Dynamic therapeutic approach for individuals affected with aggressive periodontitis

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Dynamic Therapeutic Approach for Individuals Affected With Aggressive Periodontitis

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ABSTRACT Management of patients affected with aggressive periodontitis is complicated by several poorly understood etiological and modifying factors that create difficulty in establishing a universal treatment recommendation. The goal of this manuscript is to underscore the complexity of therapy and to provide some guidelines in the decision-making process and interdisciplinary therapy. A dynamic approach is presented to formulate strategies in diagnosis and treatment planning that is both patient- and site-specific.

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In assessing a patient with any disease, it is important to identify causative and modifying factors that contribute to the disease initiation and progress in order to offer a treatment. However, because of a complex combination of incompletely understood etiological and risk factors in periodontitis, it is not possible to assign a simple cause-and-effect diagnosis (e.g., streptococcal sore throat). Therefore, a classification system is utilized to study disease patterns and types in large populations of patients to provide a framework for studying the epidemiology, etiology, and treatment outcomes for a given group of similar diseases.[1-3] As a starting point, such a system can serve to generate a clinical framework for periodontal diagnosis. In clinical management of specific patients, a diagnosis should be made for the individual within the classification framework.[4]

The classification of "aggressive periodontitis" was adopted by the 1999 workshop of American Academy of Periodontology to describe a specific pattern of diseases previously classified as periodontosis, localized juvenile periodontitis, generalized juvenile periodontitis, early onset periodontitis, and rapidly progressive periodontitis.[5,6] This classification was adopted to avoid using a patient's age as criteria for categorizing periodontal disease. Instead, the classification is based on clinical, radiographic, historical, and laboratory findings.

Clinical Features of Aggressive Periodontitis

Aggressive periodontitis is a specific type of periodontitis with identifiable clinical and laboratory findings that are not characteristic for chronic periodontitis.

According to the consensus report of American Academy of Periodontol-

ogy on aggressive periodontitis the common features of the disease are:

- Patients are clinically healthy except for the presence of periodontitis;
- Rapid attachment loss and bone destruction; and
- Familial aggregation.

The following secondary features may also be present:

- Amounts of microbial deposits are inconsistent with the severity of periodontal tissue destruction;
- Elevated proportions of *Aggrigatibacter Actinomycetemcomitans* and *Porphyromonas gingivalis* in some populations;
- Phagocyte abnormalities;
- Hyper-responsive macrophage phenotype, including elevated levels of PGE2 and IL-1;
- Progression of attachment loss and bone loss may be self-arresting; and
- Lack of caries or low caries index.

The diagnosis and classification is based on clinical, radiographic, and historical data and, some, or possibly all, of the above characteristics. Laboratory testing may not be essential for assigning a diagnosis for aggressive periodontitis even though it could be helpful in clinical decision-making. The influence of modifying risk factors (e.g., cigarette smoking, emotional stress, drugs, sex hormones, etc.) should be considered to manage individual patients who are affected with aggressive periodontitis.

There are enough specific features to classify aggressive periodontitis into localized and generalized forms. These features are presented in **TABLE 1**.

A thorough review of medical and family history will aid to identify individuals whose periodontal disease may be associated with specific syndromes or systemic conditions. Individuals with significant systemic modifiers of the innate and adaptive immune responses should be classified as "periodontitis as a manifestation of systemic disease." Negative effects of certain systemic conditions will increase the patient's susceptibility to microbial plaque and consequently to severe and extensive periodontal attachment loss and early tooth loss. This group of diseases includes neutropenia, hypophosphatasia, leukemias, Chediak–Higashi syndrome, leukocyte adhesion deficiency, Papillon–Lefèvre syndrome, trisomy 21, histiocytosis, and agranulocytosis. Proper management of these patients requires management of systemic diseases that may be responsible for the patient's severe periodontitis in conjunction with periodontal infection control.[6,7]

TABLE 1

Characteristics of Localized and Generalized Aggressive Periodontitis According to American Academy of Periodontology

Localized Aggressive Periodontitis

- Age of onset around puberty
- Robust serum antibody response to infecting agents
- Localized first molar/incisor presentation with interproximal attachment loss on at least two permanent teeth, one of which is a first molar, and involving no more than two teeth other than first molars and incisors

Generalized Aggressive Periodontitis

- Usually affecting individuals under the age of 30 but patients may be older
- Poor serum antibody response to infecting agents
- Pronounced episodic nature of the destruction of attachment and alveolar bone
- Generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and incisors

Treatment Planning

The first factor in developing a treatment plan for patients who are affected by aggressive periodontitis is to identify the esthetic and functional needs amongst the patient, restorative dentist, and periodontist. It is critical to identify patient expectations, realistic attainability of the treatment desires, treatment limitation, risk of future breakdown, and dynamic nature of the therapy. When functional or esthetic concerns are present, treatment strategy needs to address the strategic value (risk and cost benefit considerations) of the remaining teeth in providing a functional and esthetic outcome, if infection control and periodontal stability is to be achieved. In these circumstances, a longer provisional phase period of nine to 12 months is advisable to evaluate the outcome of periodontal therapy and periodontal stability before committing to a definitive reconstructive phase. Occasionally, alternative restorative suggestions, such as shortened dental arch or transitional fixed or removable prosthesis, may be considered. This communication is especially important when there are major restorative treatment needs, particularly when considering implant therapy.

Patients with a history of severe chronic periodontitis and aggressive periodontitis may be at additional risk of adverse peri-implant soft-, and hard-tissue outcomes. Peri-implant infections share both bacteriological and histopathological similarities to both aggressive and chronic periodontitis.[8-14] Patients with a history of periodontitis pose a risk for peri-implant diseases, thus, in younger patients with aggressive periodontitis, especially the general-

ized type, it is prudent to defer major implant therapy to later stages when the patient is older.[15-17] This strategy may reduce the chance of early peri-implant complications and provide a better long-term prognosis of implant prosthesis.

When no major functional or esthetic concerns are present, the aims of the therapeutic approaches are primarily on maintaining the periodontium. Localized aggressive periodontitis has been reported to be self-limiting, whereas individuals with the generalized form continue to lose periodontal attachment and teeth over time; however, the residual periodontal lesion and bony defects are major contributing factor for future periodontal disease as the patient ages.[5,18,19] Also, some cases of localized aggressive periodontitis progress to generalized aggressive periodontitis, that may resemble or transform to chronic periodontitis at a later stage.[19-21]

Additionally, risk factors have similar long-term influences on both chronic periodontitis and aggressive periodontitis, although one could argue that with younger patient age and greater initial attachment loss may dictate a poorer long-term prognosis in aggressive disease. Since patients are mostly evaluated at a cross-section of a time, it requires a careful assessment of history and the clinical presentation of periodontitis to identify a past history of aggressive disease to identify a "burn out" stage that may be masked or confused with chronic periodontitis among the middle-aged or older population or with periodontitis as a manifestation of systemic disease.

