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RESEARCH ARTICLE

LOCALIZED AGGRESSIVE PERIODONTITIS: CASE DEFINITION, DIAGNOSTIC CRITERIA AND TREATMENT OPTIONS

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ABSTRACT

The purpose of this research is to highlight the current etiological and therapeutic concepts of aggressive periodontitis which is rapidly progressing and aggressive in nature with photographic depiction. It leads to destruction of periodontal tissues and loss of teeth. The need in present time is requirement of advanced diagnostic techniques to learn about current disease activity and rate of progression. It also requires strategies to keep the disease under control with proper maintenance regime and prevent tooth loss, because it can result into complicated prosthetic rehabilitation in a very young patient. The evidence suggests that aggressive periodontitis is influenced by microbiological, genetic, and host factors. This paper presents clinical, microbiological, immunological, and genetic aspects of pathogenesis of aggressive periodontitis, as well as diagnostic criteria of the disease and appropriate nonsurgical and surgical treatment options.

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INTRODUCTION

Aggressive periodontitis, first described in 1923 as "diffuse atrophy of the alveolar bone" (Gottlieb, 1923), has undergone a series of terminology changes over the years to be finally named as "aggressive periodontitis" in 1999 (Gottlieb, 1923 and Guzeldemir, 2006). The disease which includes both localized and generalized forms was previously known as "early onset periodontitis" which included the three categories of periodontitis—prepubertal, juvenile, and rapidly progressing periodontitis (Ranney, 1993 and Caton, ?). It is interesting that the first ever reported detailed description of a recognized disease in early hominid evolution is a case of prepubertal periodontitis in an 2.5–3-million-year-old fossil remains of a juvenile Australopithecus africanus specimen which showed the typical pattern of alveolar bone destruction with migration of the affected deciduous molars (Ripamonti, 1988 and Ripamonti, 1989). Black in the year 1886 (Black, 1886), used the terms phagedenic pericementitis and chronic suppurative pericementitis to describe patients who suffered from a rapid destruction of alveolar bone. 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.

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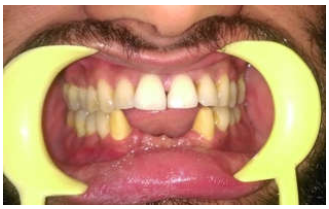
Based on histological observations on extracted teeth from affected sites, he believed that the disease was due to defective deposition of cementum or cementopathia. In the year 1942, Orban and Weinmann introduced the term periodontosis to describe the periodontal destruction in young individuals (Orban, 1999). Generalized aggressive periodontitis (GAgP) is characterized by "generalized interproximal attachment loss affecting at least 3 permanent teeth other than first molars and incisors" (Lang, 1999).

It is a multifactorial disease where interplay of microbiologic, genetic, immunologic, and environmental/behavioral risk factors decides the onset, course, and severity. Pathogenic bacteria in the dental plaque especially Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis (Schacher, 2007 and Armitage, 2000), have an indispensable role which elicits an aggravated host response which in turn is determined by the genetic and immunologic profile of the patient modified by environmental risk factors like smoking. This paper attempts to describe the diagnostic features, etiological agents along with the periodontal management options of generalized aggressive periodontitis with the help of a case daignosed on clinical features and OPG as localized Aggressive periodontitis. Finally an attempt to summarize the available protocol for a comprehensive management of Aggressive periodontitis is done.

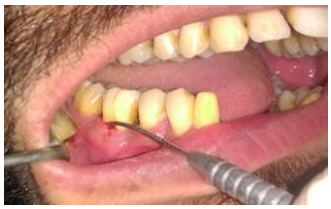
Classification

- Localized aggressive periodontitis (LAP): Localized to first molar/incisor interproximal attachment loss
- Generalized aggressive periodontitis (GAP): Generalized interproximal attachment loss affecting at least three permanent teeth other than incisors and first molar

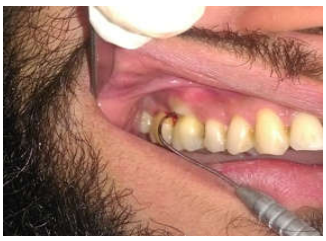
Clinical and Radiographic features



Preoperative Loss of lower central incisors due o mobility

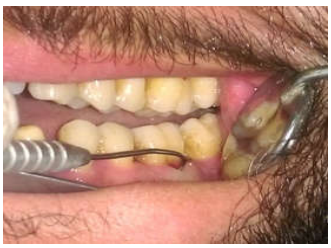


Grade III furcation involvement diagnosed with nabers probe in 46.

