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AGGRESSIVE PERIODONTITIS: A REVIEW

Dental Science

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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] 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.[2] 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.[3] 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.[4]

CLASSIFICATION

Table 1: Diagnostic Criteria to Distinguish Chronic and Aggressive Periodontitis [5]

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 Commensurate with etiologic factors localized aggressive periodontitis)	Commensurate with etiologic factors

CLINICAL AND RADIOGRAPHIC FEATURES

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.[6] 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.

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

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.

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 Opolysaccharides 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 serotypespecific polysaccharide antigen. The prevalence of the serotypes is influenced by a number of factors.

TREATMENT PLAN

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 welldocumented 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.

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.[7] 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).[8]

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. 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.

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