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REVIEW ARTICLE

Chronic versus aggressive periodontitis - A comprehensive review from parity to disparity

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Abstract

The imbalance between bacterial virulence and host response leads to the destruction of tooth and tooth-supporting structures, leading to most commonly occurring chronic periodontitis and rare, but not uncommon aggressive periodontitis and the dilemma between diagnosis, prognosis, and treatment planning of these two entities have always been a challenge to clinician at least in few situations. Hence, this article aims at reviewing the shared characteristics (similarities) and unique characteristics (dissimilarities) of chronic and aggressive forms.

Introduction

Over the past 20 years, it has been proven that periodontitis is the result of interaction between host's defense mechanisms and biofilms containing complexes.[1] Research regarding carriers of the disease is a topic of research these days. The disease does not occur till the natural balance between the host and pathogen gets disturbed. Environmental influences and an opportunistic increase in the number of organisms lead to such disturbances.[2]

The imbalance between bacterial virulence and host response leads to the destruction of tooth and tooth-supporting structures, leading to most commonly occurring chronic periodontitis and rare, but not uncommon aggressive periodontitis (AgP) and the dilemma between diagnosis, prognosis, and treatment planning of these two entities have always been a challenge to clinician at least in few situations. Hence, this article aims at reviewing the shared characteristics (similarities) and unique characteristics (dissimilarities) of chronic and aggressive forms.

Definition

Chronic periodontitis has been defined by the American Academy of Periodontology (AAP) as "an *infectious disease* resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss, and bone loss."

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 (AAP).[3]

Nomenclature and change in concepts of etiology

Chronic periodontitis

Ancient Egyptian and Chinese writings would suggest that periodontal diseases were recognized possibly 5000 years ago. John W Riggs (1811–1885) importance of local irritants in the etiology was emphasized and was known as "Riggs' disease."Armitage and cullinan (1870-1920) called it a "pyorrhea alveolaris" that means pus oozing out of alveolus. From 1920 to 1970, the major influence was the classic pathology paradigm, and from 1970 to the present day, the infection/host response paradigm. The AAP in the 1966 World Workshop in periodontics accepted the term chronic marginal periodontitis, but the workshop failed to produce a definite system of classification

for periodontitis. In 1982, Page and Schroeder identified five distinctly different forms of periodontitis as prepubertal, juvenile, rapidly progressive, adult, and acute necrotizing ulcerative gingivoperiodontitis.[4] 1989 AAP classification usage of adult periodontitis was continued, and in 1999, it was addressed as chronic periodontitis.

In 1890 black and miller gave non specific plaque hypothesis. In 1976, Walter J. Loesche proposed "Specific Plaque Hypothesis." Else Theilade, in 1986, updated "non-specific plaque hypothesis" (non-specific plaque hypothesis revisited). In 1994, Philip D. Marsh considered ecological plaque hypothesis. George Hajishengallis *et al.* (2012) gave key stone hypothesis.[5]

AgP

Black in 1886 has called it phagedenic, pericementitis and chronic suppurative pericementitis. In 1923 gottileb called it diffuse atrophy of alveolar bone." In 1928, Gottlieb attributed this condition to the inhibition of continuous cementum formation, "deep cementopathia." In 1938, Wannenmacher described incisor and first molar involvement and called the disease "parodontitis marginalis progressiva." Later periodontosis was the name given by Weinmann in 1942 and Thoma and Goldman called it paradontosis. Juvenile periodontitis by caput and butler and continued usage by AAP in 1977 and 1986 later called early-onset periodontitis by Page and Baarb which was accepted by AAP in 1989, finally in 1999 named as AgP discarding all the earlier believes and terminology 2017 AAP classification the name is removed due to insufficient evidence calling it as AgP.[6]

Clinical features

Chronic periodontitis

Periodontal inflammation is consistent with the local factors attachment as well as alveolar bone loss is seen. Localized chronic periodontitis has a clear pattern in 30% of teeth. Generalized chronic periodontitis occurs without a clear pattern and >30% of teeth are affected.[7]

