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# AN APPRAISAL OF DIFFERENCES IN CHRONIC AND AGGRESSIVE PERIODONTITIS!

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ABSTRACT

There are two varieties of vicious periodontal diseases i.e. chronic periodontitis and aggressive periodontitis, one has a systemic etiology while the other has a local etiology. Since the 18th century various terms have been attributed to this disease such as alveolar atrophy, diffuse atrophy, Schmutz Pyorrhea, periodontosis, paradentosis, juvenile periodontitis, early onset periodontitis, periodontitis simplex, paradentitis marginalis superfacilis, paradentitis, chronic marginal periodontitis, adult periodontitis etc. Distinguishing features of aggressive periodontitis include rapid rate of disease progression, discrepancy between the amounts of microbial deposits and the extent of destruction and familial aggregation. Chronic periodontitis, however, has more prevalence in adults, consistency between microbial deposits and amount of destruction and slow rate of disease progression. This article reviews countless variances between these two diseases which is essential for understanding the etiology and formulating a treatment plan for the individual.

## Introduction

Periodontitis is an intricate and multifactorial ailment that is a result of a combination of factors such as plaque, genetic factors, environmental aspects such as smoking, anxiety, oral hygiene levels and systemic diseases.[1] It is well established that both chronic and aggressive periodontitis are complex illnesses that arise in vulnerable hosts and originate by biofilms that form on tooth surfaces. These disease manufacturing biofilms encompass microorganisms that are indigenous to the oral cavity.[2] A host immune-inflammatory reaction to these biofilms is accountable for the destruction of periodontal tissues observed.[3] The treatment of both types of periodontitis includes anti-infective nor-surgical and surgical periodontal therapy.[4] If untreated, the final outcome is tooth loss.

## Historical evaluation of shifting diagnostic terms[1,5]

The earliest literature on periodontitis reports back to the 1890s when Dr. John Riggs described periodontal disease as Riggs Disease or pyorrhoea.

- Late 1800s- Chronic Periodontitis was regarded as as a slowly progressive annihilation of periodontium due to accretion of "lime deposits" on the teeth or calcic inflammation of the peridental membrane.
- G.V Black- applied the terms "phagedenic pericementitis" and "chronic suppurative pericementitis" to describe patients that suffered from a rapid destruction of alveolar bone.
- Bernard Gottlieb 1923- described a bizarre form of periodontal disease that principally affects the incisors and first molars and termed it as diffuse atrophy of alveolar bone. He suggested that the disease was caused by a lack of cementum barrier.
- Lyons 1950- degenerative non inflammatory disease
- Becks – "Genuine paradentosis"
- Orban and Weinmann 1942- pioneered the term "Periodontosis" to depict severe periodontal disease in young individuals.
- 1966- World Workshop in Periodontics "Periodontosis" term eliminated
- Butler 1969- "Juvenile Periodontitis"
- Paul Baer 1971- inscribed a paper in which he proposed a definition for aggressive periodontitis based on the clinical features of the disease.
- American Academy of Periodontology 1989- "Early onset periodontitis". This term was easily adopted because it portrays the premature onset development of the disease and its prevalence in younger age group.
- 1999- Classification of American Academy of Periodontology- a consensus report accepted the term "Aggressive Periodontitis" as a novel name for this disease swapping the term "Early onset periodontitis" and "Chronic periodontitis" for "Adult periodontitis".

## Distinction in clinical features of chronic (CP) and aggressive periodontitis (AgP)

- Rate of progression- The rate at which the supporting periodontal tissue is lost is considered as an important factor to distinguish between the forms of periodontitis. Chronic periodontitis has a slower pace of disease progression; whereas aggressive periodontitis progresses at a faster rate (3-4 times faster than chronic periodontitis).[6] A radiographic survey has shown that a quicker linear pattern of progression is observed in aggressive periodontitis as compared to chronic periodontitis 0.31 mm/year vs 0.20 mm/year.
- Familial aggregation of cases is seen in Aggressive Periodontitis cases.[6]
- Age of onset- Age of onset or age at the time of revealing of the disease is an important factor in deciding the diagnosis of the periodontal disease. According to the 1999 Classification, age is not an apt descriptor for utilization in diagnostic categories.[2] Given the comparable amounts of periodontal damage in terms of probing pocket depths, clinical attachment loss and bone loss, individuals with aggressive periodontitis are considerably youthful than individuals with chronic periodontitis. However, there is no specific disconnecting age to differentiate between chronic and aggressive periodontitis.
- Localised AgP has a circumpubertal onset, a strong antibody response and a first molar/incisor presentation. Generalized AgP affects individuals below 30 years of age with meagre serum antibody response to the contaminating agents. Localized and generalized forms of chronic periodontitis are two clinical appearances of the identical disease.
- Patterns of destruction– Chronic periodontitis does not entail a pattern of specific number and types of teeth involved. However, generalized aggressive periodontitis includes situations where there is generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors.[7]
- Clinical indications of inflammation- It was observed that the clinical signs of inflammation such as redness and swelling were significantly less in localised aggressive periodontitis cases, which suggest that it is a degenerative non-inflammatory disease.[6,8] However, deep probing depths with enormous loss of periodontal support are also observed in the late stages of aggressive periodontitis. In cases of generalized aggressive periodontitis, however, intense gingival inflammation is seen. These differences in clinical picture are almost certainly related to the occasion of initial presentation and amount of local deposits on the tooth surfaces that accumulate over time.
- Relative abundance of plaque and calculus- The amount of local deposits in cases of localized aggressive periodontitis are very scanty although no affected site is biofilm free. The composition of the biofilm in aggressive periodontitis cases is relatively simple and sparse, whereas that in chronic periodontitis cases is complex and thick.[9]

