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Treatment Parameters For Aggressive Periodontitis

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Abstract
Generalized aggressive periodontitis results in rapid destruction of the periodontium and can lead to early tooth loss in the affected individuals if not diagnosed early and treated appropriately. General dentists frequently encounter patients with aggressive periodontal disease and should be able to diagnose and manage this disease properly. It is important for the general dentist to diagnose, inform, and treat the periodontal patient accurately, using referral and nonsurgical, surgical, and antimicrobial/ antibiotic therapy. With the exponential rate of developments in periodontal research, regenerative therapy, tissue engineering, and genetic technologies, the future seems promising in regard to options at managing the disease. This article attempts to describe the current treatment options along with a suggested protocol for comprehensive management of generalized aggressive periodontitis patients.

Key Words
Aggressive periodontitis, treatment modalities.

Introduction

Aggressive periodontitis, as the name implies is a type of periodontitis where there is rapid destruction of periodontal ligament and alveolar bone which occurs in otherwise systemically healthy individuals generally of a younger age group but patients may be older. It tends to have a familial aggregation.[1],[2],[3] Aggressive periodontitis is an autosomal dominant triad with reduced penetrance. Parents, offspring and siblings of patients affected with aggressive periodontitis have a 50% risk of this disease.

Classification

At the International Workshop for Classification of Periodontal diseases and conditions in 1999, the classification of periodontal diseases was revised (Armitage 1999). Aggressive periodontitis now replaces the term "juvenile or early onset periodontitis". Aggressive periodontitis can be further classified into localized and generalized categories based on the specific features of the disease process.

The localized form of aggressive periodontal disease has a circumpubertal onset. Intraoral/radiographic examination reveals that the disease process is limited to the first molars and incisors with interproximal attachment loss on at least two permanent teeth, one of which is a permanent molar, and involving no more than two teeth other than the first molars and incisors.Several species of bacteria are detected in the localized form; however, Actinobacillus actinomycetemcomitans produces several virulence factors that help it evade the host's defense mechanisms. Following the initial assault, the host's defense mechanism produces a robust serum antibody response to the infecting agents, overcoming the neutrophil function abnormalities and localizing the disease process as a result.[4]

The generalized form of aggressive periodontal disease usually affects men and women over the age of 30, although patients may be older.Generalized aggressive periodontitis has a poor serum antibody response to the initial assault[4]. This response, along with the periodontal pathogen virulence factors, produces a disease process in which intraoral/radiographic examination reveals that the disease process has generalized interproximal bone loss affecting at least three permanent teeth other than the first molars and incisors.This attachment loss is episodic in nature and has periods of quiescence of variable lengths.This form of the disease frequently is associated primarily with Actinobacillus actinomycetemcomitans and Porphyromonas gingivalis and neutrophil function abnormalities . Other bacteria involved in the etiology of aggressive periodontitis are Eikenella corrodens and Capnocytophaga species.[5] It is important to note that not all of the features of Aggressive periodontitis need to be present or will be present in all cases.

Clinical Features

With regard to clinical and paraclinical aspects Aggressive periodontitis can be distinguished from chronic periodontitis. It is defined by following characteristics (Land et al . 1999) [6]

1) Except for the presence of periodontitis patients are clinically healthy.
2) Rapid attachment loss and bone destruction.
3) Familial aggregation.

Non constant characteristics of the disease -

1) Amounts of microbial deposits are inconsistent with severity of periodontal tissue destruction.
2) Elevated proportions of Actinobacillus actinomycetemcomitans and in some populations Porphyromonas gingivalis may be elevated.
3) Phagocyte abnormalities.
4) Hyper – responsive macrophage

Virulence Factors Of Actinobacillus Actinomycetemcomitans [7],[4]

Factors that promote colonization and persistence in the oral cavity - Adhesions, Invasions, Bacteriocins, Antibiotic resistance
Factors that interfere with the host's defenses - Leukotoxin, Chemotactic inhibitors, Immunosuppressive proteins , Fc-binding proteins
Factors that destroy host tissues - Cytotoxins, Collagenase, Bone resorption agents , Stimulators of inflammatory mediators
Factors that inhibit host repair of tissues - Inhibitors of fibroblast proliferation , Inhibitors of bone formation

phenotype including elevated levels of PGE2 and IL – 1 N38;.