AgP (Lang and Lindhe, 1999): Primary features include non-contributory medical history, rapid attachment loss and bone destruction, and strong familial aggregation of cases. Secondary features include microbial deposits inconsistent, increased AA levels and, Porphyromonas gingivalis, phagocyte abnormalities, hyperresponsive macrophage phenotype, which include increased prostaglandin E2 (PGE2)] and interleukin-1β (IL-1β) as a response to bacterial endotoxins and self-arresting attachment and bone loss.[2] Localized AgP shows circumpubertal onset, with localized first molar/incisor pattern with interproximal attachment loss on at least two permanent teeth, one of which is the first molar and involving not more than two teeth other than first molars and incisors and robust serum antibody response for infecting agents. Generalized AgP usually affects persons under 30 years of age, but patients may be older, generalized 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 and poor serum antibody response to infecting agents.[7]

Radiographic findings

Chronic periodontitis

Fuzziness and discontinuity in lamina dura with wedge-shaped radiolucent area reduced height of interdental septum and horizontal bone loss are commonly seen.[8]

AgP

Bilateral bone loss mostly in mandibular molars and incisors with arc-shaped radiolucency and later may be generalized bone loss but predominantly in premolar areas.[3]

Disease progression

Chronic periodontitis progression is measured by the continuous model, the random or episodic-burst model, the asynchronous, and multiple burst model of disease progression.[8]

AgP progression is measured by burst hypothesis: Alternate stages of quiescence and destruction.[4]

Pathogenesis

Microbiological

Bacteria

Plaque accumulation is considered the primary initiating agent in the etiology of gingivitis and chronic periodontitis. Attachment and bone loss are associated with an increase in the proportion of Gram-negative organisms in the subgingival biofilm, with specific increase in organisms such as *P. gingivalis*, *Tannerella forsythia*, and *Treponema denticola* - otherwise known as the "red complex."[9] A.a has been considered the primary pathogen for AgP, especially in its localized form. Six serotypes of A.a (a, b, c, d, e, and f) are described. Filifactor alocis is Gram-positive anaerobic rod which is elevated in AgP patients. *Treponema lecithinolyticum* and *Treponema socranskii* are elevated in GAP along with P.g. sulfate-reducing bacteria and desulfomicrobium orale Yamabe suggested Archaea a methanogenic organism, especially methanobrevibacter oralis as putative periodontal pathogen for AgP.[10]

Virus

Plaque-induced gingivitis has increased inflammatory content and virus exists in latent state, which gets activated in conditions such as immunosuppression, infection, stress level, and hormonal effect leads to induction of periodontopathogenic property further causing periodontal destruction.[11] Herpes virus-infected sites show more break down and the presence of virus enhances the bacterial adherence which results in increased

bacterial load which further increase and alter the inflammatory mediators.[12] Herpes was less associated with aggressive when compared to chronic.[13]

Immunological

The onset, progression, and severity of chronic periodontitis depend on the individual host's immune response. Patients may show alterations in their peripheral monocytes, which are related to the reduced reactivity of lymphocytes or an enhanced B-cell response. Macrophages, periodontal ligament cells, gingival fibroblasts, and epithelial cells synthesize pro-inflammatory mediators that modify innate and adaptive immune responses at periodontal sites. "The differences between both disease entities only reflect variations in the degree of the severity of susceptibility rather than actual different immune pathologies.[14]" PGE2 production, in particular, has been shown to be highly elevated in AgP subjects when compared to periodontally healthy individuals and patients with chronic periodontitis. Specific antibodies against AgP-associated microorganisms and cleaved complement fragments have also been detected in crevicular fluid from AgP lesions. Substantial titers of antibodies against Aggregatibacter actinomycetemcomitans and P. gingivalis have also been detected in the serum of AgP patients.[14]

Genetic

A great deal of research is underway attempting to identify the genes and polymorphisms associated with all forms of periodontitis. Much attention has focused on polymorphisms associated with the genes involved in cytokine production. Genetic variations such as single nucleotide polymorphisms (SNPs) and genetic copy number variations may directly influence innate and adaptive immune responses as well as the structure of periodontal tissues. Conflicting data in the literature indicate inconclusive knowledge of SNPs potential role in the heritability and etiopathology of periodontitis.