Since the aggressive forms of periodontitis have distinctive features from chronic periodontitis and potentially respond differently to therapy, the authors are suggesting a tailored treatment planning approach in treatment of patients with aggressive periodontitis.[4,22] This approach is dynamic, both in planning and therapeutic recommendations. Unlike chronic periodontitis where there are more established protocols for therapy, the authors are suggesting that within the overall classification of localized or generalized aggressive periodontitis, existing therapeutic approaches need to be tailored individually based on clinical features and perceived patient- and site-specific diagnoses. This decision-making process is adaptive to following clinical parameters:

- Systemic association;
- Chief complaint and patient expectations (functional and esthetic demands including restorative/reconstruction needs);
- Onset of the disease and patient's age;
- Pattern (distribution) of the disease;
- Severity of attachment loss (pocket depths and clinical attachment loss, radiographic bone loss and bony defects);
- Quantity of etiological factors (plaque and calculus index/code);
- Severity of gingival inflammation (gingival index and bleeding on probing);
- Bacteriological association and consideration of systemic antibiotic therapy and adjunctive local anti-infective therapy;
- Initial mechanical debridement (scaling and root planing) protocol;
- Periodontal re-evaluation and supportive (maintenance) therapy;
- Surgical therapy for infection control and repair of periodontal defects; and
- Implant therapy.

This dynamic approach should be applied to both diagnostic (such as radiographic and laboratory tests) and therapeutic procedures. Following is a review and implementation recommendations considering above clinical parameters to provide guidelines for infection control and periodontal repair of individuals with aggressive periodontitis.

Mechanical Debridement

Scaling and root planing, as initial phase nonsurgical therapy, is traditionally performed by quadrants at different appointments. The efficacy of this approach has been demonstrated by a classical series of studies by Badersten et al.[23-25] Furthermore, a number of systematic reviews on the efficacy of different modalities of mechanical nonsurgical periodontal therapy have been published.[26-30] However, the effectiveness and the efficiency of the traditional mechanical therapy have mostly been analyzed in a patient population affected by chronic periodontitis. Aggressive periodontitis is considered a more site-specific and bacterial-specific periodontal infection that is strongly correlated to the host or early-in-life periodontal infection.[16,31,32] While mechanical debridement is effective in the reduction of bacterial plaque, specific bacteria repopulate periodontal pocket within three to seven days after treatment, restoring bacterial counts to almost pretreatment levels.[33]

Periodontal pathogens commonly associated with aggressive periodontitis, such as *A. actinomycetemcomitans, Bacteroides species,* and *P. gingivalis,* colonize different intraoral habitats in addition to periodontal pockets, including the tongue, buccal mucosa, saliva, and tonsils.[34-37] Saliva probably acts as the major vector of bacterial transmission in most inter-individual cases.[38] When there is an increase in periodontal pathogens around teeth, similar microbial flora are observed around neighboring implants as well, indicating an intraoral transmission of those presumptive periodontal pathogens.[39,40] Therefore, if periodontal treatment does not result in elimination of pathogens from the mucous membranes as well as periodontal pockets, these surfaces may function as:

- Source of reinfection for the healing and healthy periodontium after treatment;[33,41]
- Source of transmission to family members;[42] and
- Reservoir for infection of tissues around implants.[39,40]

Quirynen described that periodontal pathogens are present in various ecologic niches and that the transmission of these may occur from individual to individual as well as within the oral cavity among sites.[41] This evidence reinforces the need for a full-mouth approach to periodontal infection control especially in cases of aggressive periodontitis rather than treating individual sites. Full-mouth disinfection control as described by Quirynen (**TABLE 2**) consists of mechanical debridement within a short span of time (24-48 hours) with adjunctive use of local anti-infective agents for additional disinfection (i.e., chlorhexidine) during the initial healing period (two weeks).[43] This protocol may be supplemented with systemic antibiotic therapy when indicated. This approach considerably reduces the chance of re-infection of treated pockets by bacterial translocation from other untreated pockets or the intraoral sites. Furthermore, Guerrero also reported greater clinical improvement using an enhanced mechanical debridement within 24 hours and full-mouth disinfection through use of systemic antibiotic therapy and chlorhexidine rinses for two weeks, compared to traditional quadrant scaling and root planing for treatment of patients with generalized aggressive periodontitis.[44]

Considering a dynamic approach, the decision to perform nonsurgical mechanical debridement is dependent on presentation of etiological and local factors (plaque, calculus, and pocket depths). In cases of generalized aggressive periodontitis, full-mouth scaling and root planing utilizing a full-mouth disinfection approach is performed preferably within 48 hours. In cases of localized aggressive periodontitis generalized scaling (debridement) and localized root planing will be performed in one session.

When initial heavy inflammation with tenacious and heavy calculus and deep pockets are present, the patient will be scheduled for an early re-evaluation within two weeks of initial scaling and root planing to retreat areas with residual detectable calculus, with the aim of mechanical disruption of biofilm (if indicated, systemic antibiotic will be administered after initial scaling and root planing regardless of the need for early re-evaluation). Patients without adequate home care (more than 20 percent O'Leary plaque index) will be scheduled for biweekly plaque control appointments. Remaining patients will be re-evaluated monthly for the first three months.

TABLE 2

Original Protocol of Full-Mouth Disinfection Introduced by Quirynen et al.[49]

- Full-mouth scaling and root planing (the entire dentition in two visits within 24 hours, i.e., two consecutive days) under local anesthesia
- Brushing of dorsum of tongue for one minute with 1 percent chlorhexidine gel
- Mouthrinsing twice with 0.2 percent chlorhexidine mouth rinse for one minute (during the last 10 seconds, the patient had to gargle in an attempt to reach the tonsils)
- Subgingival irrigation of all pockets three times within 10 minutes with chlorhexidine 1 percent gel after both sessions of scaling and root planing and repeated on Day 8, using a syringe with marks at 6 and 8 mm
- Mouthrinsing at home with 10 ml of 0.2 percent chlorhexidine mouthrinse twice daily for one minute for the following two weeks
- Oral hygiene instructions including toothbrushing, interdental cleaning with interdental brushes or other aids, and tongue brushing

Antibiotic Therapy

A number of studies have demonstrated minimal improvement and high percentage of nonresponders when using mechanical debridement alone in treatment of patients with aggressive periodontitis.[45-48] This is due to the fact that nonsurgical therapy alone does not completely eradicate certain subgingival periodontal pathogens, including *A. actinomycetemcomitans* black-pigmented Bacteroides species, and Capnocytophaga species.[49-51]

Similarly, local antibiotic therapy does not seem to eliminate *A. actinomycetemcomitans* when used in treatment of localized aggressive periodontitis.[49,52,53] To target these specific presumptive periodontal pathogens that are highly associated with aggressive periodontitis, mechanical debridement needs to be supplemented with systemic antibiotics. Using this strategy, the number of spirochetes, *A. actinomycetemcomitans* and *Capnocytophaga* were reduced to undetectable levels and significant improvement in clinical outcomes were observed.[50,51,54,55] The use of systemic antibiotics may enhance gains in attachment level and alter the subgingival bacterial profiles.[56] Additionally, full-mouth scaling and root planing, along with systemic combination metronidazole and amoxicillin or metronidazole alone, and antimicrobial rinses have been advocated for patients with generalized aggressive periodontitis.[46-48,57-59]

Some authors propose systemic antibiotic therapy for all cases of moderate to severe periodontitis without microbial testing. This recommendation is made regardless of a diagnosis of chronic or aggressive periodontitis.[57,60,61] However,

TABLE 3

Recommendation for Use of Oral Systemic Antimicrobial Therapy Based on Detection of Putative Periodontal Pathogens (adopted from Slots)

