ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 4, Issue – 3, May - 2021, Page No. : 82 - 92

Aggressive Periodontitis: A Narrative Review

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Citation of this Article: Dr. Shilpa Kaundal, Dr. Rajan Gupta, Dr. Parveen Dahiya, Dr. Mukesh Kumar, Dr. Sunita Melwani, "Aggressive Periodontitis: A Narrative Review", IJDSIR- May - 2021, Vol. – 4, Issue - 3, P. No. 82 – 92.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

Aggressive periodontitis which encompasses a number of clinical entities probably results from tissues destructive mechanisms which are common to most forms of periodontal diseases. The unique attributes of the disease process are due to the virulence of the pathogens and the host susceptibility which may be due to the heritable or acquired susceptibility factors, which permit expression of periodontitis at a relatively younger age. The purpose of this review is to highlight the current etiological and therapeutic concepts of aggressive periodontitis which is rapidly progressing and aggressive in nature.

Keywords: Periodontitis, Aggressive Periodontitis, Periodontal diseases, Aggregatibacter actinomycetemcomitans, periodontal destruction.

Introduction

Periodontal disease is an endogenous microbial disease that damages the dental structure and the periodontium. The disease derives from the cellular and humoral response of the host, altering the homeostasis of the periodontal tissues and causing inflammation and destruction by means of bacterial enzymes and virulence factors.

Periodontal disease is one of the major dental diseases that affect human populations worldwide at high prevalence

rates. [1] The World Health Organization (WHO) reported that 10–15% of the world populations suffer from severe periodontitis. [2] According to a report the prevalence, severity, and extent of periodontitis was given in the US adult population using combined data from the 2009–2010 and 2011–2012 cycles of the National Health and Nutrition Examination Survey (NHANES). For the first time in NHANES history, sufficient numbers of Non-Hispanic Asians were sampled in 2011–2012 to provide reliable estimates of their periodontitis prevalence. Periodontitis prevalence was positively associated with increasing age and was higher among males. Periodontitis prevalence was highest in Hispanics (63.5%) and Non-Hispanic blacks (59.1%), followed by Non-Hispanic Asian Americans (50.0%), and lowest in Non-Hispanic whites (40.8%). There are many forms of periodontal diseases. The most common forms include: Gingivitis, Aggressive Periodontitis, Chronic Periodontitis, Periodontitis as a manifestation of systemic diseases, and Necrotizing Periodontal disease. The evidence suggests that aggressive periodontitis is influenced by microbial, genetic, and host factors.[3]

Aggressive periodontitis, first described in 1923 as "diffuse atrophy of the alveolar bone" has undergone a series of terminology changes over the years, and has been named as "Aggressive Periodontitis" in 1999.[4]

Jan Lindhe defined Aggressive Periodontitis as "a group of rare, severe, rapidly progressing forms of periodontitis characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families.

Aggressive periodontitis (AgP) is a disease which is characterized by rapid loss of periodontal tissues affecting systemically healthy individuals during adolescence and adulthood, and forms a group of periodontal diseases.[5] Aggressive periodontitis can be distinguished from chronic periodontitis by age of onset, rapid rate of progression, nature and composition of the subgingival microflora, alterations in host's immune response and a familial aggregation of diseased individuals.

A person's lifestyle also plays a fundamental role in the appearance or inhibition of aggressive periodontitis. With respect to the appearance of aggressive periodontitis, local and systemic factors are present, such as age, gender, stress, and socioeconomic level. Disease severity increases with age, there is a greater prevalence in women linked with hormonal changes at the pubertal stage, periods of stress diminish the immune response of the organism, and the disease has been associated with low socioeconomic level, which is characterized by deficient habits of hygiene and diet.[6]

Aggressive periodontal disease is an oral health mystery. Our current understanding of this disease is that specific bacteria invade the oral cavity and the host reacts with an inflammatory response leading to mass destruction of the alveolar bone. Aggressive periodontal disease is typically observed in a population under the age of 30 and occurs so rapidly that it is difficult to treat. Aggressive periodontal disease has a tremendous effect on patient's overall quality of life and needs to be investigated more extensively in order to develop methods for earlier definitive diagnosis and effective treatments. One of the mysteries of aggressive periodontal disease is the relatively nominal amount of plaque present on the tooth surface in relation to the large amount of bone loss. There seems to be a hidden factor that lies between the response by the patient's immune system and the bacterial threat that is present. A better mechanistic understanding of this disease is essential to provide meaningful care and better outcomes for patients.