Although formal genetic studies of AgP support the existence of a gene of major effect, it is unlikely that all forms of AgP are due to the same genetic variant (Hart, 1996; Loos et al., 2005). This notion is consistent with the fact that numerous diseases and syndromes with similar clinical appearance are known to result from different genetic polymorphisms. Based on the current knowledge that AgP subjects have a high prevalence of polymorphonuclear neutrophil (PMN) functional defects that they produce high levels of inflammatory mediators in response to lipopolysaccharide stimulation and that connective tissue homeostasis is relevant in periodontitis, several loci have been proposed as genes conferring increased susceptibility to AgP. Allelic variations in the Fc receptor for IgG2 have also been suggested to play a role in suboptimal handling of A. actinomycetemcomitans infections. PMNs expressing the R131 allotype of FcγRIIa show decreased phagocytosis of A. actinomycetemcomitans.

Taken together, these data indicate that there is evidence for a strong genetic component in AgP. It is very likely that the low number of identified and replicated susceptibility loci for AgP is the result of too low sample numbers and issues in study design than to a lacking biological association.

Environmental

In addition to microbial, immunologic, and genetic factors, the development and progression of chronic periodontitis is further influenced by environmental and behavioral factors such as smoking and psychological stress. There are only a few studies in literature that specifically examine the relationship between smoking and AgP. Most studies agree that smoking is a risk factor for patients with AgP, resulting in increased periodontal disease severity.[15]

Diagnosis

Assessment of clinical findings

Chronic periodontitis is diagnosed by the presence of gingival changes as may be evidenced for gingivitis plus the presence of reduced resistance of the tissues to periodontal probing with a deeper gingival sulcus or "pocket" which reflects loss of periodontal attachment. Tooth mobility and migration must also be assessed. It is, however, important to realize that mobility is not by itself diagnostic of periodontitis and may be the result of occlusal trauma as may be migration of teeth which may be segmental or single tooth migration. Mobility and migration solely related to periodontitis are usually late symptoms of the disease and are possibly of more importance in assessing prognosis and treatment planning.[4] In patients with significant findings, a more thorough clinical assessment that includes the evaluation of periodontal probing depth and attachment loss at six sites per tooth should be performed. In addition, patients who are at risk for developing AgP should be more closely monitored. These are primarily subjects with a known familial aggregation of AgP. The diagnosis of systemic health is made by checking the medical history of the patient, usually with the use of a questionnaire and a short interview; this process is repeated every other year. In subjects with suspected AgP, the destruction primary care physician can be consulted to rule out underlying systemic illnesses or conditions.[3]

Assessment of radiographic findings

Radiographic evidence of periodontal bone loss is a very specific but not very sensitive diagnostic sign of periodontitis. It has been suggested to screen for bone loss in younger children and adolescents with the use of the bitewing radiographs that are regularly obtained for the early detection of dental caries; in this population with erupting permanent teeth, periodontal probing can be difficult. A distance of 2 mm between the cementoenamel junction and the alveolar bone crest in these patients could be a sign of periodontitis and may warrant a more thorough examination.

The vertical loss of alveolar bone around the first molars and incisors, which begins around puberty in otherwise healthy teenagers, is a classic diagnostic sign of LAP. Radiographic findings may include an "arc-shaped loss of alveolar bone extending from the distal surface of the second premolar to

the mesial surface of the second molar." Bone defects are usually wider than those that are usually seen with chronic periodontitis.[3]

Assessment of genetic features

Because recent large-scale and well-controlled studies could not replicate earlier reports from smaller collectives of an association of candidate genes (e.g., IL-1) with AgP, it seems unwarranted at the present time to test putative AgP patients for the presence of DNA polymorphisms or other genetic markers.[3]

Assessment of host defense

In a suspect case of rapidly progressing AgP, one may consider the use of one of the commercially available tests for markers of ongoing periodontal inflammation and tissue breakdown in either gingival crevicular fluid or saliva (e.g. matrix metalloproteinase-8) or other areas. Dip test: Test used for the detection of matrix metalloproteinase-8 (MMP8) in gingival crevicular fluid and integrated microfluidic platform for oral diagnostics test used for the detection of MMP8 in saliva. Still, none of the available tests to date can reliably discriminate AgP from other conditions, and thus, the utility of these tests to assess ongoing tissue breakdown is disputed.[3]

Prognosis

Prognosis for chronic periodontitis depends on overall factors which includes age, severity, control, compliance; systemic and environmental factors; local factors, plaque and calculus, and subgingival restoration; and anatomic factors tooth mobility and prosthetic and restorative factors.