Detection any of the following:
- *A. actinomycetemcomitans*
- Red complex[71]
  - *Tannerella forsythia*
  - *P. gingivalis*
  - *Treponema denticola*

Antibiotic recommendation:
- 250 mg amoxicilllin-375 mg metronidazole /TID/eight days
- In cases of penicillin allergy:
  - Metronidazole alone (500 mg/TID/8 days)
- In case of metronidazole and penicillin allergy:
  - Clincamycin 300 mg/TID/8 days or
  - Azithromycin (250-500 mg/QD/ 4-7 days)

Detection of:
- Enteric gram-negative rods

Antibiotic recommendation:
- Ciprofloxacin (500 mg/BID/8 days)

No detection of any of the following:
- *A. actinomycetemcomitans,*
- Red complex: [71]
  - *Tannerella forsythia*
  - *P. gingivalis*
  - *Treponema denticola*
- Enteric gram-negative rods

No systemic antibiotic therapy

QD=once daily, BID=twice daily, TID=thrice daily
Recommendation is for or systemically healthy adults with normal body weight.

widespread use of systemic antibiotic therapy in a large population of people affected by periodontitis has a potential of selecting antibiotic resistance species.[62] Moreover, patients with aggressive periodontitis benefit more than the patients with chronic periodontitis from adjunctive use of systemic antibiotic therapy.[63,64]

Clinically, the magnitude of change in some sites may be greater when antibiotics are used, making it a more viable treatment option.[22] The effectiveness of systemic antibiotic treatment increases when it is administered immediately after scaling and root planing.[65] Both the American Academy of Periodontology and the European Federation of Periodontology indicate that the adjunctive use of systemic antibiotic therapy benefits patients with aggressive periodontitis. However, both also emphasize that the optimal drug, dosage, and duration to provide the greatest effect is not completely understood.[63,66] Nevertheless, the choice of antibiotic treatment is best deferred to the result of bacteriological sampling.[67]

It should be mentioned here that there is growing evidence supporting the potential role of viruses in pathogenesis of aggressive periodontitis. It appears that a high periodontal load of active viruses such as the Epstein–Barr virus or cytomegalovirus is associated with aggressive periodontitis. There are hypotheses of synergistic viral and bacterial co infections in the pathogenesis of aggressive periodontitis; but their role, if any, in the initiation of the disease is not defined.[68-70] Therefore, the potential benefit of an antiviral treatment strategy is not currently established.

When utilizing the concept of dynamic therapeutic approach for the use of antibiotic therapy, one has to make a clinical judgment as to when and how (local or systemic) to administer antibiotic therapy. As mentioned earlier, the decision for the type of antibiotic is best to be deferred to bacterial sampling since the presence or absence of certain pathogens may change the decision to administer different choices or combination of antibiotic regiments. **TABLE 3** summarizes treatment recommendations based on the detection of putative periodontal pathogens. When a patient with classification of aggressive periodontitis is not positive for any of the red complex pathogens *(P. gingivalis, T. Forsythia, T. denticola), A. actinomycetemcomitans,* and enteric gram-negative rods, a retest may be indicated if clinical presentation of a given case would constitute suspicion of specific infection (i.e., in cases of severe periodontal disease specially among young individuals).[71] However, once a negative detection is confirmed, no systemic antibiotic therapy is advised. This strategy is employed to avoid unnecessary exposure of patients to a course of systemic antibiotic therapy. Yet, a subsequent bacterial retesting is recommended if no significant clinical improvement is observed after initial mechanical debridement and/ or subsequent surgical therapy. Use of local antimicrobial therapy may be considered for specific sites with supra bony pockets to reduce gingival inflammation only when no systemic antibiotic therapy is indicated.

Moreover, several local anti-infective agents are reported to provide favorable clinical outcome in control of inflammation and antibacterial property that might be of significance in management of periodontal infection among patients with aggressive periodontitis including 0.2 percent chlorhexidine, 10 percent povidone-iodine for professional use and 0.1-0.5 percent sodium hypochlorite for patient self-care.[43,72,73]

Surgical Therapy

There is a general agreement that favorable therapeutic outcome can be achieved treating patients with localized aggressive periodontitis even with cases of severe periodontal attachment loss. The rationale for surgery among patients with aggressive periodontitis is in part related to the perceived need to remove tissue invaded by *A. actinomycetemcomitans* as well as *P. gingivalis*.[74-77] Because the presence of these species within epithelial cells may not be eliminated after nonsurgical and systemic

antibiotic therapy, it is plausible that recolonization may occur, which might contribute to recurrent or refractory disease.[78] Furthermore, since periodontal lesions associated with deep pockets, molars, furcation sites, and angular bone defects respond less favorably to repeated nonsurgical instrumentation, a surgical approach may be considered for debridement versus repeated nonsurgical instrumentation on the sites with evidence of inflammation after nonsurgical therapy.[79-81] Surgical techniques for root debridement have been successfully utilized to treat localized aggressive periodontitis in combination with systemic antibiotics with significant improvements in probing depths and attachment levels, and evidence of radiographic bone fill after five years of maintenance.[51,82-85] Also to repair bony defects, osseoinductive surgical procedures with the use of autografts, allografts, alloplast, with or without barrier membranes, have been reported with successful and favorable outcomes in treating localized aggressive periodontitis lesions.[86-92] Each of the above studies contained few subjects and defects, making comparisons between the groups difficult. In contrast. Gunsolley reported that among patients with localized aggressive periodontitis who received treatment, there was no difference in periodontal attachment gain over the 15-year period for those who received scaling and root planing alone compared to those who were treated surgically.[19]

The fact that different authors report favorable results using different nonsurgical as well as surgical modalities (including different bone graft materials and membranes and in different combinations) may suggest that the healing of aggressive periodontitis lesions may be related to the diagnosis, site-specific healing potential, the timing of the surgical intervention, and defect morphology rather than the choice of materials and surgical techniques. In choosing inductive techniques, it is important to consider the potential for post surgical complication of using barrier membranes. Sites with membrane show a significant variability in result due to potential of exposure and bacterial contamination. Furthermore, membrane contamination is highly associated with less favorable results.[93-95]

IN CHOOSING inductive techniques, it is important to consider the potential for post surgical complication of using barrier membranes.

The result from Nowzari et al. underscores the importance of full-mouth infection control prior to considering surgical repair of periodontal lesions.

In cases of generalized aggressive periodontitis, there is an overall reluctance among clinicians to perform surgical therapy. Some of the reasons for this reluctance are severe attachment loss on presentation, possible risk of unknown or undetected systemic disease, a history of unfavorable surgical outcomes with previous experiences, or a reluctance to perform surgery in patients with unknown prognosis and risk factors.[22] While a cautious approach to surgery in patients with generalized aggressive periodontitis is prudent, in sites where there is continuous clinical inflammation and deep pockets of >5 mm with bleeding on probing, a surgical treatment for root debridement such as Modified Widman flap with no osseous resection is indicated.[96] However, any resective surgery should be avoided at this stage to allow a potential for repair of osseous defects. Many authors have reported good success of osseous inductive surgery to repair intraosseous defects in patients with generalized aggressive periodontitis.[65,97-99] Once the infection is under control, osseous corrective surgery may be indicated to correct residual osseous defects during the long-term maintenance phase of the treatment. An apically positioned flap with osseous recontouring (osseous surgery) is a very effective approach in correcting remaining shallow to medium intraosseous defects, reducing pockets and subgingival detection of *A. actinomycetemcomitans*.[100,101] Resective types of periodontal surgery are more effective than access flap surgery in combating subgingival *A. actinomycetemcomitans* apparently due to the excision of *A. actinomycetemcomitans*-infected gingival tissue and pocket reduction to levels permitting adequate oral hygiene measures in the long-term maintenance of patients.[102]

In choosing corrective/recective surgical approaches, it is critical to observe bony defect anatomy and morphology as well as esthetic implications of such treatment modalities with an understanding of the risk-benefit outcome, the ultimate goals of periodontal therapy and reconstructive treatment needs for a particular patient.

