A Review of Aggressive Periodontitis and an Associated Case Report

Abstract: Aggressive periodontitis is a debilitating oral disease that results in rapid destruction of the periodontal tissues. It has recently been reclassified and ongoing efforts are now being made to understand its pathogenesis and record its prevalence in the population. This case report gives a brief outline of studies that have investigated why this disease tends to occur in otherwise young healthy adults. The mechanisms behind such extensive periodontal damage are discussed. A case report of a patient with aggressive periodontitis, treated in the Dublin Dental School and Hospital, is presented and the rationale behind his treatment is discussed.

Clinical Relevance: An example of one of the modalities of treatment for a patient with a hopeless periodontal prognosis.

In 1999, the International Workshop for a Classification of Periodontal Diseases and Conditions was convened to formalize a new classification system for periodontal disease. The older World Workshop classification system of 1989 had a number of shortcomings, such as overlap of disease categories, no gingival disease component included, an inappropriate emphasis on age of onset and rate of progression, and inadequate or unclear classification criteria. The 1999 classification system solved some of these problems and removed age and rate dependent categories, such as prepubertal or juvenile periodontitis and rapidly progressive periodontitis. These categories now come under the broad heading of aggressive periodontitis. According to the 1999 classification, otherwise known as the Armitage classification, aggressive periodontitis as a diagnosis is based on several criteria:

- Affected individuals seem healthy;
- Rapid rate of progression;
- Familial aggregation;
- Generally lower levels of microbial deposits than other forms.

Clinical features

Clinically, it is broken down into two types:

- Localized – usually has a circumpubertal onset with periodontal destruction often being centred around the first permanent molars and central incisors. However, atypical patterns are possible. It is also frequently associated with Actinobacillus actinomycetemcomitans and abnormal neutrophil function, but with an elevated antibody response.
- Generalized – usually seen in patients under 30 years of age (but can be variable) with generalized interproximal attachment loss affecting at least three permanent teeth other than the first permanent molars and central incisors. The attachment loss usually occurs in pronounced episodic bursts of destruction, with Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis most commonly associated with the disease. Neutrophil abnormalities and a poor antibody response are often associated findings.

The estimated prevalence of aggressive periodontitis ranges from 0.1% to 15% among Caucasians, Hispanics and African Americans, with a greater prevalence in African Americans when compared to Caucasians.

Aetiological factors

In an effort to understand the aetiology and progression of this disease most studies have focused on the following associations.
Familial aggregation

Family linkage studies can be used to confirm a genetic predisposition to a disease and search for susceptibility or linkage genes. Some cases of aggressive periodontitis show Mendelian inheritance patterns. However, no single mode of inheritance has been shown that would account for the various forms of phenotypic expression that have been observed. A number of studies have examined family groups and have found autosomal dominant, autosomal recessive and X-linked patterns of inheritance all to be possible.

Nucleotide polymorphisms

While family studies provide valuable information, they do not indicate specific genes involved in disease processes. Gene polymorphisms have been investigated as markers for aggressive periodontitis. These polymorphisms are errors in the genetic sequence that can result in up-regulation or down-regulation of a gene, which can cause increased or impaired function of the phenotypic expression.

The most intensely studied include IL-1, IL-4, IL-10,7 TNF-α,9 Fcγ receptors10 and α,11 Human Leucocyte Antigen,8 Vitamin D receptors12 and N-formylpeptide receptor.13 Matrix metalloproteinase 1 and 3,14 Lactoferrin and Calprotectin polymorphisms.15 These studies have shown very variable results with no clear associations, except for Fcγ and α polymorphisms, which affect antibody structure and therefore interfere with their normal function. These Fc receptors link the two main aspects of the host immune response, namely the cell mediated and humoral responses, so a disruption to their structure will result in uncoordinated immune responses. Other studies have yielded contradictory results when looked at as a whole.

Neutrophil defects

Polymorphonuclear neutrophils (PMNs) are a major component of the innate defence system. PMN defects have been shown to result in increased susceptibility to periodontitis. In aggressive periodontitis, a number of these defects have been investigated:

- Adherence: some evidence exists for associations in certain types of aggressive periodontitis, with defective receptor function in PMNs, which prevents PMNs adhering to their targets.13
- Chemotaxis: there appears to be an association with decreased neutrophil chemotaxis and the localized form of aggressive periodontitis.14
- Phagocytosis and intracellular killing: some studies have shown defective phagocytic ability15 to be linked to aggressive periodontitis.