5) Progression to the attachment loss and bone loss may be self arresting.

Actinobacillus actinomy cetemcomit anshas an arsenal of virulence factors that attack the host and compromise the periodontium. Because this periodontal pathogen has a high virulence and destructive nature, the dental practitioner needs to employ systemic antibiotics in addition to surgical and/or nonsurgical therapy.

The American Academy of Periodontology has developed the following parameter on treatment of Aggressive periodontitis. Patients should be informed of the disease process, therapeutic alternatives, potential complications, expected results, and their responsibility in treatment. Consequences of no treatment should be explained. Failure to treat aggressive periodontitis appropriately can result in progressive and often lead to rapid loss of periodontal supporting tissues. This has an adverse effect on prognosis and could result in tooth loss.

Therapeautic Goals -

The goals of periodontal therapy are to alter or eliminate the microbial etiology and contributing risk factors for periodontitis, thereby arresting the progression of disease and preserving the dentition in comfort , function , and appropriate esthetics and to prevent the recurrence of disease. In addition , regeneration of periodontal attachment apparatus, where indicated may be attempted. Due to complexity of aggressive periodontal diseases with regard to systemic factors , immune defects, and the microbial flora , control of diseases may not be possible in all instances. In such cases , a reasonable treatment objective is to slow the progression of the disease.

Trearment Modalities – (Considerations)

Once aggressive periodontal disease has been diagnosed, a comprehensive periodontal treatment plan must be developed. The treatment of periodontal diseases may be divided into four phases: systemic, hygienic, corrective, and maintenance / supportive therapy. Pihlstrom described the systemic phase as the appropriate consideration of systemic diseases and their impact on the etiology or treatment of the disease. The focus of therapy in the hygienic phase is to eliminate as many of the local factors of periodontal disease (bacterial plaque and calculus) as possible.[8] The corrective phase focuses on procedures designed to correct the effects of periodontal disease.[9] In the maintenance/supportive phase, recall and therapy outcomes are assessed. Systemic antibiotics are employed in the hygienic and/or corrective phases. As treatment progresses through the four phases, the dentist uses both surgical and nonsurgical therapy to remove biofilm created by the bacterial pathogens; this procedure is in agreement with good medical practice because the bacterial load should be reduced as much as possible prior to the use of antibiotics. Systemic antibiotics are considered only for those who exhibit continued loss of periodontal attachment despite conventional mechanical periodontal therapy.

In addition to the parameter for chronic periodontitis , the following should be considered for patients who have aggressive periodontitis:

1) A general medical evaluation may determine if systemic disease is present in children and young adults who exhibit severe periodontitis , particularly if aggressive periodontitis appears to be resistant to therapy. Consultation with the patient's physician may be indicated. Modification of environmental risk factors should be considered.
2) Initial periodontal therapy alone is often ineffective. However , in the early stages of disease, lesions may be treated with adjunctive antimicrobial therapy combined with scaling and root planning with or without surgical therapy.[3]
3) The long-term outcome may depend upon patient compliance and delivery of periodontal maintenance at appropriate intervals, as determined by the clinician . If primary teeth are affected, eruption of permanent teeth should be monitored to detect possible attachment loss.
4) Due to the potential familial nature of aggressive diseases, evaluation and counselling of family members may be indicated.

Systemic Versus Topical Antibiotic Use:

There are several advantages to using systemic antibiotics instead of topical ones. Systemic antibiotics reach the periodontal pathogens via serum at the base of deep pockets, in furcation areas, and within gingival epithelial and connective tissues. The antibiotic's diffusion into the connective tissue and epithelium is important because Aactinobacillus actinomycetemcomitans invades those areas where topical agents are less effective at achieving high concentrations, however, topical agents can achieve higher gingival crevicular fluid concentration than systemic agents.[10] Systemic antibiotics also inhibit periodontal pathogens from colonizing other periodontal sites. Disadvantages include adverse drug reactions and uncertain patient compliance in following the prescribed antibiotic regimen.