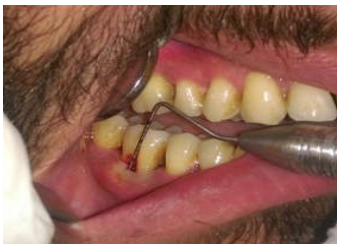


Grade III furcation involvement in 16 diagnosed with nabers probe

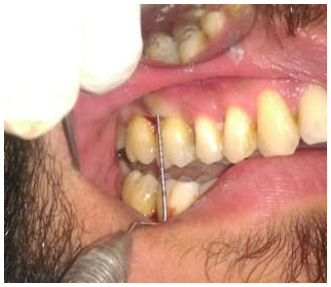
Grade IV furcation involvement in 26 diagnosed with nabers probe



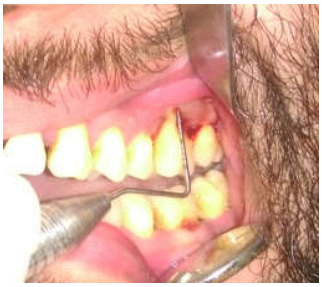
Grade III Furcation involvement In 36 diagnosed with nabers probe



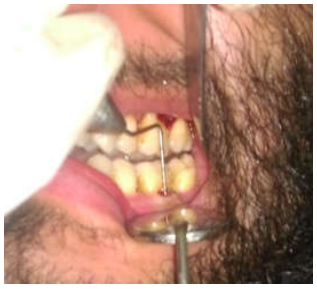
Probing pocket depth measurement In 36 diagnosed with nabers probe



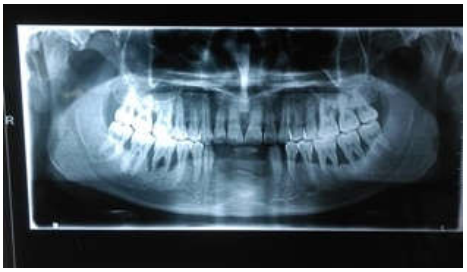
Probing pocket depth measurement Probing pocket depth measurement



Probing pocket depth measurement with UNC-15 probe at 26



Probing pocket depth measurement With UNC-15 probe at 36



OPG

The patient depicted on photographs first reported mild spacing in relation to 31,32,41,42 about 1 year before, after which he noticed it to be gradually increasing and associated with intermittent episodes of pus discharge which subsided on taking antibiotics as per advice at a local hospital, later on there was mobility for which he has to undergo extraction at local hospital. Family history of similar complaints or early tooth loss could not be found. The patient was systemically healthy with no relevant medical history. There were no abnormalities detected in extra oral examination except for slightly tender and palpable submandibular lymph nodes. Patient had undergone extraction of lower incisors (3, 32, 41, 42). The oral hygiene status of the patient was average showed by oral hygiene index. There was minimal amount of calculus and plaque. Deep pockets were found in incisor 11, 12, 21, 22, 16, 26, 36, 46. Slight flaring and labial version was found in upper anteriors. Gingival examination revealed normal color except for the labial aspect of 16, 26 where it was slightly reddish. The margins were of knife-edge contour except for the buccal aspect of 16, 26, 36 where it was bluntly rounded. The gingiva was firm and resilient except in the region on 16, 26, 36, 46 where it was soft in consistency. There was no loss of stippling in the anterior regions of upper jaw. The position of the gingival margin was apical to the CEJ in the labial aspect of 11.

There was generalized bleeding on probing, and exudation was present on the labial aspect of 26. All together there were minimal signs of inflammation other than bleeding on probing. An OPG and full-mouth IOPA X-ray were performed which revealed the generalized distribution of alveolar bone loss which was a combination of both horizontal and vertical bone especially in molars. Based on the history, examination findings, and the radiographic findings, a diagnosis of localized aggressive periodontitis was made according to the criteria by AAP 1999 classification. LAP starts at circumpubertal age, involving interproximal attachment loss of first molar, and or incisors, there will be lack of inflammation with presence of deep periodontal pocket and advanced bone loss. Amount of plaque is minimal which is inconsistent with the amount of destruction, and rarely mineralizes to form calculus, but the plaque is highly pathogenic due to the presence of elevated levels of bacteria like Aggregatibacter actinomycetumcomitans (A.a) and Porphyromonas gingivalis (P.g). Secondary clinical features like distolabial migration of incisors with diastema formation, mobility of the involved teeth, sensitivity of the denuded root, deep dull radiating pain to the jaw, and periodontal abscess lymph node enlargement may occur (Novak, 2006). GAP has generalized interproximal attachment loss affecting at least three permanent teeth other than incisors and first molar involving individuals under age 30 with destruction appears to occur episodically. There will be presence of minimal plaque which is inconsistent with