Antibody response

Antibodies are produced in response to antigenic exposure (vaccination/infection). IgM are the first set of antibodies to respond but IgG is the major responder to bacterial and viral antigens. It has been hypothesized that the increased levels of IgG found in localized aggressive periodontitis may provide some protection and localization of the destruction, whereas a deficiency in IgG in some patients may lead to the more generalized form.16

Smoking and stress

Smoking can affect antibody levels and their avidity to certain pathogens17 and has also shown to be associated with aggressive periodontitis. It has also been shown to cause a decreased response to treatment in both chronic periodontitis18 and aggressive periodontitis.19 Data from longitudinal studies show increased levels of plaque, increased attachment loss and decreased response to therapy in smokers versus non-smokers.20

Both stress and smoking have been shown to have a high predictive value on future attachment loss21 in aggressive periodontitis. Both seem to be linked to their negative affects on the immunological system due to the decreased cytokine production and increased stress hormone (such as glucocorticoids) levels in the body.

Root abnormality

Root abnormalities can be local contributing factors for aggressive periodontitis patients and can affect the pathogenesis, the severity of the periodontal destruction and treatment outcomes.22 Some conditions can render the periodontium more susceptible to rapid breakdown, such as hypophosphatasia23 in which defective cementum formation allows rapid attachment loss. Radicular multigrooving24 results in numerous deep wrinkles along the length of the root surface which can facilitate rapid breakdown of attachment as a result of multiple inaccessible retentive niches.

Herpes virus

*Human cytomegalovirus* (HCMV) is a member of the Herpes virus family. Along with *Porphyromonas gingivalis* (PG), it is strongly associated with localized aggressive periodontitis and they seem to act synergistically.25 Slots and Contreras26 described a model for viral bacterial interaction where Herpes virus activation results in suppression of periodontal defences and stimulation of pro-inflammatory factors, which would result in periodontal destruction. After quiescent periods, subsequent reactivation of the virus would aggravate the inflammatory response of the host. Yapa et al.27 showed HCMV in 64.7% and Epstein-Barr virus (EBV) in 70.6% of aggressive periodontitis patients, with only 6.3% of healthy subjects showing EBV present. In the aggressive periodontitis group, co-infection existed in 41% of cases.

Bacterial factors

Periodontal micro-organisms co-exist and proliferate in biofilms. In excess of 500 taxa have been identified in oral biofilms. These biofilms form enclosed micro-environments that consist of heterogeneous layers and channels that allow a microbiological homeostasis to exist while protecting bacteria from host responses. Different bacteria will proliferate more efficiently under certain conditions and these biofilms often indirectly facilitate these bacteria by producing a suitable micro-environment. Some species are considered more periodontopathic than others and, in aggressive periodontitis, Gram-negative rods like *Porphyromonas gingivalis* and *Aggregatibacter (Actinobacillus) actinomycetemcomitans* are frequently implicated. These bacteria are the two most studied and possess many virulence factors that enable them to evade host defences, colonize periodontal sites, break down periodontal tissues and alter the biofilm environment to suit their own proliferation. *Porphyromonas gingivalis* has been shown to express different types of fimbriae.
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**Treatment considerations**

The aims of treatment in most cases will be to halt the progression of disease, eliminate any contributing risk factors and prevent recurrence, if possible. In some particularly aggressive cases, the periodontal support is so compromised that the eventual loss of the dentition is inevitable.

The specific treatment regimen depends on the extent and severity of the periodontal destruction which can only be assessed by a complete periodontal and radiographic examination. Localized or generalized aggressive periodontitis are generally treated in a similar fashion to chronic periodontal disease, which usually involves oral hygiene instruction and reinforcement, smoking cessation, full mouth debridement of both supra- and sub-gingival plaque deposits, followed by supportive periodontal therapy and maintenance. Particularly deep periodontal pockets (>7 mm) or refractory sites that are resistant to non-surgical debridement are usually treated by surgical therapy. Microbiological sampling and antibiotic sensitivity testing may also be appropriate. It has been shown that systemic antimicrobial administration of amoxicillin and metronidazole (regimens vary in different institutions) immediately after surgical/non-surgical disruption of the plaque biofilms leads to increased long-term gain of attachment levels, especially when *Aggregatibacter actinomycetemcomitans* is present. Local delivery devices for other antimicrobials, such as chlorhexidine or tetracycline, have also been used but do not appear to be as successful as systemic antimicrobials.