Antibiotic Selection:

Once a patient has been diagnosed with periodontitis, treated and evaluated appropriately, and has not responded favourably to conventional therapy , the adjunctive use of an antibiotic[11] may be indicated.

Most periodontal investigators and many practitioners believe that to obtain the best results in the treatment of Actinobacillus actinomycetamcomitans associated aggressive periodontitis, whether localized or generalized , the use of an adjunctive antibiotic along with mechanical therapy is necessary. A likely reason for this may be ability of Actinobacillus actinomycetamcomitans to penetrate the epithelial surface of the pocket. Widespread disease may necessiciate the need to incorporate systemic antibiotics into the treatment plan. Culture and sensitivity testing are strongly recommended to select the antibiotic regimen that will be the most efficacious . when culture and sensitivity testing are not feasible , the practitioner has to make the choice of antibiotic based

Suggested Oral Antibiotic Dosages (Walker C< Karpinia K 2002)[12]:

Generic Name	Usual Adult Dosage	Length Of Treatment	Maximum Child Dosage	Dosage Suggestions
Amoxicillin / clavulanic acid	250 or 500 mg . tid	10 days	Weight < 20kg : 20 -40 mg / kg in divided doses , tid	Given without regard to meals (given with food helps eliminate some of the stomach distress)
Amoxicillin plus metronidazole	375 mg amoxicillin , tid, plus 250 gm metronidazole , tid	7 days	Not recommended for children under 16 years of age	Given without regard to meals
Clindamycin hydrochloride	1560- 300 mg. qid	10 days	8 – 12 mg / kg in 3 – 4 equally divided doses	Give without regard to meals (given with food helps eliminate some of the stomach distress)
Doxycycline hyclate	100 mg bid first day followed	10 – 14 days	Age > 8 years : 4 mg / kg divided	Given 1 hour before or 2 hours
	by 100 mg a day either as single dose or 50 mg , bid		into equal dose , bid , on 1st day ; followed by 2 mg/kg as single dose or divided into equal doses , bid	after meal
Metronidazole	250 mg . tid or qid	10 days	Not recommended for children under 16 years of age	Given without regard to meals
Minocycline hydrochloride	200 mg bid first day followed by 100 mg. bid	10- 14 days	Age > 8 years : 4 mg/ kg divided into equal doses bid on first day : followed by 2 mg/ kg , bid	Given 1 hour before or 2 hours after meal
Tetracycline hydrochloride	250 mg . qid	14 – 21 days	Age > 8 years : 25 – 50 mg / kg in equal doses, bid	Given hour before or 2 hours after meal

on patient presentation and history.[12]

In case of classical Actinobacillus actinomycetamcomitans associated aggressive periodontitis , the practitioner might choose tetracycline HCL , or one of its derivatives (doxycycline hyclate or minocycline hydrochloride), in conjunction with conventional therapy. If a favourable response is not obtained with tetracycline or if the disease appears to be particularly aggressive the combination of amoxicillin and metronidazole would be suggested.

Very good results have been reported in treatment of aggressive periodontitis cases using the combination of metronidazole plus amoxicillin. Van Winkelhoff et al.[13] have reported that use of metronidazole (250 mg, tid) and amoxicillin (375 mg , tid) , simultaneously administered for a period of 10 days following periodontal scaling and root planning, eliminated Aactinobacillus actinomycetamcomitans in 97 % or more of the patients and resulted in improved clinical status. The oral dosages, treatment regimens, and absorption suggestions for the administration for the administration of systemic antibiotics are summarized in the following table :

The decision to incorporate adjunctive therapy into the treatment protocol for aggressive periodontitis should be based on accurate scientific knowledge and sound clinical judgement. The need for an adjunctive antibiotic should be firmly established in the clinician's mind as well as the expected outcome of the therapy.[12].