Maintenance and Reconstructive Restorative Therapy

When clinical improvement is achieved with initial infection control, a two-month maintenance schedule is planned for the first six to nine months.

After nine months, radiographic evaluation of residual bony defects may be indicated to correct remaining osseous defects and deep pockets (>5 mm specially on posterior teeth). Based on the clinical outcome of therapy patient should be scheduled on a long-term maintenance interval of two to three months for a close monitoring of periodontal condition, especially in cases of generalized aggressive periodontitis. Once periodontal prognosis and stability are established, reconstructive periodontal, implant restorative therapy can be planned.

A flow chart is illustrated in **DIAGRAM 1** to provide a guideline using a dynamic therapeutic approach. Use of such approach is exemplified in periodontal treatment planning of the following cases of individuals affected with generalized or localized aggressive periodontitis. The quotation marks are used to reference patients own words of their history and chief complaints for case description.

Case 1

This case presents a 40-year-old African-American male from a North African origin with no systemic condition (nonsmoker ASA I). He reported to Department of Advanced Periodontology with a chief complaint of "loose front tooth" and esthetic concern about maxillary anterior diastema. His dental history consists of almost a lack of any professional dental visits since he never had any pain or "cavities." He reports of oral hygiene regiment of daily Miswak (a teeth cleaning device/brush made from a twig of the *Salvadora persica* tree) use. He reported of "loosening" of his teeth within the past five years and enlargement of the space between his maxillary anterior teeth. Furthermore, he presented a history of early edentulism of both parents at a similar age due to "loose teeth and gum infections." Despite the lack of professional dental visit there are no existing caries and no restorative therapy for any previously existed caries. He was also positive for red complex bacteria (*P. gingivalis, T. Forsythia, T. denticola*) and *A. Actinomycetemcomitans*.

Clinically, generalized gingival inflammation, generalized deep pockets of 5-10 mm with generalized bleeding on probing were present. Radiographically, generalized advanced horizontal bone loss, vertical bone loss, and circumferential intraosseous defects were present.

ONCE THE INFECTION is under control, osseous corrective surgery may be indicated to correct residual osseous defects during the long-term maintenance phase of the treatment.

Although there are detectable etiological factors (plaque and calculus) they are not abundant and not consistent with the extensive attachment loss and deep vertical and circumferential defects. This patient does not have any contributing systemic or other local factors (occlusal traumatism or severe malpositioning or crowding) that can be associated with extent of periodontal attachment loss. Either the attachment loss started early in his youth or has rapidly progressed through adulthood, which, in both cases, would be classified as aggressive periodontitis. Hence, this case is an example of generalized aggressive periodontitis (**FIGURE 1**).

Considering No. 8 root fracture, esthetic demands, restorative needs and advanced loss of attachment; prognosis of the maxillary dentition is poor. However, the mandibular arch does not require a functional or esthetic rehabilitation. Therefore, a reasonable treatment approach will be a transitional maxillary removable prosthesis and maintenance of mandibular teeth from Nos. 19 to 30 (**FIGURE 1**). The infection control stage will consist of:

- Full-arch maxillary extraction and delivery of an immediate transitional complete denture;
- Extraction of Nos. 17, 18, 30, and 31;
- Mandibular right and left quadrant scaling and root planing in a single session, one week after extraction of nonmaintainable/poor prognosis teeth;
- Systemic antibiotic therapy with amoxicillin and metronidazole (250 mg each one every eight hours for eight days);
- Oral hygiene instructions and 0.2 percent chlorhexidine for two weeks; and
- Periodontal re-evaluation six weeks post scaling and root planing followed by three-month periodontal maintenance.

Surgical therapy was not indicated since neither deep pocket depths (>5 mm), nor clinically detectable inflammation was detected after initial therapy. **FIGURE 1** demonstrates the clinical outcome with generalized probing depths of 2-3 mm and localized 4 mm probing on mesiolingual of No. 19 and distolingual of No. 29, one-year post-treatment. Implant therapy of maxillary arch may be considered at this stage, since periodontal infection is deemed under control.

Case 2

This case is presentation of a 35-year-old Asian female with no systemic condition (nonsmoker ASA I). The patient reported with the chief complaint of protruded maxillary anterior tooth No. 10 and diastema. She reported of a