The aim of this review is to highlight the current etiological and therapeutic concepts of aggressive periodontitis which is rapidly progressing and aggressive

in nature. It leads to the destruction of periodontal tissues and loss of teeth.

Historical Background

Based on histologic observations, **Black in the year 1886**, used the terms "phagedenic pericementitis and chronic suppurative pericementitis" to describe patients who suffered from a rapid destruction of alveolar bone.[7]

Gottlieb in the year 1923 described an unusual form of periodontal disease that involved some or all of the permanent incisors and first molars of young individuals. Based on histological observations on extracted teeth from affected sites, he believed that the disease was "due to defective deposition of cementum or cementopathia". Gottlieb in the year 1928 applied the principles of classical pathology, which stated that "all human nonneoplastic diseases could be classified as either inflammatory or non-inflammatory and used the term diffuse atrophy of the alveolar bone" to describe a condition in which adolescent patients did not exhibit the intense gingival inflammation ordinarily seen in other adult patients with periodontitis, he believed that the disease was a noninflammatory or degenerative condition.[8]

In **1938 Wannenmacher** described incisor–first molar involvement and called the disease "parodontitis marginalis progressive".[9] Several explanations evolved for the etiology and pathogenesis of this type of disease. Many authors considered this to be a degenerative, non-inflammatory disease process and therefore gave it the name "periodontosis".[10] Other investigators denied the existence of a degenerative type of periodontal disease and attributed the changes observed to trauma from occlusion.[11]

In the year **1942**, **Orban and Weinmann** introduced the term periodontosis to describe the periodontal destruction in young individuals.[12]

A **1950 report by the American Academy of Periodontology** defined "**periodontosis**" as a "degenerative non-inflammatory destruction of the periodontium originating in one or more of the periodontal structures, characterized by migration and loosening of the teeth in the presence or absence of secondary epithelial proliferation and pocket formation or secondary gingival disease.

The terms **"juvenile periodontitis"** and **"early-onset periodontitis"** were introduced in 1969 and 1989, respectively, and were widely used during the last three decades of the 20th century.[13] The latter terms were readily adopted because they portray the early-onset development of the disease and its occurrence in younger age groups. Other terms were proposed to describe the disease, such as precocious advanced alveolar atrophy and precocious periodontitis, but were not widely adopted.

In the **1999 Classification Workshop of the American Academy of Periodontology**, a consensus report adopted the term **"aggressive periodontitis"** as a new name for this unique disease classification, replacing the term **"early-onset periodontitis".**[14]

However, in the 2017 classification system, the distinction between chronic and aggressive periodontitis has been removed on the basis that there was little evidence from biological studies that chronic and aggressive periodontitis were separate entities, rather than variations along a spectrum of the same disease process. The exception was classical localised juvenile (aggressive) periodontitis, where a clearly defined clinical phenotype exists, however, there was unease about including this as a distinct and separate entity within the classication system.

Classification

The overall classification system of 1999 aimed to differentiate the more common forms of periodontitis i.e. chronic and aggressive periodontitis. Moreover, this

classification system emphasis on disease severity and has meticulously tried to segregate each disease under a different category.

The 2017 world workshop classification system for periodontal and peri-implant diseases and conditions was developed to derive knowledge from both biological and clinical research. Its aim was to create a consensus knowledge base to enable and promote new classifcation globally. In 2017 classification "Aggressive and Chronic Periodontitis" terms were removed and were put under the term "Periodontitis".

Table 1: Diagnostic Criteria to Distinguish Chronic and Aggressive Periodontitis[15]

Criterion	Aggressive Periodontitis	Chronic Periodontitis
Rate of progression	Rapid	Slow; rapid episodes possible
Familiar aggregation	Typical	Can be present when families share imperfect oral hygiene habits
Presence of etiologic factors (plaque, calculus, overhanging restorations, etc.)	Often minimal	Often commensurate with observed periodontal destruction
Age	Often in young patients	Often in older patients (>55 years) but can be found in all age groups
Clinical inflammation signs	Sometimes lacking (especially in localized aggressive periodontitis)	Commensurate with etiologic factors

Epidemiology

The prevalence of this disease in a given population may be determined by where the population lives and by the composition of the population pertaining to the type and proportions of race and ethnic groups. Most studies show comparable disease prevalence in male subjects and female subjects. Furthermore, among children and young adults the detection frequency of subjects with disease is higher in the older age groups than in the younger age groups. For these reasons epidemiologic studies of aggressive periodontitis may not yield accurate estimates of the disease if these studies use study samples that are not representative of their target populations, and the more diverse a population is the more biased are the disease estimates if a convenience sample is used.