destruction and presence of bacteria like P.g, A.a, and Tannerella forsythia are detected in plaque (Novak, 2006). Two kinds of gingival responses are seen in Aggressive periodontitis (AP) patients. First response is severe acutely inflamed tissue which is ulcerated and red in color with spontaneous bleeding indicating destructive stage and the other one with pink gingiva free of inflammation, with some degree of stippling and deep periodontal pockets are present representing quiescence stage (Novak, 2006). A contemporary case definition of aggressive periodontitis is presented by Albandar in 2014[13]. Key diagnostic criteria of this disease include: Early age of onset, Involvement of multiple teeth with a distinctive pattern of clinical attachment loss and radiographic bone loss, A relatively high rate of disease progression and the absence of systemic diseases that compromise the host's response to infection. Although in some patients the disease may start before puberty, in most patients the age of onset is during, or somewhat after, the circumpubertal period.

A typical patient shows disease onset at an early age (i.e., before 25 years of age), although identification of the affected patient usually occurs after disease commencement. Initially, the periodontal lesions show a distinctive pattern, depicted radiographically as vertical bone loss at the proximal surfaces of posterior teeth, and the bone loss usually occurs bilaterally. In advanced cases of aggressive periodontitis the periodontal lesions may be depicted radiographically as a horizontal loss of bone. The primary teeth may also be affected, although early exfoliation of these teeth is not common. Aggressive periodontitis may be localized or generalized, in localized aggressive periodontitis (LAP), tissue loss usually starts at the permanent first molars and incisors, and with increasing patient age the disease may progress to involve the adjacent teeth. The generalized form of aggressive periodontitis involves most or all of the permanent teeth.Localized aggressive periodontitis typically presents "arc-shaped" mirror image radiolucency in the first molars starting from the distal aspect of second premolars to the mesial aspect of the second molar. In generalized aggressive periodontitis, radiographs may show generalized bone destruction ranging from mild crestal bone resorption to severe extensive alveolar bone destruction depending on the severity of the disease. The defects may be a combination of vertical and horizontal defects.

Diagnosis: Early diagnosis is of utmost importance for the prevention of extensive attachment loss and bone loss experienced in aggressive periodontitis. Diagnosis is made according to the criteria set by the American Academy of periodontology 1999 classification of periodontal diseases and conditions, using history, clinical features, and radiographic features aided by microbial examination if needed. Family history may reveal a history of early tooth loss in the parents or immediate blood relatives of the patient. The amount of microbial deposits will be inconsistent with the amount of destruction when compared to chronic periodontitis and plaque will be minimal. Comparison of serial radiographs helps in assessing the rapid rate of bone destruction and can aid in the diagnosis of the disease.

Microbiology: Microbiology has renovated and improved clinical application of various antibiotics specific to particular disease causing microorganisms. Since long time A.a has been considered the primary pathogen for aggressive periodontitis, especially in its localized form. Six serotypes of A.a (a, b, c, d, e, and f) are described based on the composition of O polysaccharide of their lipopolysaccharide and there are phenotypically nonserotypeable strains of A.a which lack expression of serotype-specific polysaccharideantigen (Kononen, 2000). A highly leukotoxic clonal type of A. A serotype b was first isolated, in the early 1980s, from an 8-year-old male child with localized aggressive periodontitis. Prevalence of A.a in LAP varies from 70 to 90%, (Asikainen, 1986 and Elamin, 2011). but there are studies which states there is no association between A.a and the periodontal disease rather prevalence of levels of P.g, T.denticola, and P.intermedia are significantly associated with aggressive periodontitis. In a study done by Takeuchi for detection of microorganisms in sub gingival flora of Japanese population using polymerase chain reaction (PCR) it was found that the prevalence of A.a was less in patients with LAP whereas elevated levels of P.g, Tannerella forsythia, T.denticola, P.intermedia, and Campylobacterrectus was detected. Albander found elevated levels of IgG and IgA to P.g and A.a and IgA to P.intermedia in subjects with GAP than LAP and no difference was found at the antibody levels of C.rectus, E. corridens, F.nucleatum. Filifactor alocis in gram positive anaerobic rod which has the potential of being periodontal pathogen and the levels of these bacteria is elevated in aggressive periodontitis patients. Treponema lecithinolyticum and Treponema socranskii are elevated in GAP. Sulfate reducing bacteria, Desulfomicrobium orale, has been suggested to be involved in various categories of periodontal destruction, possibly synergistically with the red complex periodontal pathogens. Yamabe suggested Archaea a methanogenic organism, especially Methanobrevibacter oralis as putative periodontal pathogen for aggressive periodontitis.