**Microbiological differences amid chronic and aggressive periodontitis**

A comparison of subgingival microbiota between chronic and aggressive periodontitis is difficult due to the variability in case definitions and study designs in the available literature.[10]It has now become clear that the microbiota allied with LAP is dissimilar from that associated with GAP or chronic periodontitis, while the microbiota allied with GAP is dissimilar from that of chronic periodontiits.[2]

Mombelli et al 2002 executed a systematic review where he assessed whether the presence of absence of certain periodontal pathogens such as Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans etc, make out amid chronic and aggressive forms of periodontitis. The systematic review concluded that such was not the case and these microorganisms cannot differentiate the two forms of periodontitis.[11] Authors like Faveri et al 2008[12], Gajardo et al 2005[13] have directly compared the subgingival microbial profile in chronic and aggressive periodontitis patients. One such study that comprised of 17 chronic periodontitis patients and 6 generalized aggressive periodontitis patients was conducted where culture analysis of subgingival microbiota was done and it was found that P.gingivalis was isolated from 76.5% of chronic periodontitis patients and 100% in generalized aggressive periodontitis group. Also, C. Rectus was found in 23.5% of CP patients and 50% of GAP patients.[13] Lafaurie et al compared the subgingival microbiota by PCR methods in CP versus GAP patients. No key discrepancy was found with regard to the percentage of patients harboring P. gingivalis for chronic periodontitis vs. generalized aggressive periodontitis (approximately 76% vs. approximately 73%, respectively), T. forsythia (approximately 62% vs. approximately 54%), C. rectus (approximately 38% vs. approximately 32%), A. actinomycetemcomitans (approximately 17% vs. approximately 27%) and enteric rods (approximately 30% vs. approximately28%).[14] Riep et al conducted a study to compare the microbial profiles of patients with chronic versus aggressive periodontitis with oligonucleotide probes. The only statistically significant difference was found for T.lecithinolyticum in GAP subjects.[15]

Aggressive and chronic periodontitis can be distinguished by in depth herpesvirus analyses but not in line with the mere presence or absence of herpesvirus genomes.

**Immunological differences between chronic and aggressive periodontitis**

A study by Ford et al 2010 has considered the possibility of immunological differences between both forms of periodontitis.[16] The author has suggested some differences in the pattern of Toll like receptor activation in both chronic and aggressive forms of periodontitis. Toll like receptors are a set of proteins that play a fundamental role in the innate immune system, expressed by cells such as macrophages, dendritic cells that identify the pathogen associated molecular patterns of putative periodontal pathogens. Another immunological difference suggested by the author in chronic and aggressive forms of periodontitis is with regards to synthesis of defensins, that are antimicrobial peptides involved in the resistance of epithelial surfaces to microbial colonization. These fine differences however, do not suggest a different immunopathology for the two forms of periodontitis.

**Comparison of neutrophil function between chronic and aggressive periodontitis**

It is suggested that patients with localised aggressive periodontitis have an inherited trait of defective neutrophils characterised by reduced chemotaxis, phagocytosis and intracellular microbial killing responses.[17] This type of response is because of continual exposure to microbial products and inflammatory mediators. It is suggested that uncontrolled periodontal infections activates or primes neutrophils to a state of increased activity where they are able to combat the microbial infections. Such primed neutrophils are observed aggressive periodontitis cases.