Different studies conclude that, among different geographic locations and race groups, aggressive periodontitis is most prevalent in African populations and their descendants. The disease prevalence in African populations is between 1% and 5%. On the other hand, in Caucasians residing in north and mid-Europe the prevalence is 0.1%, and in south European populations the prevalence is ~0.5%. In North America, the disease affects approximately 0.1–0.2% of Caucasians, 0.5–1.0% of Hispanics and 2.6% of Black people. The countries in South America comprise a diverse racial composition, with some countries, such as Chile and Argentina, comprising mainly a Caucasian population, whereas other countries, such as Brazil, comprise a mixed-race population. Nonetheless, only a few studies with representative samples have been conducted in Asian countries. The disease prevalence in Asian populations is between 0.2% and 1.0%.[16]

These finding shows that aggressive periodontitis is a significant health problem in certain populations. There is a lack of information on the epidemiology of this disease in many parts of the world. Epidemiological studies of aggressive periodontitis in high-risk populations are important and could provide vital data on the determinants of this disease, and this information is needed for the establishment of effective health-promotion measures.

Clinical and radiographic features

Clinical Characteristics Localized aggressive periodontitis usually has an age of onset around puberty. Clinically, it is characterized as having "localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors". The localized distribution of lesions in localized aggressive periodontitis is characteristic but as yet unexplained.[17] The following possible reasons for the limitation of periodontal destruction to certain teeth have been suggested:

1. After initial colonization of the first permanent teeth to erupt (the first molars and incisors), Actinobacillus actinomycetemcomitans evades the host defenses by different mechanisms, including production of polymorphonuclear leukocyte chemotaxis-inhibiting factors, endotoxin, collagenases, leukotoxin, and other factors that allow the bacteria to colonize the pocket and initiate the destruction of the periodontal tissues. After this initial attack, adequate immune defences are stimulated to produce opsonic antibodies to enhance the clearance and phagocytosis of invading bacteria and neutralize leukotoxic activity. In this manner, colonization of other sites may be prevented. A strong antibody response to infecting agents is one characteristic of localized aggressive periodontitis.

2. Bacteria antagonistic to A. actinomycetemcomitans may colonize the periodontal tissues and inhibit A. actinomycetemcomitans from further colonization of periodontal sites in the mouth.

3. A. actinomycetemcomitans may lose its leukotoxin producing ability for unknown reasons. If this happens, the progression of the disease may become arrested or retarded and colonization of new periodontal sites averted.

4. The possibility that a defect in cementum formation may be responsible for the localization of the lesions has been suggested. Root surfaces of teeth extracted from patients with localized aggressive periodontitis have been found to have hypoplastic or aplastic cementum. A striking feature of localized aggressive periodontitis is the lack of clinical inflammation despite the presence of deep periodontal pockets.

Furthermore, in many cases the amount of plaque on the affected teeth is minimal, which seems inconsistent with the amount of periodontal destruction present. The plaque that is present forms a thin biofilm on the teeth and rarely mineralizes to form calculus." Although the quantity of plaque may be limited, it often contains elevated levels of A. actinomycetemcomitans, and in some patients, Porphyromonas gingivalis.

Aggressive periodontitis (AgP) comprises a group of rare, often severe, rapidly progressive forms of periodontitis often characterized by an early age of clinical manifestation and a distinctive tendency for cases to aggregate in families. In the absence of an etiologic classification, aggressive forms of periodontal disease have been defined based on the following primary features **(Lang et al.1999):**

- Non-contributory medical history
- Rapid attachment loss and bone destruction
- Familial aggregation of cases

Secondary features that are considered to be generally but not universally present are

- Amounts of microbial deposits inconsistent with the severity of periodontal tissue destruction
- Elevated proportions of Actinobacillus actinomycetemcomitans (recently renamed Aggregatibacter actinomycetemcomitans) and, in some Far East populations, Porphyromonas gingivalis
- Phagocyte abnormalities
- Hyper-responsive macrophage phenotype, including elevated production of prostaglandin E2 (PGE2) and interleukin-1β (IL-1β) in response to bacterial endotoxins
- Progression of attachment loss and bone loss may be self-arresting.