Genetics: A strong association was found between interleukin (IL)-1a (889) (Kornman, 1992) and IL-1a 3954 allele 2 polymorphism and aggressive periodontitis. IL-1 gene cluster was not associated with AP according to Fiebig in caucasians. IL-4-590 T/T, IL-4-34 T/T genotype are associated with AP. IL-6-174G allele increased the risk of AP and IL-6-572 C/G polymorphism is associated with pathogenesis. Nibali found link between IL-6-1363, 1480 polymorphism and LAP susceptability. IL-10 promoter polymorphisms at positions 1082 G-A, 819C-T, and 590C-A showed that haplotype ATA is a putative risk indicator for GAP. FPR348 T-C gene polymorphism showed association with AP in African American subjects. Fc gamma RIIIb-NA2 allele and Fc gamma RIIIb-NA2/NA2 genotype and composite genotype FcaRIIIb-NA2/NA2 and FC gamma RIIIa-H/H131 may be associated with GAP. FC gamma polymorphisms can lead to modulation of neutrophil superoxide production and predispose to AP. VDR, FcaRIIIb composite genotype may be associated with susceptibility to generalized early onset periodontitis. TLR-4 399 Ile polymorphism showed a protective effect against AP. TNFA gene polymorphism (1031, 863,857, 308, and 238) was not associated with aggressive periodontitis. HLA-DR4, HLA-A9, B-15 are found in high frequency in rapidly progressive periodontitis patients and HLA-DQB1 plays a crucial role in pathogenesis of AP (Kaĵ, 1987 and Shapira, 1994).

DISCUSSION

The key to successful treatment is early diagnosis. Early diagnosis helps in prevention of progression of the disease thus avoiding the possibility of advanced tissue destruction and

alveolar bone loss. The earlier the diagnosis is the better the prognosis of the dentition will be. Furthermore since it has a tendency for familial aggregation, it is important to do a periodontal examination of siblings and other close blood relatives of the patient which helps in early diagnosis of the disease in the family members. Management of GAgP patients essentially consists of a nonsurgical phase, surgical therapy an interdisciplinary therapy and a lifelong supportive periodontal therapy.

Nonsurgical/Etiotropic Phase of Therapy: Nonsurgical therapy remains the first line of antimicrobial therapy in localized and generalized GAgP. Early stages of the disease with mild to moderate periodontal and bone destruction may be managed entirely by nonsurgical therapy with systemic antibiotics as an adjuvant to mechanical therapy. Therapy should start with attempts at controlling or eliminating the etiologic agents and modifiable risk factors for the disease. The disease has a strong genetic predisposition. The host response of the patient or the susceptible individual to pathogenic bacteria in the dental plaque plays a vital role in the pathogenesis and expression of the disease, and this host response is genetically determined and is an unmodifiable risk factor is at present by the current treatment measures. However, since the expression of the disease in susceptible individuals is also influenced by microbial and environmental risk factors, the disease can be successfully kept under control by controlling the microbial and environmental factors. This underlies the importance of optimal plaque control both by personally employed methods used by the patient himself and professionally employed plaque control measures by the dental team to the patient. Even a minimal amount of plaque is enough to elicit untoward host response in those patients susceptible to the disease, and a reduced resistance to the invasion of subgingival plaque can be compensated for by a correspondingly strong emphasis on total plaque control.

Mechanical plaque control can be successfully achieved by educating and motivating the patient if needed with the aid of disclosing solutions regarding the need for optimal plaque control, demonstration of brushing techniques (modified Bass technique for patients without gingival recession and modified Stillman technique in patients with hypersensitivity and generalized recession), and use of interdental cleansing aids like dental floss and interdental brushes where indicated. This behavioral modification from the patient needs a positive reinforcement and encouragement from the dental team. Regular recall appointments to monitor the efficacy of the patient's plaque control measures are essential. Chemical plaque control agents like chlorhexidine 0.12% or 0.2% mouthwashes, and 1% povidone iodine can be advised for further plaque control as an adjunct to the patient's mechanical plaque control measures. Additionally use of fluoride mouthwashes is advised to help in remineralization of the exposed root surfaces, and for patients complaining of hypersensitivity, use of desensitizing toothpastes and mouthwashes is mandatory. Smoking has been well documented as a significant risk factor for aggressive periodontitis with GAgP patients who smoke having more affected teeth and more loss of clinical attachment than nonsmoking patients with GAgP. Furthermore the response to periodontal therapy, both nonsurgical and surgical, regenerative therapy, and implant therapy is less than in nonsmokers, but former smokers respond similar to nonsmokers. This underlies the therapeutic effect of smoking cessation and cessation of other forms of tobacco, and patients should be advised of the benefits of smoking cessation and the potential risks of smoking in worsening their periodontal condition, and if needed expert counsseling for cessation of the habit should be sought.