The prognosis of AgP depends on if it is localized or generalized, localized has a better prognosis then generalized because less number of teeth involved and also due to the presence of antibodies (robust) in the serum of patients. Destruction at the time of diagnosis and ability to control progression are also important factors for prognosis.[3]

Treatment

In patients with chronic periodontitis, subgingival debridement is an effective treatment in reducing probing pocket depth (PPD) and improving the clinical attachment level.[16] Scaling and root planing of juvenile periodontitis lesions could not predictably suppress A. actinomycetemcomitans below detection levels. Unfortunately, the response of AgP to conventional therapy alone has been limited and unpredictable. The earlier the disease is diagnosed, the more conservative the therapy and the more predictable the outcome.[3]

A comprehensive meta-analysis of non-surgical treatment by Haffaje *et al.* reported that the added effect of adjunctive antibiotic therapy has been found to be modest in chronic periodontitis; however, future studies are needed to confirm these results.[17] Sgolastra *et al.* supported the effectiveness of scaling and root planing and metronidazole and amoxicillin in AgP.[18]

A systematic review and meta-analysis showed significant improvement in periodontal parameters in favor of tetracycline as local drug delivery compared to placebo in chronic periodontitis, whereas this treatment failed to either stop the progression of attachment loss at these sites or eliminate A.a in LAP. It is possible that this failure was caused by the inability of tetracycline to adequately penetrate the pocket epithelium or possibly as a result of the repopulation of *A. actinomycetemcomitans* from other potential reservoirs in the mouth.[19]

Host modulation in AgP acts by controlling the action of mmp, proteinases, collagenases, and gelatinases. The adjunctive use of SDD with SRP is statistically more effective than SRP alone in reducing PD and achieving CAL gain in chronic periodontitis than AgP.[20]

In the treatment of deep pockets, open flap debridement results in greater PPD reduction and clinical attachment gain in chronic periodontitis.[21] Resective periodontal surgery can be effective to reduce or eliminate pocket depth in AgP patients. However, it may be difficult to accomplish if adjacent teeth are unaffected.[22]

Periodontal regenerative procedures have been successfully demonstrated in patients with chronic and AgP. Potential for regeneration in patients with AgP appears to be good; expectations are limited for patients with severe bone loss. This is especially true if the bone loss is horizontal and if it has progressed to involve furcation.[3]

Response to treatment

Chronic periodontitis

The bacterial response following treatment is fairly consistent following scaling and root planing in chronic periodontitis. Depending on the regimen, adding antibiotics to the treatment may further suppress the pathogenic microbiota.[3]

In AgP, the combination of oral hygiene instructions, along with subgingival scaling and root planing, reduced, but did not eliminate, the number of spirochetes, A. actinomycetemcomitans, and Capnocytophaga species resulted in a small improvement in post-treatment probing depths. Moreover, the presence of multiple periodontal pathogens may predict challenges in achieving a favorable outcome for AgP.[23]

Present consensus

Despite substantial research on AgP since the 1999 workshop, there is currently insufficient evidence to consider aggressive and chronic periodontitis as two pathophysiologically distinct diseases. The current multifactorial models of disease applied to periodontitis appear to account for a substantial part of the phenotypic variation observed across cases as defined by clinical parameters.[24]

Conclusion

Ongoing and future research may help to define prognostic

subtypes or profiles within aggressive versus chronic periodontitis indicating a higher risk for rapid periodontal progression or a poorer response to therapy. The task force affirmed that the diagnosis of aggressive or chronic periodontitis has important implications related to therapy, long-term prognosis, and specialty referral. In addition, future research involving modulation of host inflammatory responses may clarify the reasons for the differences in clinical outcomes between patients.

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