Clinical Features of Localised and Generalized Aggressive Periodontitis

The international classification workshop identified clinical and laboratory features deemed specific enough to allow subclassification of AgP into localized and generalized forms **(Lang et al. 1999; Tonetti & Mombelli 1999)**. The following features were identified: Localized aggressive periodontitis (LAP):

- Circumpubertal onset
- Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors.
- Robust serum antibody response to infecting agents.

Secondary features of LAP may also be present including;

- diastema formation with disto-labial migration of the incisors
- increased mobility of the affected teeth, sensitivity due to exposed root,
- deep dull pain that radiates to the jaw
- periodontal abscess with lymph node enlargement

Generalized aggressive periodontitis (GAP)

- Usually affecting persons under 30 years of age, but patients may be older
- Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors.
- Pronounced episodic nature of the destruction of attachment and alveolar bone.
- Poor serum antibody response to infecting agents.

Radiographic Features of Localized Aggressive Periodontitis

Radiographically, the periodontal lesion often presents with alveolar bone loss in a vertical pattern at the interproximal surface of the permanent first molars and usually horizontal bone pattern of bone loss at the interproximal surface of the incisors as the bone is thinner than at the interproximal surface of the molars.

The alveolar bone loss patterns are usually bilateral and similar on both sides and have been referred to as being a ‘mirror-image’ pattern. In advanced cases the alveolar bone loss may

be depicted as a horizontal bone loss pattern radiographically.

Radiographic Features of Generalized Aggressive Periodontitis

In GAP, generalized bone destruction is present that ranges from mild crestal bone resorption to severe alveolar bone destruction, depending on the severity of the disease. There may be a combination of vertical and horizontal bone loss defects.

Microbiological features

Patients with aggressive periodontitis present certain secondary features, such as elevated proportions of Aggregatibacter Actinomycetemcomitans (Aa) and in some populations, Prophyromonas gingivalis (supposedly in subgingival plaque). Multiple research studies examined a broad spectrum of bacteria using DNA technologies. In one-half of the studies Aggregatibacter actinomycetemcomitans was implicated as a risk marker, and in another half Porphyromonas gingivalis, Tannerella forsythia, and Selenomonads emerged as markers of risk. Several specific microorganisms frequently are detected in patients with localized aggressive periodontitis (A. actinomycetemcomitans, Capnocytophaga sp., Eikenella corrodens, Prevotella intertnedia, and Campylobacter rectus), A. actinomycetemcomitans has been implicated as the primary pathogen associated with this disease. As summarized by Tonetti and Mombelli, this is based on the following evidence:

(1) A. actinomycetemcomitans is found in high frequency (approximately 90%) in lesions characteristic of localized aggressive periodontitis,

(2) sites with evidence of disease progression often show elevated levels of A. actinomycetemcomitans,

(3) many patients with the clinical manifestations of localized aggressive periodontitis have significantly elevated serum antibody titers to A. actinomycetemcomitans,

(4) clinical studies show a correlation between reduction in the subgingival load of A. actinomycetemcomitans during treatment and a successful clinical response, and

(5) A. actinomycetemcomitans produces a number of virulence factors that may contribute to the disease process.

Serotypes

Six serotypes (a, b, c, d, e and f) of A. actinomycetemcomitans have been described based on the composition of structurally and antigenically distinct O-polysaccharides of their lipopolysaccharides. In addition, a novel serotype g has also been proposed. There also exist phenotypically non-serotype strains of A. actinomycetemcomitans that lack expression of serotype-specific polysaccharide antigen. The prevalence of the serotypes is influenced by a number of factors. Although some A. actinomycetemcomitans-positive subjects harbor multiple serotypes, the majority carry only one clonal type. Moreover, a number of mutations within the core genome of A. actinomycetemcomitans contribute to further differences, where the highly virulent JP2 genotype has attracted most of the attention.

Leukotoxin

One of the most studied virulence factors of Aggregatibacter actinomycetemcomitans is leukotoxin. This toxin is a 116 kDa protein produced by 56% of strains isolated from LJP patients.

