patient's age between 3-10 years?			
2	If the patient age is 3-10 years, did he/ she develop early shedding?			
3	Did the patient suffer from gingival inflammation or gingival bleeding?			
4	Did the patient manifest early signs of food impaction or sensitivity?			
5	Did the patient develop teeth mobility?			
6	Did the patient adhere to the daily oral hygiene instructions?			
7	Did the patient suffer from allergic manifestations to drugs, food, clothes, etc.?			
8	Did the patient suffer from recurrent common cold, fever, bronchial asthma, dermatitis, chicken pox or GIT problems?			
9	If lesions appeared, were the oral or dermal manifestations symmetrically distributed (mirror image)?			
10	Did you perform RAST test or skin prick test for hypersensitivity?			
11	Did the patient present abnormal CBC records (neutropenia, lymphocytosis, anemia, eosinophilia, etc.?			
12	Is there a family history of disturbed immune response of any form?			

TABLE Evidence collection based on the proposed hypothesis.

Atopic diseases (allergic predisposition) are group of diseases that are based on improper immune response in the form of over production of IgE that is directed against unharmful immunoglobulin. The basic mechanism of action depends on the hyper-response against allergens or micro-organisms within the early period of life which may affect the normal progression from T helper cells type 2 (TH-2) to T helper cells type 1 (TH-1) causing persistence of TH-2. The genetic background has an important role in triggering the environmental factors (14-16). They are characterized by a wide range of different signs and symptoms; allergic rhinitis, eczema, asthma, food allergy, etc. Atopic diseases usually differ in severity and causative agents. The prophylactic strategy against atopic disease is based mainly on providing a hygienic environment to children (14-16).

A child with atopy means that he/she is over sensitive to common allergens which stimulate the immature immune system to produce greater amounts of IgE. Not all atopic patients manifest an atopic disease. Atopic diseases usually appear at early life time (at birth till 5 years) and recess spontaneously (reversible in nature) at the age of 8-13 years. Remission of the symptoms is intimately related to age (17, 18).

The allergic predisposition causes allergic inflammation and affects different organs such as lungs, skin, bone,

teeth, connective tissue, nervous system, brain and heart with different resolution time. In food allergy, atopy resolves at the age of 5 years while the atopic asthma and rhinitis resolute by the age of 13-14 years and may extend to 22 years (19).

Based on our proposed hypothesis and the nature of atopic diseases, we truly believe that localized aggressive periodontitis is caused by hyper responsive immune system either accompanied with healthy or diseased subgingival environment. In consistence, deficient neutrophil chemotaxis was also observed in localized aggressive periodontitis (20). It was also believed that extraction of primary teeth would stop the disease spreading to permanent teeth (7). A similar expression of Toll like receptors (TLRs) type 2, 9, 14 with both atopic diseases and different forms of periodontitis was found. Furthermore, single gene polymorphism, higher serum IgE and super-antigen specific IgE were associated with atopic diseases (20- 22).

In the past century, the American Academy reported higher incidence of localized aggressive periodontitis among the African Americans. Different age ranges were reported between countries but it never exceeds the 30 years of age. The highest disease activity was reported at an early age manifested by clinical attachment loss (≥ 4 mm) and obvious alveolar bone loss (3).

As a self-limiting disease, it was found that the progression rate of localized aggressive periodontitis usually slows down or stop with age (4). Such a stage is called a burn out stage. No radiographic progression on the diseased regions is reported over years even in the untreated patients (12, 23), nor clinical signs of inflammation r elated t o t he p eriodontal t issues (12). Such phenomenon is opposite to the traditional behavior of periodontal diseases which include periods of remission and exacerbation (24).

CONCLUSION AND RECOMMENDATIONS

According to the literature-based evidence, treating localized aggressive periodontitis can be shifted to simulate atopic self-limiting diseases. Furthermore, changing the term from localized aggressive periodontitis into localized atopic periodontitis is recommended.

Prospective long term surveys are recommended to detect the detailed disease behavior. Controlling the general hygiene and early diagnosis at a young age is extremely important to protect against such diseases and to stop their progression.

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