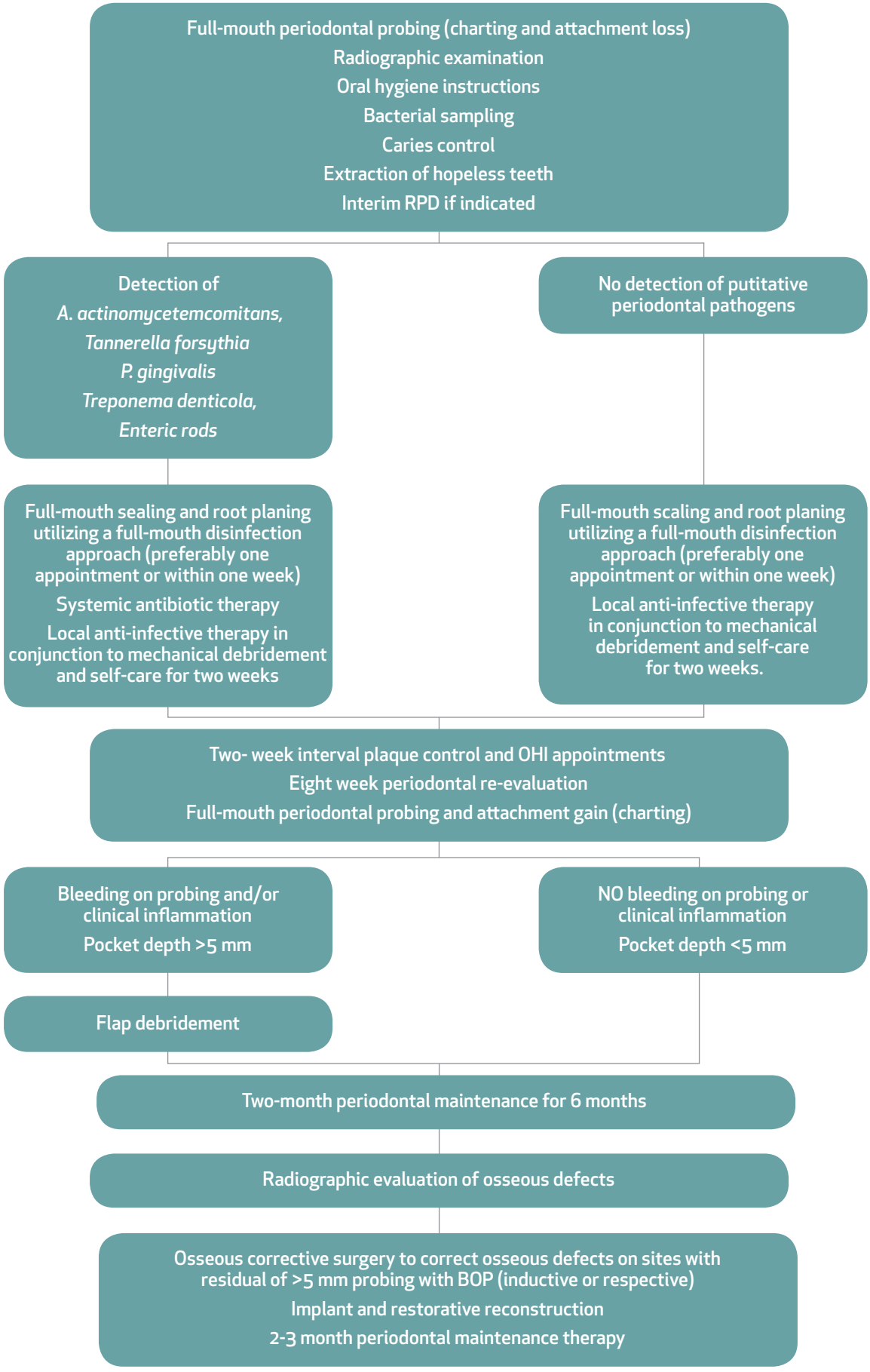

DIAGRAM 1. Dynamic therapeutic approach in treatment planning for individuals affected by aggressive periodontitis.

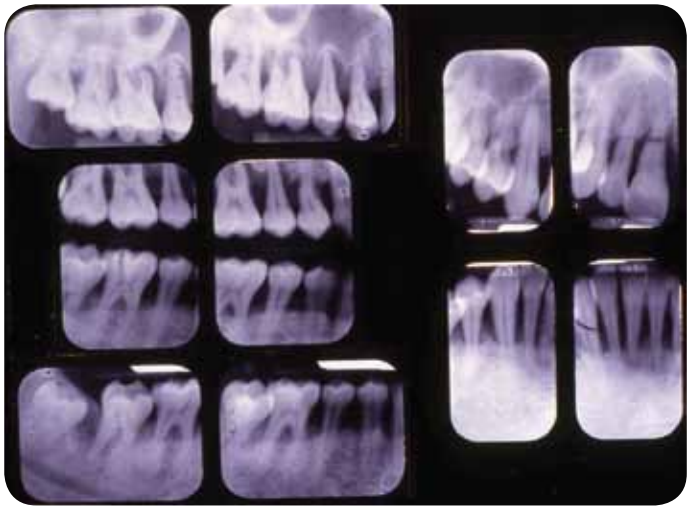

FIGURE 1A. Patient's right.

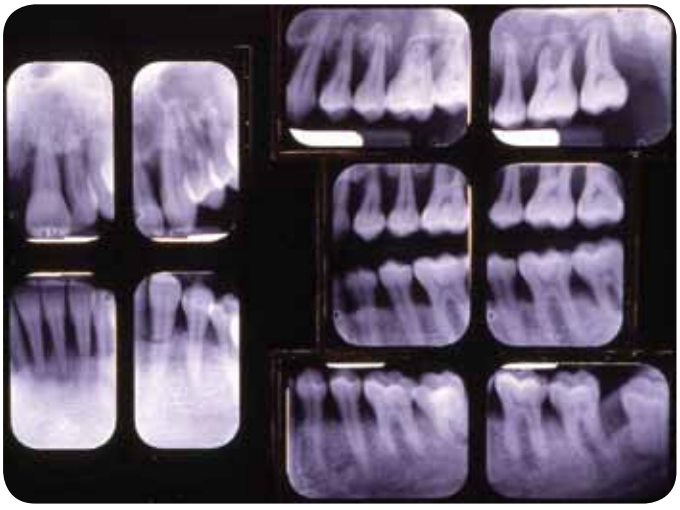

FIGURE 1A. Patient's left.

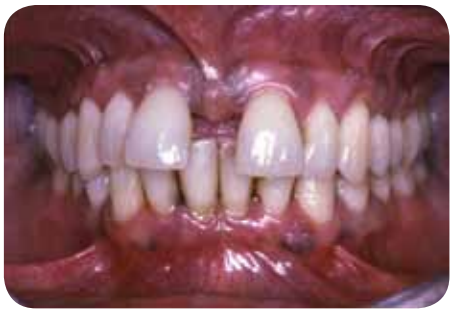

FIGURE 1B.

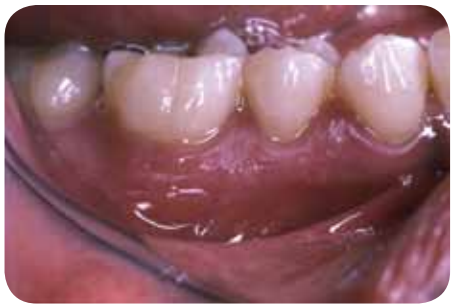

FIGURE 1C.

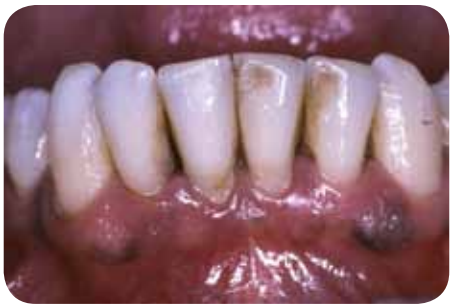

FIGURE 1D.

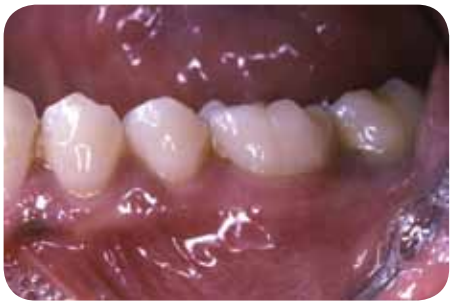

FIGURE 1E.

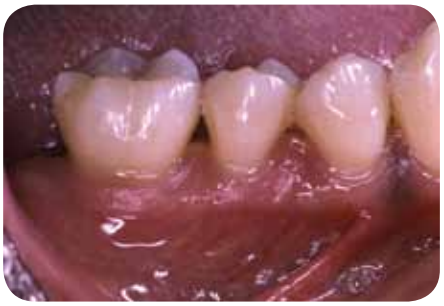

FIGURE 1F.

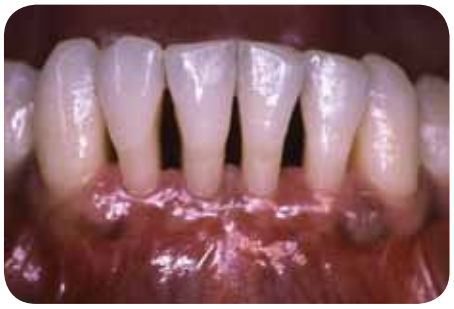

FIGURE 1G.