Mechanical Antimicrobial Therapy: Scaling and root planing (SRP) which eliminates the microbial bacterial load from the periodontal pockets and removes the local etiologic factors is performed either as a quadrant-wise SRP at 2-week interval or as a full mouth scaling and root planning completed on the same day. However, both modalities have been found to be efficacious with significant improvement in clinical parameters, and the clinician should select the treatment modality based on the practical considerations related to the patient preference and clinical workload. Another approach to mechanical antimicrobial therapy is a one-stage full mouth disinfection therapy devised by Quirynen et al, which was found to result in an improved clinical outcome and microbial improvement in early onset periodontitis compared to quadrant-wise SRP. Full-mouth disinfection therapy includes full-mouth debridement (scaling and root planning, brushing of the tongue with 1% chlorhexidine for 1 minute, rinsing of the mouth with a 0.2% chlorhexidine solution for 2 minutes, and irrigation of periodontal pockets with 1% chlorhexidine solution), completed in 2 appointments within a 24-hour period.

Photodynamic Therapy and Laser Irradiation: These have been tried as adjuncts to mechanical therapy to inhibit the pathogenic bacteria in periodontal pockets. Photodynamic therapy (PDT) is a noninvasive photochemical approach for infection control which combines the application of a nontoxic chemical agent or photosensitizer with low-level light energy and has shown clinical evidence of efficient eradication of periodontal bacteria from subgingival sites. This novel therapeutic approach of antimicrobial therapy seems promising and is getting attention recently either as a monotherapy or as an adjunct to SRP in the nonsurgical treatment of aggressive periodontitis. Both PDT and SRP have been shown to have similar clinical results in the nonsurgical treatment of aggressive periodontitis. 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. A regular recall visit preferably at one-week intervals should be performed especially at the initial stages of the treatment to monitor the efficiency of the patient's plaque control measures and to assess the response of the patient towards nonsurgical therapy.

Systemic antibiotics are indicated in aggressive periodontitis since the pathogenic bacteria like Aggregatibacter actinomycetem-comitans and Porphyromonas gingivalis have been found to be tissue invasive and mechanical therapy is insufficient to eliminate the bacteria from these sites. Systemically administered antibiotics with or without scaling and root planning and or surgery provided greater clinical improvement in attachment level change compared to similar periodontal therapy without antibiotics. Earlier tetracyclines were used extensively for this purpose since systemic tetracycline was found to be a useful adjunct to mechanical periodontal therapy in patients with aggressive periodontitis,

but the concern for tetracycline resistance has shifted the focus to the use of other antibiotics both as combination therapy or serial antibiotic therapy. The preferred combination antibiotic therapy at present for treatment of GAgP is 250 mg of amoxicillin thrice daily along with metronidazole 250 mg twice daily for 8 days. It is one of the most evaluated drug combinations in GAgP, and there is ample evidence now to show that Amoxycillin-Metronidazole combination as an adjunctive treatment in GAgP at initial therapy significantly improves the results and hence should be preferred over other antibiotic regimens as the first-line treatment. The usefulness of microbial testing may be limited because of the variability of test reports between different labs and the mixed flora, and hence an empiric use of antibiotics like the above-mentioned combination may be more clinically sound and cost-effective than bacterial identification and antibiotic-sensitivity testing in the treatment of aggressive periodontitis. Single-agent therapy with Doxycycline, azithromycin, metronidazole, and clindamycin is effective when used adjunctively to nonsurgical procedure of SRP in AgP patients. The criteria for selection of antibiotics are not clear in AgP; the choice depends on the case, disease-related factors and patient-related factors like compliance, allergies, and potential side effects.

Topical application of antimicrobial agents and local drug delivery is also a treatment option especially if there are localized areas of exudation and deep pockets not responding adequately to mechanical and systemic antibiotic therapy. Local drug delivery delivers the drugs at high concentrations at the site of infection when compared to systemic antibiotic therapy. Furthermore, this is an option in patients where there is intolerance to systemic administration of the antibiotic. Several local anti-infective agents combined with SRP appear to provide additional benefits in PD reduction and CAL gain compared to SRP alone. Over the past 20 years, locally delivered, anti-infective pharmacological agents, most recently employing sustained-release vehicles, have been introduced to achieve this goal. Though there is more evidence on its application in chronic periodontitis, till future researches are available; the same agents can be employed in aggressive periodontitis patients as well empirically. Adjunctive use of LDD agents like controlled release biodegradable chlorhexidine gluconate chip, tetracycline fibers, and minocycline-Hcl gel has been tried in aggressive periodontitis with superior clinical outcomes. The decision to use local anti-infective adjunctive therapy remains a matter of individual clinical judgment, the phase of treatment, and the patient's status and preferences. An evaluation of the response to nonsurgical treatment is done 2-3 weeks after treatment during which the gingival and periodontal status of the patient will be reevaluated and compared with the pretreatment values to assess the response to therapy and to assess the areas which need surgical therapy. Sites with persisting pockets >5 mm depth, vertical bone defects which need regenerative therapy, difficult to instrument areas like furcation involvement, and areas which need recontouring or resective osteoplasty are indications for surgery.