Out Come Assessment:

The desired outcome of periodontal therapy is:

1) Significant reduction of clinical signs of gingival inflammation;
2) Reduction of probing depths;
3) Stabilization or gain of clinical attachment;
4) Radiographic evidence of resolution of osseous lesions;
5) Progress toward occlusal stability ;
6) Progress toward the reduction of clinically detectable plaque to a level compatible with periodontal health.

Recent Treatment Advancements For Aggressive Periodontitis:

It is generally accepted that mechanical removal of contaminants and adjunctive use of antibiotics and disinfectants make up the conventional treatment for aggressive periodontitis. Furthermore, the clinician is informed that biofilm structure of dental plaque confers remarkable resistance to species within the biofilm. Also there is an increased concern regarding the development of antibiotic resistance. Because of variability in design of existing studies it has not yet been concluded which antimicrobial agent, dose , and duration provide the optimal clinical and microbiologic effect in this group of patients. For these reasons alternatives that might offer the possibility of efficient removal of bacteria from hard tissue surfaces are being sought.

Laser irradiation of subgingival sites to eradicate periodontopathic microorganisms is also being considered in the nonsurgical therapy of periodontitis patients. Diode laser treatment has shown a superior clinical and microbiological effect when used along with SRP, compared to SRP alone or laser therapy alone in aggressive periodontitis patients [1],[14].

PDT is based on the principle that a photo-activable substance, the photosensitizer, binds to the target cell and can be activated by light of a suitable wavelength.[15] During this process, free radicals of singlet oxygen are formed, which produce an effect that is toxic to the cell. This so-called ''photodynamic action'' was described as a process in which light, after being absorbed by dyes, sensitizes organisms for visible light-inducing cell damage. Although PDT is more widely known for its application in the treatment of neoplasms, there is also an interest in antimicrobial photodynamic therapy because a large number of microorganisms (including oral species) have been reported to be killed in vitro by this approach. Potential of some key virulence factors (lipopolysaccharide and proteases) have also been shown to be reduced by photosensitization. The bactericidal efficacy of PDT against periodontal pathogens has been shown in a study using a rat model, and the results show that toluidine blue–mediated lethal photosensitization of Porphyromonas gingivalis is possible in vivo and that this results in decreased bone loss Sigush et al[16] showed that PDT using a photosensitizer and a 662-nm laser light source are advantageous in reducing the periodontal signs of redness and bleeding on probing (BOP) in dogs. Histologic examination of the periodontal tissues of the rats after PDT showed no adverse effects , even with highest light doses and toluidine blue concentration used[17]. Development of resistance to PDT seems to be unlikely because its bactericidal activity is caused by singlet oxygen and other reactive species such as hydroxyl

radicals, which affect a range of cellular targets. Photosensitization may be important in dealing with aggressive periodontitis because the photosensitizer is capable of penetrating through the epithelium and connective tissues. Considering that PDT is not truly a mechanical therapy , residual presence of calculus , residual calculus is expected to occur. Because of the lower presence of calculus , aggressive forms of disease and patients are more likely to benefit from the antimicrobial effect.

Another new remedy for treatment of aggressive periodontitis being tried is use of ozonized solutions (Sorokina and Zaslavskaja, 1997). The effect of ozonized water on oral microorganisms and dental plaque was studied by Nagayoshi et al. They found that ozonized water should be useful in reducing the infections caused by oral microorganisms in dental plaque. Ozone was found to have a potent antibacterial effect explained by the fact that it causes disruption of the envelope integrity through peroxidation of phospholipids. Nagayoshi et al. found that ozonized water should be useful in reducing the infections caused by oral microorganisms in dental plaque. Concerning the results obtained by Agapov et al. ozone can cause stimulation of body's own defenses which is in a good agreement with the present results of this study and in good conformity with the results obtained by Lukinykh and Kosiuga who studied the efficacy of hygienic treatment of the oral cavity in combination with ozone therapy. They proved that this combination mechanically removed soft dental deposits and also decreased bacterial contamination[5].