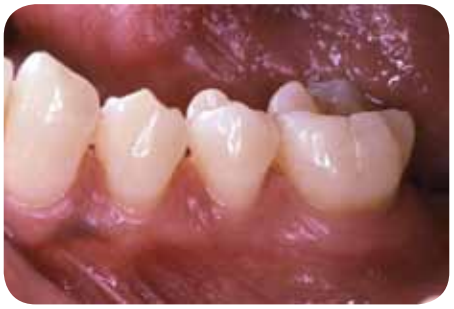

FIGURE 1H.

FIGURE 1. Clinical and radiographic presentation of case 1: Full-mouth preoperative radiographic (**A**) and clinical (**B**) presentation. Note lack of caries despite advanced periodontal infection. Preoperative clinical presentation of mandibular arch (**C, D AND E**) and one-year postoperative clinical presentation (**F, G, AND H**).

"gum infection" on her lower front teeth when she was in her late teens. She also reported that within the past year she had noticed an increase in maxillary anterior spacing between Nos. 9 and 10 and flaring of No. 10. Additionally tooth No. 31 was recently removed due to "gum abscess." There was a history of sporadic dental treatment and "a general cleaning" a few years prior to her periodontal consultation. She presented with severe

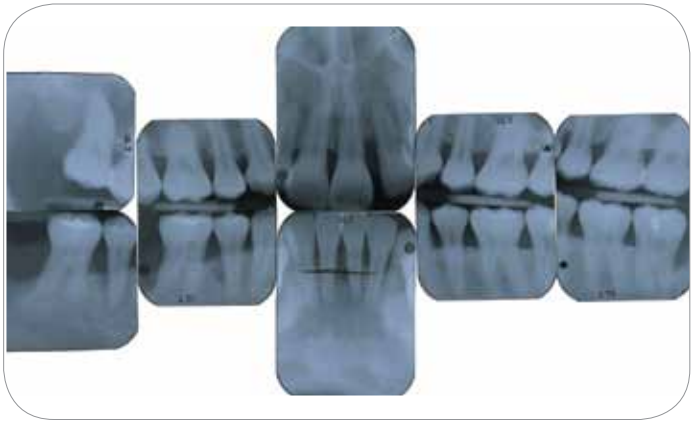

FIGURE 2A.

FIGURE 2B.

FIGURE 2C.

FIGURE 2. Radiographic and clinical presentation of case 2: Preoperative (**A**) and three-year postoperative radiographic (**B**) presentation of maxillary and mandibular teeth. Note radiographic bone gain and re-establishment of lamina dura of Nos. 2, 3, 10, 14, 18, 24, 25, 29, and 30 following resolution of infection and orthodontic therapy. (**C**) Clinical presentation of pre- and postorthodontic therapy to correct diastema and alignment of the teeth. *(Orthodontic treatment courtesy of James Clark, DDS, Mission Viejo, Calif. Restorative evaluation courtesy of Christopher Travis, DDS, Laguna Hills, Calif.)*

attachment loss on anterior and first molars and a generalized mild-to-moderate attachment loss. There were patterns of site-specific infection that may be correlated to presence of local etiological factors. However, despite the lack of professional dental care and inadequate home care (O'Leary plaque index of 100 percent) no caries or restorative treatment for previously existing caries were present. Initially generalized bleeding on probing with pocket depths of 6-10 mm and areas of moderate to advanced horizontal and vertical osseous defects were detected (**FIGURE 2**). Her bacterial sampling results were negative for red complex bacteria *(P. gingivalis, T. Forsythia, T. denticola)* enteric gram-negative rods and *A. actinomycetemcomitans.* Given her history and pattern of disease, her periodontal diagnosis was classified as a history of localized aggressive periodontitis that was transforming to a generalized form of chronic periodontitis.

Her treatment plan consisted of:

- Oral hygiene instruction (initial plaque index of 100 percent);
- Bacterial sampling;
- Four quadrants of scaling and root planing in two appointments within one week. No systemic antibiotic were administered since no *A. actinomycetemcomitans, P. gingivalis, T. Forsythia, T. denticola,* or enteric gram-negative rods were detected by microbial testing;
- Two weeks rinse with 0.2 percent chlorhexidine, re-evaluation for plaque control and OHI;
- Periodontal re-evaluation six weeks post initial scaling and root planing for pocket depth and attachment gain;
- Flap debridement No. 29 distal due to 7 mm pocket post scaling and root planing with bleeding on probing, suppuration, persistent gingival inflammation and intraosseous defect;
- No. 18 autogenous bone graft to correct distal 2-3 wall intraosseous defect;
- Nos. 14 and 15 palatal approach osseous surgery to correct shallow interproximal crater for pocket reduction and flap debridement of No. 4 to repair mesial vertical intraosseous defect; and
- Orthodontic therapy to realign teeth and reduce maxillary anterior diastema one year after periodontal therapy and assessment of periodontal stability.

At three-year postoperative evaluation, generalized 2-3 mm probing depths, clinically healthy gingiva, radiographic resolution of osseous defects, and re-establishment of a clear lamina dura indicating periodontal stability. At this stage the patient is ready for definitive restorative plan, which includes:

- Bonded restorations to contour inter proximal contact of Nos. 8, 9 and 10; and
- Implant-supported restoration No. 31 to increase occlusal support and masticatory function.

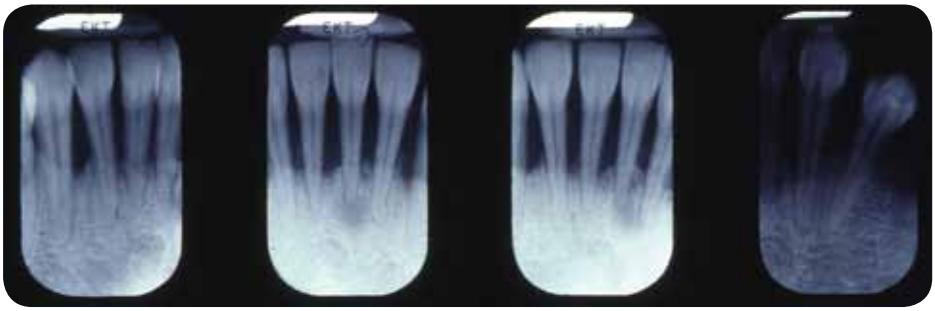

FIGURE 3A.

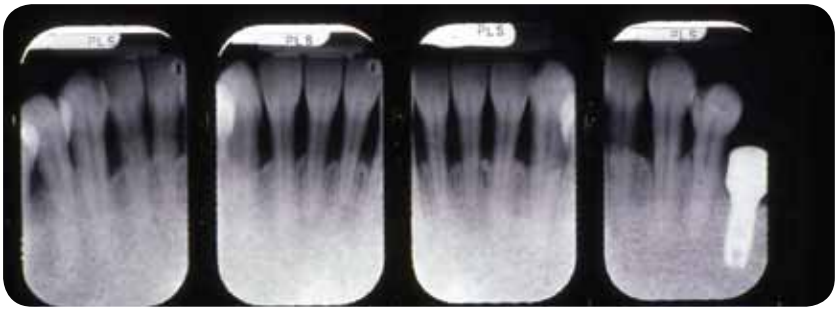

FIGURE 3B.

FIGURE 3. Radiographic presentation of case 3: Preoperative (**A**) and seven-year postoperative (**D**) presentation of mandibular anterior teeth. Note radiographic bone gain following resolution of infection.