Location: It is a proteinaceous toxin secreted from the cell membrane of Aggregatibacter actinomycetemcomitans.

Structure and composition: Leukotoxin is a member of the RTX family of toxins that produce pore-forming hemolysins. The leukotoxin operon consists of four coding genes designated ltxC, ltxA, ltxB, and ltxD and an upstream promoter gene.

- ltxA encodes the structure of the toxin.
- ltxC encodes for components required for posttranslational acylation of the toxin.
- ltxB and ltxD encodes for transport of the toxin to the bacterial outer membrane.

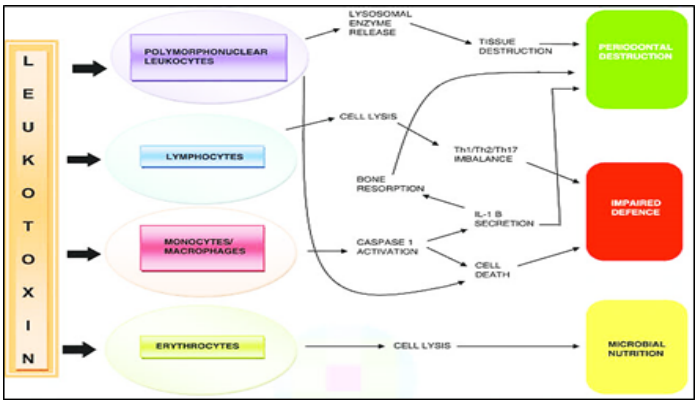

Figure 1: Effect of Aggregatibacter actinomycetemcomitans leukotoxin on human blood cells causing periodontal inflammation and tissue destruction.

Implants In Relation To Aggressive Periodontitis

The goal of periodontal therapy is the maintenance of the dentition and/or its implants placement in a state of health, comfort, function, and aesthetics for the duration of a patient's life. Patients with a history of aggressive periodontitis represent a unique group of individuals who previously submitted to a bacterial challenge. Therefore, it is important to address the management and survival rate of implants in these patients. In Aggressive Periodontitis patients, often unmodifiable factors would potentially plays an important role in implant success. [(27)]

These factors include:

1. Genetic polymorphisms;
2. Alterations of the immune system (phagocyte abnormalities and hyper-responsive macrophage phenotype, altered PMN transendothelial migration and signaling functions, reduced chemotactic response, and depression in phagocytosis and superoxide production).
3. Depression, stress, and loneliness;
4. Oral hygiene,
5. Tobacco consumption.

Though reports exist regarding the successful placement of implants in patients with AgP, the general consensus is that the placement of implants in these patients is unpredictable. The long-term prognosis has been questioned in these patients. **De Boever, Kim KK et al.** performed a study in patients with GAP conducted over 18 months and reported that marginal bone loss and inflammation was not found around all implants. The survival rate of those implants was100%.[(28)]

Another 5 years follow-up study, was conducted by **Ellegaard B et al**. in patients with GAP had shown that 45% of implants exhibited marginal bone loss of 1.5 mm and 30% cases displayed pockets of more than 6 mm. [(29)]

A study on a patient with GAP was conducted by **Yalçin S, Wu AY, and Kim KK et al** over 18 months reported that marginal bone loss and inflammation was not found around all implants. The survival rate of those implants was100%.On the contrary, it has been found that once the disease is controlled, the implants can be placed successfully**. Mengel et al.** showed a successful follow-up of upto 10 years in partially edentulous subjects of GAP with osseointegrated implants. However, the bone and attachment loss at the implants were higher than in periodontally healthy subjects**.**[(30)]

In a prospective longitudinal 10 years cohort study which was done by **Karoussis IK et al** has demonstrated that oral implants may successfully be placed and maintained in patients with and without a history of periodontitis. However, patients with a history of periodontitis yielded lower survival (90.5% vs. 96.5%), significantly higher complication (28.6% vs. 5.8%) and significantly lower success rates (e.g., 71.4% vs. 94.5%) than patients who had lost their teeth for reasons other than periodontitis.[(31)]