Surgical Therapy: It essentially consists of open flap debridement either alone or as a combination with resective or 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, modified flap operation/Kirkland flap (sulcular incision flap) achieve this aim without eliminating the pockets. A resective flap procedure like undisplaced flap will eliminate the pockets as well but compromise the esthetics and function of the dentition by root exposure and resultant hypersensitivity and hence is not preferred usually when compared to modified Widman flap or sulcular incision flap. Laser-assisted surgery (Nd: YAG laser) is suggested as a valid alternative to conventional scalpel surgical therapy, in individuals at increased surgical risk like in coagulation and platelet function disorders. Regeneration of the periodontal supporting structures lost due to periodontal disease so that the form and function of the periodontium is reestablished has been an elusive or difficult-to-achieve goal for periodontal therapists. Various modalities are being employed for periodontal regeneration which includes use of bone replacement grafts, barrier membranes or guided tissue regeneration (GTR), biologic modifiers like growth and differentiation factors (GDF), and extracellular matrix proteins like enamel matrix proteins (EMD) or use of a combination of the above techniques and materials which has been extensively reviewed elsewhere.

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. Bone grafting is indicated in vertical defects, and the success of the procedure depends on the type of defect. Three-walled or intrabony defect is the ideal defect for bone grafts and has a better success rate compared to a two-walled and one-walled defect. The type of bone graft which gives the maximum benefit with minimum tissue reaction is autograft, but there are limitations of obtaining it in large quantities as is needed in most cases of generalized aggressive periodontitis. A more feasible option is to use commercially available bone grafts, which are allograft, xenograft, or alloplastic materials.
Allografts used for periodontal grafts include mineralized freeze-dried bone allografts (FDBAs) which are osteoconductive, and decalcified freeze-dried bone allografts (DFDBAs) which are osteoinductive. Decalcification of the graft exposes the complex bone morphogenic proteins (BMPs) from its matrix which can induce osteoblastic proliferation in the recipient site.

DFDBA, because of its osteoinductive property, has shown to have better results than the alloplastic materials which are osteoconductive. Allogeneic freeze-dried bone (FDBA) mixed with tetracycline powder along with systemic tetracycline has demonstrated a better clinical outcome in treatment of juvenile periodontitis. Xenografts used are either bovine derived or coral derived. An osteoconductive bovine-derived anorganic bone, Bio-Oss, has been successfully used in periodontal defects with resulting bone regeneration and new attachment in these defects. Human histologic studies have shown that a combination of Bio-Oss with either purified porcine collagen (Bio-Oss Collagen) or a synthetic cell-binding polypeptide (Pepgen P-15) has the capacity of inducing regeneration of the periodontal attachment apparatus when placed in intrabony

defects. Coralline grafts implanted into human periodontal defects have produced better clinical results when compared to nongrafted sites. Synthetic grafts/alloplastic grafts have been considered primarily as defect fillers. The most commonly used among alloplastic graft materials is hydroxyapatite (HAP) which is osteoconductive and has shown to have similar clinical effect to FDBA. Other alloplastic grafts which can be used are beta tricalcium phosphate and bioactive glass. A synthetic hydroxyapatite/equine type I collagen/chondroitin sulphate biomaterial (Biostite) has been found to show comparable improvements to Bio-Oss in terms of clinical attachment gain, pocket depth reduction, and radiographic bone fill in the treatment of deep intraosseous defects.

Guided Tissue Regeneration: Guided tissue regeneration promotes regeneration by acting as a barrier which prevents apical migration of epithelium and exclude gingival connective tissue from the healing wound, thus allowing the pluripotent periodontal ligament cells to populate the site of healing enhancing new cementum and new attachment procedures. GTR has shown to have a greater effect on probing measures of periodontal treatment than open flap debridement alone, including improved attachment gain, reduced pocket depth, less increase in gingival recession, and more gain in hard tissue probing at reentry surgery. Research has shown that GTR in conjunction with bone grafting has better potential for regeneration compared with either technique alone, and this outcome has been confirmed in aggressive periodontitis also with the use of bioresorbable membranes (Bio-Gide).