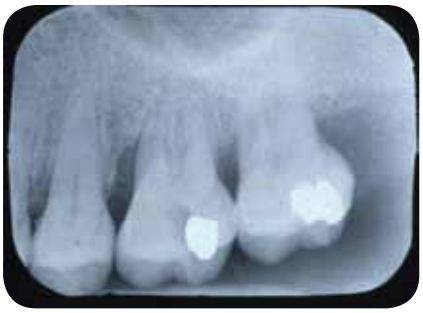

FIGURE 4A.

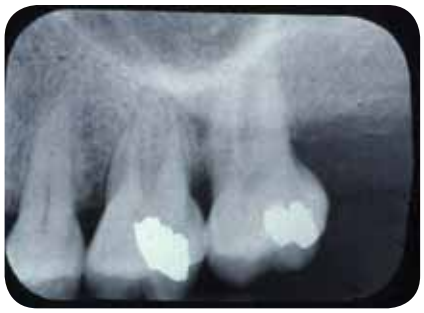

FIGURE 4B.

FIGURE 4. Preoperative (**A**) and postoperative (**B**) radiographic presentation of case 4: Note resolution of mesial vertical intraosseous defect of tooth No. 14 and interproximal craters of No. 12, 13, 14, and 15 one year following periodontal osseous surgery and flap debridement.

Case 3

Clinical and radiographic appearance of mandibular anterior sextant of a 28-year-old female affected with generalized aggressive periodontitis is presented on **FIGURE 3**. Putative periodontal pathogens *A. actinomycetemcomitans* and *T. forsythia* were detected by microbial sampling. Periodontal treatment included scaling and root planing in conjunction with amoxicillin and metronidazole. No surgical therapy was performed in this sextant since initial therapy resulted in reduction of probing depths and resolution of periodontal inflammation. Seven-year postoperative evaluation demonstrates radiographic bone repair and disappearance of periapical periodontitis of mandibular anterior teeth. At this point, the patient was ready to proceed with implant therapy to replace posterior missing teeth.

Case 4

The case presented on **FIGURE 4** exemplifies an adult patient (46-year-old female) with a history of localized aggressive periodontitis, which had been self-limiting and transformed to a secondary chronic periodontitis. There is a site-specific infection by gram-negative rods affecting mesial of tooth No. 14. Treatment for this patient included initial scaling and root planing, systemic antibiotic therapy for initial infection control followed by maxillary left osseous surgery to correct residual osseous craters between Nos. 12, 13, 14 and 15, as well as No. 14 vertical intraosseous defect. Postoperative radiographic appearance demonstrates periodontal healing and repair of osseous defects.

Case 5

The case presented on **FIGURE 5** exemplifies a 32-year-old female with a history of localized aggressive periodontitis. No other area of attachment loss was detected in this patient. There is a site-specific infection by *P. gingivalis* affecting mesial of tooth No. 19. Treatment for this patient included initial scaling and root planing and systemic antibiotic therapy for initial infection control. Upon re-evaluation, bleeding on probing, and residual pocket depth indicated a need for flap debridement of No. 19 mesial intraosseous defect. Surgical photograph indicates remaining calculus on mesial of No. 19 post scaling and root planing. One-year clinical and radiographic appearance demonstrated periodontal healing and repair of osseous defect.

Case 6

This case is presentation of a 24-year-old African-American male He is systemically healthy with no known systemic contributing factors (nonsmoker ASA I). This is a young individual with severe attachment loss and nonmaintainable

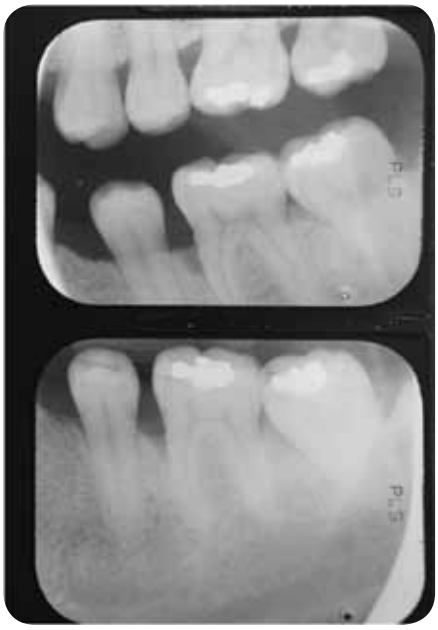

FIGURE 5A.

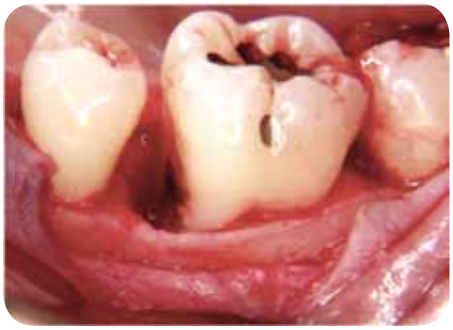

FIGURE 5B.

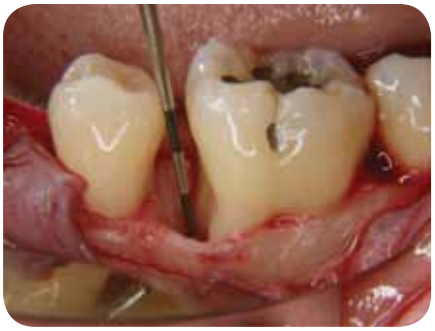

FIGURE 5C.

FIGURE 5. Clinical and radiographic presentation of case 5: Preoperative (**A**) radiograph, flap debridement, and removal of residual calculus (**B AND C**), one-week postoperative healing (**D**), and one-year postoperative clinical and radiographic presentation (**E AND F**). Note soft-tissue healing and radiographic bone gain.

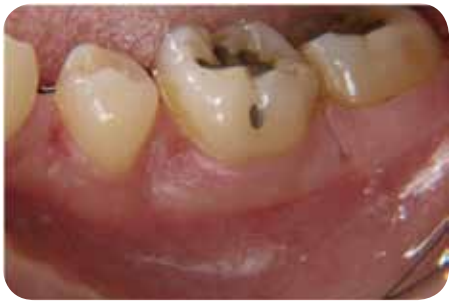

FIGURE 5D.

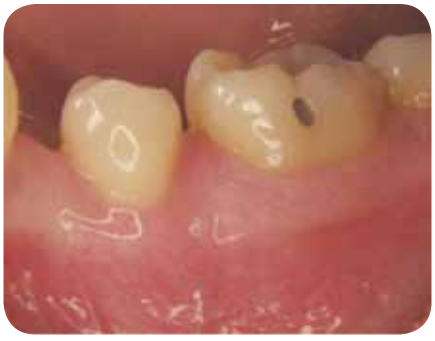

FIGURE 5E.

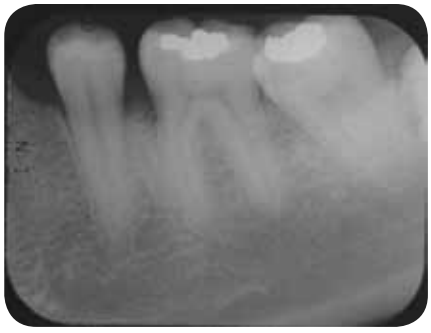

FIGURE 5F.

teeth: Nos. 3, 4, and 5. A fixed partial denture was previously fabricated to replace No. 7 that was previously removed two to three years prior to periodontal consultation because of "looseness" and "gum infection." Initially generalized bleeding on probing with generalized pocket depths of 5-10 mm and areas of moderate to advanced horizontal and vertical osseous defects were detected (**FIGURE 6**). His periodontal condition is classified as generalized aggressive periodontitis.