**Response to treatment**

Chronic periodontitis: A meta-analysis of non-surgical treatment in chronic periodontitis patients reported that after scaling and root planing at sites with probing pocket depth of 4-6mm, a mean reduction in pocket depth of 1 mm and a clinical attachment level gain of 0.5mm can be expected. In sites with pocket depth > 7mm, a pocket depth reduction of 2mm and clinical attachment gain of 1mm could be expected. The added effect of antibiotics with scaling and root planing showed a significant additional 0.2-0.6mm decrease in pocket depth and 0.1-0.2mm of CAL gain.[18] Right away after subgingival debridement, there is a significant decrease in the number of gram negative organisms with an increase in gram positive cocci. This new microbiota stays stable for 4-8 weeks before returning to baseline by 12-24 weeks.[19] A systematic review by Heitz-Mayfield et al 2002[20] has shown that in pockets more than 6 mm surgical treatment resulted in an added 0.6 mm mean probing depth reduction and 0.2 mm added attachment level gain over non surgical therapy alone. In 4–6 mm pockets, surgical treatment gained an additional 0.4 mm decrease in probing depth, but a loss of 0.4 mm in attachment level beyond scaling and root planing. Bone grafting in the intrabony defects in chronic periodontitis patients by using different materials, led to a reduction in probing depth and an increase in clinical attachment level gain by 0.5-1mm in addition to the effects achieved only by surgical debridement.[21] A meta-analysis of studies on guided tissue regeneration in chronic periodontitis patients has shown better attachment levels and bone fill by 2.7mm and 2.1mm respectively.[22]

Aggressive periodontitis: The low prevalence of aggressive periodontitis makes it hard to conduct controlled clinical trials of various treatment modalities. Slots and Rosling assessed the result of scaling and root planing clinically and microbiologically on 20 deep pockets and 10 normal sites in six patients with LAP. They observed reduction but not elimination of microbial species like spirochetes and Aggregatibacter actinomycetemcomitans with minor improvement in probing depths. Slots & Rosling, in the final step of their staged treatment study, administered 1 g of tetracycline HCl per day for 14 days following subgingival debridement. The authors noted that after tetracycline treatment the number of spirochetes, A. actinomycetemcomitans and Capnocytophaga species were reduced to almost zero levels, and that this corresponded to a 0.3 mm gain in attachment level.[23] Lindhe & Liljenberg treated 16 cases of localized aggressive periodontitis with a combination of tetracycline and modified Widman flap surgery. After 5 years of maintenance, they found significant improvements in probing depths and attachment levels with evidence of radiographic bone fill.[24]

Hughes et al carried out a prospective intervention study of 79 patients suffering from generalized aggressive periodontitis. The patients received thorough non-surgical periodontal therapy in four visits. After 10 weeks, the initially deep sites showed a mean reduction in pocket depth of 2.11 mm and a mean attachment level gain of 1.77 mm. In addition to this, 32% of patients did not respond to this treatment, and smoking was observed as the biggest factor correlated with nonresponse.[25] Guerrero et al evaluated 18 patients with generalized aggressive periodontitis taking metronidazole amoxicillin in conjunction along with nonsurgical therapy. They found that subjects who were fully compliant in taking their medications had probing depth reductions of 0.9 mm and attachment level gains of 0.8 mm beyond those who were noncompliant or only partially compliant.[26]

Deas and Mealey suggested that both chronic and aggressive forms of periodontitis respond well to anti-infective therapies. Also, all forms of regenerative therapies also work well in both the cases. The long term success however, depends upon the stringency of supportive periodontal therapy.

**Implants in chronic and aggressive periodontitis**

It is controversial whether implant therapy in aggressive periodontitis patients is characterised by an increased incidence of failure in terms of peri-implant diseases and bone loss. A systematic review has shown that crestal bone loss around

implants in patients with generalized aggressive periodontitis as compared with implants in healthy patients or chronic periodontitis patients was not significantly greater in short-term studies but was significantly greater in long-term studies. In short term studies, the success rates of implants were between 97.4% and 100% in patients with generalized aggressive periodontitis and in long term studies it is between 83.3% and 96%.[27]

Mengel and Flores-de-Jacoby evaluated implant survival in 12 periodontally Healthy (PH), 12 Chronic Periodontitis (CP), and 15 generalized aggressive periodontitis (GAP) patients. In the 3 years after placing the abutment, marginal bone loss around implants was 1.14 mm in the GAP, 0.86 mm in the CP and 0.70 mm in the PH subjects. The authors reported that the 3-year implant survival rate was 100% in the PH and CP patients, and 97.4% of GAP patients. There were no major differences for the implant survival rate between the three groups.[28] De Boever et al report that the 46.8 ± 26.7 months implant survival rate was 84.8% in the GAP group and 96% in the CP group, and the 48.1 ± 25.9 months implant survival rate was 97% in the PH group. There was no difference in the survival rate of implants between PH and CP patients, however, GAP group had poorer implant survival implant rate.[29] Mengel et al. reported on a 5-year implant survival rate of 88.8% in GAP group and 100% in CP group.[30]

Thus, implant therapy is not contraindicated in aggressive periodontitis patients, however, a stringent oral hygiene regimen and a regular maintenance therapy is to be followed.

## Conclusion

The clinical division between chronic and aggressive periodontitis is not clear cut and is burdensome for clinicians. The treatment for both types of periodontitis, is however almost similar. The clinical differentiation is only required to comprehend the etiology and pathogenesis of the type of periodontitis. Also, it has an impact on research studies conducted on these patients. Thus, the diagnosis of the type of periodontitis purely lies at the hands of the clinician and it is what the clinician thinks is best for a given case.

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