Surgical Therapyessentially consists of open flap debridement either alone or as a combination with regenerative procedures. The main aim of a flap procedure is to get access and visibility to root and furcation areas so that a thorough instrumentation and debridement can be performed. Flap techniques like modified Widman flap[18], modified flap operation/Kirkland flap (sulcular incision flap)[19], [1] achieve this aim without eliminating the pockets.

A sulcular incision flap or papilla preservation flap will be the ideal technique to minimize recession in the anterior regions due to esthetic reasons, and Modified Widman flap or conventional/sulcular incision flap will be the technique of choice in the posterior regions when opting for bone grafting and another regenerative therapy. A papilla preservation flap is preferred for bone grafting when there is spacing between the teeth to obtain maximum coverage of the graft material at the interdental region and to prevent shrinkage of papilla on healing.

Biomodification of the root surface (Root conditioning) with citric acid, tetracycline, or fibronectin is preferable when performing bone grafting or GTR for better clinical results[20].

Use of biologic mediators like growth factors (insulin-like growth factor (ILGF), platelet-derived growth factor (PDGF)) use of platelet-rich plasma which contains PDGF, extracellular matrix proteins like emdogain, etc. are of promising results. Application of enamel matrix proteins alone[1] or in combination with bone grafts including bioactive glass has shown to result in the successful treatment of intrabony defects in aggressive periodontitis .

The importance of supportive periodontal therapy has to be stressed in management of aggressive periodontitis. Regular SPT was found to be effective in maintaining clinical and microbiological improvements attained after active periodontal therapy in early onset periodontitis. The maintenance therapy starts soon after the phase I therapy or nonsurgical therapy and should be continued throughout the lifetime of the patient.

Cosmetic concerns in young aggressive periodontitis patients will be high since the disease can result in flaring, protrusion, pathologic migration, and even extrusion of the anterior teeth. Malocclusion, pathologic migration and potential occlusal traumatism which can cause secondary trauma from occlusion can be corrected by orthodontic therapy in generalized aggressive periodontitis patients already stabilized by periodontal therapy. Orthodontic treatment can be commenced once attachment gain and bone stability is achieved after periodontal therapy but is generally advised to postpone till 3 months to 1 year after active periodontal therapy.

Psychotherapy has to be started immediately following the first appointment and should be continued concomitantly for total rehabilitation of the patient for a variable duration depending upon the psychologic status of the individual patient. A recent study reported that psychotherapy offered at 3 levels (individual, group, and conjoint family psychotherapy) to Generalized aggressive periodontitis patients gave positive psychologic effects that restored their ability to socialize in their environment contributing to their positive experience in life.

Host modulation therapy with systemically and locally administered agents is under research for therapy in aggressive periodontitis. Adjunctive use of locally administered alendronate gel with SRP for host modulation has shown promising results in aggressive periodontitis.

With further understanding of the genetic risk factors, a futuristic application of genetic screening tests will be in identifying the susceptible individuals and instituting the preventive measures to keep the gene expression and thus the disease under control[1].

Conclusion:

Management of aggressive periodontitis is more challenging because of its strong genetic predisposition as an unmodifiable risk factor. The key to successful management at present lies in early diagnosis of the disease and rigorous treatment employing the different treatment modalities mentioned along with systemic antibiotic therapy followed by meticulous lifelong maintenance therapy. With the current treatment modalities, successful long-term maintenance of the dentition in a healthy and functional state can be achieved. A comprehensive periodontal treatment consisting of mechanical/surgical and systemic antimicrobial therapy is found to be an appropriate treatment regimen for long-term stabilization of periodontal health with arrest of periodontal disease progression in 95% of the initially compromised lesions. Further understanding of the etiology, risk factors, pathogenesis, and host immune response in aggressive periodontitis along with advances in regenerative concepts, tissue engineering, and gene

therapy is needed for formulating better management protocols in the treatment of generalized aggressive periodontitis.

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