Biologic Mediators and Extracellular Proteins: A wide array of regenerative materials is being considered for use in periodontitis. 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 or in combination with bone grafts including bioactive glass has shown to result in the successful treatment of intrabony defects in aggressive periodontitis. Beneficial effects of platelet-rich plasma, Platelet rich fibrin, Concentrated growth factor (PRP)/PRF/CGF in the treatment of periodontal defects have been demonstrated by clinical and radiographic measurements together with reentry results showing marked improvements from baseline with increased stabilization of whole dentition including the hopeless teeth. Various commercially available regenerative materials including bone replacement grafts, GTR membranes, enamel matrix derivatives, are in the market for use in periodontal therapy with varying results, and the choice of the material depends on the dentist's preference and experience with the products helping in clinical judgment of the therapeutic results of individual products and procedures and their cost-benefit ratio.

Role of Maintenance Therapy in Management of 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. Or in other words, “maintenance therapy never ends” for a GAgP patient. In order to maintain the optimal results got by surgery and to prevent the recurrence of the disease, a lifelong maintenance therapy is mandatory because of the strong genetic susceptibility of the individual to the disease. The frequency of the recall visits depends on the response of the individual to treatment and presence of other risk factors like environmental factors but generally will be more frequent than that in chronic periodontitis or in localized aggressive periodontitis. Any site which shows signs of recurrence of the disease like bleeding on probing which is considered as the first clinical sign of inflammation should be treated vigorously and monitored for resolution of the signs. A comprehensive management for total rehabilitation of the GAgP patients not only involves control of infection and arrest of progression and/or regenerative therapy by the periodontist but also incorporates a multidisciplinary approach to attend the esthetic, functional, and psychologic problems faced by the patient. An orthodontic therapy with concomitant periodontal monitoring and prosthetic rehabilitation, if possible with the use of implants and psychologic counseling, may be needed for patients with advanced forms of the disease. 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 GAgP 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. A combined periodontal and orthodontic treatment demands a detailed evaluation in both specialties, particularly when the periodontium is reduced. Periodontal evaluations are scheduled concomitantly with orthodontic appointments to monitor the periodontal stability as the tooth movement occurs.

Gingival recession with loss of interdental papilla especially in the anterior teeth is unaesthetic especially when the patient smiles and the feasibility of root coverage periodontal plastic surgery will be limited in generalized aggressive periodontitis because of the large number of teeth involved and the advanced interdental bone loss. A porcelain, resin, silicone, or copolyamide removable gingival prosthesis (gum veneer/gingival mask) can be fabricated to mask the recession and improve the appearance of the anterior teeth. The restoration of the teeth lost due to periodontitis should be done with fixed or removable prosthesis depending on the bone support of the remaining teeth. Contradictory to the earlier concept that implants are not a feasible option in AgP patients, the use of implants and implant-supported prosthesis to restore the lost teeth is increasingly considered as a treatment option in well-maintained AgP patients even though the risk of bone loss and attachment loss around implants is higher than that in chronic periodontitis patients or periodontally healthy individuals, with researches showing good survival of implants over a 10-year period. Several reports are there which have successfully used osseointegrated implants in oral rehabilitation of partially edentulous patients treated for AgP.

Perhaps the least recognized and the most underestimated aspect in the total rehabilitation of a patient with AgP presenting with multiple tooth loss and/or advanced periodontal destruction necessitating extraction of multiple teeth is the need for psychological counseling and psychotherapy. It aims at attending the psychologic effect and potential mental depression following tooth loss due to rapid

periodontal destruction which provides the patient with relatively less time to cope with the situation. The emotional effects of tooth loss are devastating for some patients and have a dramatic impact on their life, and they take longer time to come to terms with the tooth loss. Preparing the patients with advanced disease having multiple teeth with hopeless prognosis emotionally for extraction also has to be dealt with carefully by the dentist, if needed using multiple appointments, and the extent of the impact that bad news, such as having to lose teeth, has on an individual is most often dependent on the way in which the information is communicated. Depression, anxiety and social withdrawal are seen in patients with tooth loss, and resulting compromised esthetics can be helped with therapy, relaxation techniques, and, in some cases, antidepressants. Any of the above symptoms should be addressed with a qualified psychotherapist to improve the quality of life. 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. In addition, stress reduction protocols may help in management of the disease as such in the view of the recent suggestions of the proposed mechanisms by which stress can contribute to the onset, exacerbation and maintenance of the periodontal disease. A recent study reported that psychotherapy offered at 3 levels (individual, group, and conjoint family psychotherapy) to AgP patients gave positive psychologic effects that restored their ability to socialize in their environment contributing to their positive experience in life. The above facts suggest that psychotherapy be incorporated for the future protocols for treatment of AgP patients suffering from emotional effects of tooth loss.