Treatment Plan

The overall treatment concepts and goals in patients with aggressive periodontitis are not markedly different from those in patients with chronic periodontitis. Therefore, the different treatment phases (systemic, initial, re-evaluation, surgical, maintenance, and restorative) are similar for both

types of periodontitis. However, the considerable amount of bone loss relative to the young age of the patient and the high rate of bone loss warrants a well-thought-through treatment plan and an often more aggressive treatment approach, in order to halt further periodontal destruction and regains as much periodontal attachment as possible. [32]

Nonsurgical Periodontal Therapy

Scaling and root planing: Scaling and root planing in patients with LAP improves the clinical parameters, but with the limited data present it is unclear to know the predictability and long-term stability of scaling and root planing (SRP) in LAP. The effect of SRP is well-documented in patients with GAP. Patients with GAP respond well to SRP in short term (6 months), after 6 months, relapse, and disease progression is reported despite frequent recall visits and oral hygiene reinforcements.

Systemic antibiotics: Treating patients with aggressive periodontitis is challenging. The disease responds less predictably to conventional mechanical periodontal therapy; hence scientists have been exploring adjunctive treatment to improve the outcome, predictability of the conventional mechanical therapy. Systemic antibiotics like tetracycline, metronidazole, combination of metronidazole and amoxicillin, clindamycin, and azithromycin are also used as adjunct in the treatment of aggressive periodontitis.

Local antimicrobials Agents like 1% chlorhexidine gel, 40% tetracycline gel, tetracycline fibers, and chlorhexidine chip have been used as local antimicrobials in the treatment of LAP and GAP. Unsal, Purucker, Kaner, and Sakellari have done studies on treating aggressive periodontitis using local antimicrobials. The studies concluded that the adjunct effect of local antimicrobial is not clear and do not seem to improve on the adjunct effect of systemic antibiotics. Therefore, it seems reasonable that the decision to use this type of treatment modality should be made on an individual basis rather than be evidence-based.

Surgical Therapy

Modified Widman flap surgery alone or in combination with tetracycline is effective in reducing the pocket depths and pathological microbial load. Modified Widman flap with systemic administration of amoxicillin and metronidazole combination is also beneficial in treating aggressive periodontitis. **Christersson, Lindhe** and **Liljenberg, Mandell** and **Socransky,** and **Buchman** have done extensive research on access surgery alone or in combination with antibiotics in treating aggressive periodontitis and concluded that access surgery in combination with systemic antibiotics was effective than access surgery alone. [32] Teeth used as abutments for fixed constructions in aggressive periodontitis patients are more prone for extractions during follow-up period of 10 years **(Yi et al., 1995 and Lulic et al., 2007)**. Pretzel indicated double rate of tooth loss used as abutments in fixed constructions over 10 years than teeth that are not used as abutments. The reason might be because of decreased accessibility for cleaning leading to risk for reinfection and progression of disease. [33]

Maintenance therapy

Once treatment has resulted in a stable and healthy periodontium, the patient should enter a maintenance program. The purpose of this supportive periodontal therapy is to ensure that periodontal health is maintained after active therapy, so that no additional teeth are lost and disease recurrence is prevented. Supportive periodontal therapy should therefore be directed towards risk factors for disease recurrence and tooth loss. Several factors (such as smoking, diabetes mellitus, age, irregular supportive periodontal therapy and ineffective plaque control) have

been shown to increase the risk for tooth loss during supportive periodontal therapy in patients with chronic and aggressive periodontitis.

Conclusion

Aggressive Periodontitis is an impressively destructive disease that is accompanied by devastating loss of self-esteem and costly dental procedures. The exact etiology of Aggressive Periodontitis remains mysterious. Although it is doubtless that bacteria has major role in the disease, it is unclear why there is an exaggerated response to minimal plaque accumulation. Genetic predisposition is a major influence in the manifestation of AgP and familial aggregation is one of its defining characteristics. In contrast to genes affecting cytokines, another possible genetic contribution includes dysregulation of phagocytosis of invading bacteria.

The mystery of Aggressive periodontitis can be partly attributed to the complexity of the host immune response; however, further research in this area is necessary to increase our understanding of the pathophysiology. Another interesting question that arises is the clinical presentation of both localized and generalized forms of the disease. The pathophysiologic processes behind only molar and incisor teeth being affected in the localized form of the disease remain incompletely understood.

With so many anonymous, Aggressive periodontitis remains a mystery and warrants essential further research both to allow for definitive diagnosis and to increase our basic understanding so that effective new treatment strategies can be developed for better outcomes for patients.

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