Loss of posterior maxillary right molar and premolar teeth as well as potential loss of Nos. 6 and 14 created both functional and esthetic restorative demands. To consider a reconstructive plan for this patient, control and stability of periodontal disease condition is essential. A transitional removable prosthesis would be considered during periodontal therapy as a provisional replacement of the missing teeth. His treatment plan includes:

- Oral hygiene instruction (initial plaque index of 100 percent);
- Bacterial sampling;
- Extraction of Nos. 3, 4, and 5;
- Four quadrants of scaling and root planing in two appointments within one week and intraoperative 10 percent povidone-iodine irrigation due to severity of gingival inflammation and therefore a perceived need for local antibacterial therapy;
- Systemic antibiotic therapy (ciprofloxacin 500 plus metronidazole 500 mg two times a day for eight days) since *T. forsythia* and enteric gram-negative rods were detected by microbial testing;
- Two weeks re-evaluation for plaque control and OHI for two months;
- Periodontal re-evaluation in six weeks post scaling and root planing for pocket depth and attachment gain;
- Flap debridement of mandibular right and mandibular left quadrants due to residual 5-7 mm pockets post scaling and root planing with bleeding on probing, suppuration, and persistent gingival inflammation.

Implant therapy will be considered after evaluation of resolution of peri-

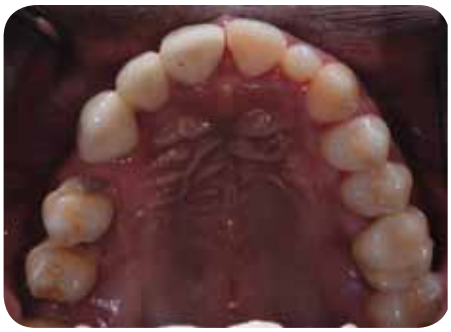

FIGURE 6A.

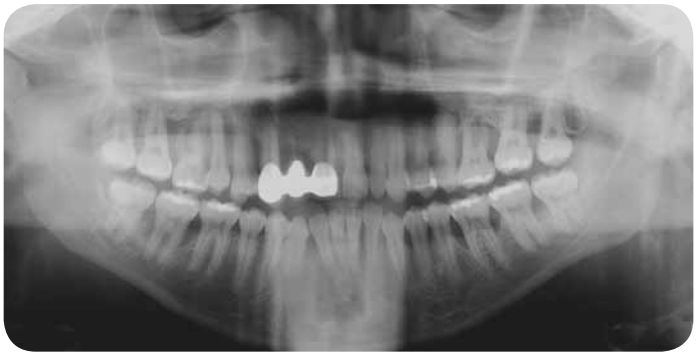

FIGURE 6B.

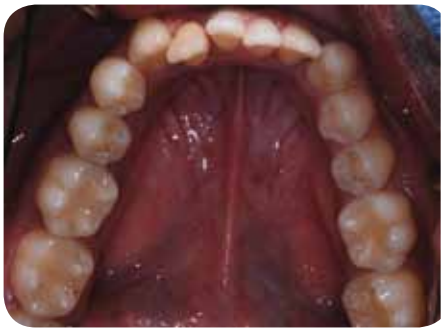

FIGURE 6C.

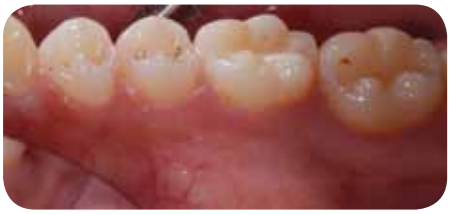

FIGURE 6D.

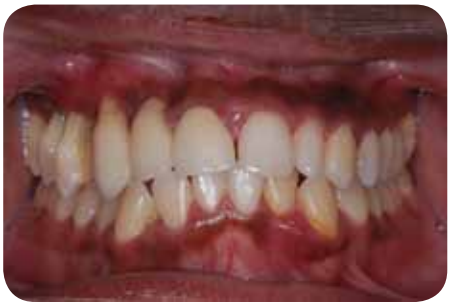

FIGURE 6E.

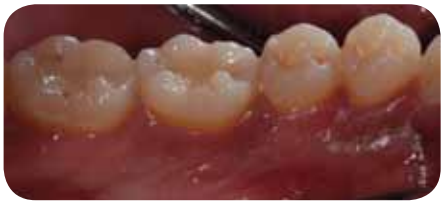

FIGURE 6F.

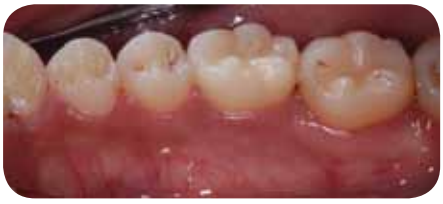

FIGURE 6G.

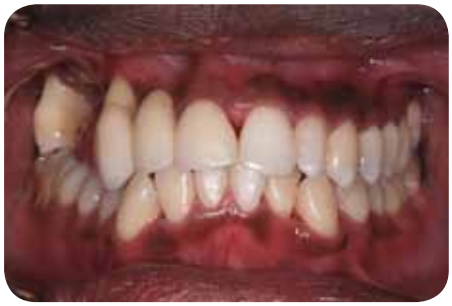

FIGURE 6H.

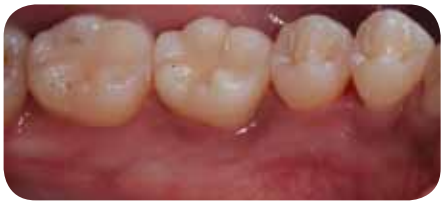

FIGURE 6I.

FIGURE 6. Clinical and radiographic presentation of case 6: Preoperative occlusal view (**A AND C**) and panoramic radiograph (**B**). Note lack of caries despite severe periodontal attachment loss. (**D**) mandibular right lingual; (**E**) retracted anterior intercuspal occlusion; (**F**) mandibular left lingual preinitial therapy; (**G, H, AND I**) postinitial therapy indicating overall resolution yet localized residual inflammation. *(Courtesy of Yvonne Tam DDS, USC Periodontology resident.)*

odontal inflammation, radiographic resolution of bony defects. This final assessment may be completed after a period of 12 months to evaluate potential breakdown of the periodontal condition.

Conclusion

Aggressive periodontitis presents with a diverse clinical manifestation. It is associated with various bacterial, viral, immunological, and systemic cofactors. Therefore, treatment outcome is very much dependent on the manifestation of disease, the patient's age, as well as functional and esthetic demands. To address a patient's treatment, the clinician needs to employ an approach tailored to each individual's clinical manifestation and historical background. When choosing a treatment modality, a "dynamic therapeutic approach" will provide flexibility in adapting decision-making to the outcome of every diagnostic evaluation and treatment modality during each phase of therapy. Ultimately, interdisciplinary communication among different providers is the key element in successful management of patients affected by aggressive periodontitis. ■■■■

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