Other Treatment Modalities and Future Trends in Management of Aggressive Periodontitis: Host modulation therapy with systemically and locally administered agents is under research for therapy in aggressive periodontitis. Subantibacterial dose of Doxycycline has been approved for use in chronic periodontitis, but its use in aggressive periodontitis has to be confirmed by research. Adjunctive use of locally administered alendronate gel with SRP for host modulation has shown promising results in aggressive periodontitis. Newer generations of regenerative materials and advances in tissue engineering for regeneration and genetic engineering to modify the genetic risk factors seem to be really promising in the future. 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.

Conclusion

The key to successful management at present lies in early diagnosis of the disease and rigorous treatment employing the different treatment modalities mentioned in the paper 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.

REFERENCES

Albandar JM. Aggressive periodontitis: Case definition and diagnostic criteria. Periodontology 2000 2014;65:13-26

Armitage GC. Comparison of the microbiological features of chronic and aggressive periodontitis. Periodontology 2000. 2010;53(1):70–88. [PubMed]

Asikainen S, Jousimies-Somer H, Kanervo A, Saxén L. Actinobacillus actinomycetemcomitans and clinical periodontal status in Finnish juvenile periodontitis patients. J Periodontol 1986;57:91-3.

Black GV. Diseases of the peridental membrane having their beginning at the margin of the gum. In: Litch WF, editor. American System of Dentistry. Vol. 1, 2nd ed. Philadelphia: Lea Brothers; 1886. p. 953-79

Caton J. Periodontal diagnosis and diagnostic aids: consensus report. In: Proceedings of the World Workshop in Clinical Periodontics; 1989; American Academy of Periodontology;

Elamin A, Albandar JM, Poulsen K, Ali RW, Bakken V. Prevalence of aggregatibacter actinomycetemcomitans in Sudanese patients with aggressive periodontitis: A case-control study. J Periodontal Res 2011;46:285-91

Gottlieb B. 1923. Die diffuse atrophy des alveolarknochens. Zeitschrift Fur Stomatologie. 21:p. 195.

Guzeldemir E, Toygar HU. From alveolar diffuse atrophy to aggressive periodontitis: a brief history. Journal of the History of Dentistry. 2006;54(3):96–99. [PubMed]

Kaĵ J, Goultschin J, Benoliel R, Brautbar C. HLA DR4: Positive association with rapidly progressing periodontitis. J Periodontol 1987;58:607-10.

Kononen E, Müller HP. Microbiology of aggressive periodontitis. Periodontology 2000 2014;65:46-78

Kornman KS, Crane A, Wang HY, di Giovine FS, Newman MG, Pirk FW, *et al.* The Interleukin 1 genotype as a severity factor in adult periodontitis. J Clin Periodontol 1997;24:72-7.

Lang N, Bartold PM, Cullinan M, et al. Consensus report: aggressive periodontitis. Annals of Peridontology. 1999; 4:p. 53

Novak KF, Novak MJ. Aggressive Periodontitis. Carranza's Clinical Periodontology. 10th ed. Vol. 1. St. Louis: Saunders-An imprint of Elsevier; 2006 p. 506-11.

Orban B, Weinmann JP. Diff use atrophy of the alveolar bone (periodontosis). J Periodontol 1942;13:31-45.

Ranney RR. Classification of periodontal diseases. Periodontology 2000. 1993;2:13–25.[PubMed]

Ripamonti U. Paleopathology in Australopithecus africanus: a suggested case of a 3-million-year-old prepubertal periodontitis. merican Journal of Physical Anthropology. 1988; 76(2):197–210. [PubMed]

Ripamonti U. The hard evidence of alveolar bone loss in early hominids of southern Africa. A short communication. *Journal of Periodontology.* 1989; 60(2): 118–120. [PubMed]

Schacher B, Baron F, Roßberg M, Wohlfeil M, Arndt R, Eickholz P. Aggregatibacter actinomycetemcomitans as indicator for aggressive periodontitis by two analysing strategies. *Journal of Clinical Periodontology.* 2007; 34(7):566–573. [PubMed]

Shapira L, Eizenberg S, Sela MN, Soskolne A, Brautbar H. 1994. HLA9 and B15 associated with the generalized form, but not the localized form of early-onset periodontal disease. *J Periodontol.,* 65:219-23.

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