Owing to the case specific nature of these treatment regimens, management of these types of patients is often best facilitated by a specialist periodontist. Because of the familial nature of this disease, periodontal examination and counselling of family members is also often required. Tailored oral hygiene and maintenance regimens are often required for individual patients and, if identified and treated early in the disease process, the prognosis for the remaining dentition can be favourable. If, however, aggressive periodontitis is diagnosed at a late stage, with severe alveolar bone loss, then the dentition is often in a state of terminal decline and prosthodontic intervention is inevitable.

**Case presentation**

The following is a case presentation of aggressive periodontitis that affected a young adult male and was treated over a two-year period in the Dublin Dental School and Hospital (DDSH). The patient, a 30-year-old healthy male, presented as an emergency to a general dental practitioner complaining that a tooth had ‘fallen out on its own’. Upon examination, the general practitioner diagnosed generalized advanced periodontal destruction, with the upper left lateral incisor lost as a result of inadequate bony support. The patient had a provisional prosthesis fabricated and a prompt referral to the DDSH was made.

A full history and periodontal examination was undertaken, along with radiographs and a full blood work-up. Clinical findings revealed a minimally restored dentition with no root abnormalities, and periodontal probing showed some subgingival deposits of calculus. Probing depths of greater than 7 mm were recorded in most sites throughout the mouth. Orthopantomographic examination (Figure 1) showed generalized severe loss of alveolar bone. Blood results were all within normal ranges. A detailed family history also revealed no familial predilection for periodontitis. A diagnosis of generalized aggressive periodontitis was made based

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**Figure 1. OPG upon presentation.**
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Immediate complete upper and lower dentures were fabricated by first extracting all posterior teeth so that posterior occlusal rims could be used to record the patient’s existing occlusal vertical dimension and set the occlusal plane to the desired level. The remaining anterior teeth were extracted at the fit appointment of the immediate complete dentures. Three months after the last natural tooth was extracted, implant assessment radiographs were taken to assess remaining bone levels (Figure 2). A total of eleven 3i (Biomet 3i Inc, Palm Beach Gardens, FL) fixtures were placed using a two-stage protocol. Six regular platform implants (four 15 mm long implants and two 11.5 mm long implants) were placed in the maxilla and five 15 mm long 3i regular platform implants were placed in the edentulous mandible (Figure 3) with the aid of upper and lower acrylic stents that were constructed by duplicating the patient’s existing dentures. After one week of healing, the upper and lower dentures were relined with a soft reline material (Tempo, Lang Dental Mfg Co, Wheeling, IL, USA).

Second stage surgery, after 3 months of healing, exposed the fixtures and the patient then began the prosthodontic phase of treatment. An irreversible hydrocolloid or alginate (Hydrogum Thixotropic, Zermack Spa, Italy) impression of the upper and lower fixtures was made using closed tray impression copings in a stock tray. Initially, the copings were attached to the implants in the mouth after the healing abutments had been removed. The impression was then made and removed from the mouth after setting, with the impression copings remaining in the mouth. These copings were then removed from the mouth and attached to implant analogues and replaced in the set impression via repositioning grooves along the side of the coping. This impression allowed preliminary casts to be poured in type IV dental stone (Jade Stone, Whipmix Corp Ltd, KY, USA) (Figure 4). This initial cast provided an approximate position of the implants relative to each other and the edentulous ridge. However, when implants are divergent relative to each other (as is usually the case when multiple implants are placed), the removal of a closed tray impression and subsequent re-attachment of impression copings can lead to distortion of their position.34

To overcome this potential source of inaccuracy, an open tray impression technique was used, in which the pick-

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### Table 1. Treatment options for late stage generalized aggressive periodontitis.

<table>
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<th>Treatment Option 1</th>
<th>Gradual transition to complete maxillary and mandibular dentures</th>
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**Figure 2.** OPG after clearance of remaining teeth (21-12-04).

**Figure 3.** OPG after fixture placement (26-05-05).

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on the patient’s age, severe periodontal destruction, good health, rapid rate of destruction and lower than expected microbial deposits. An extremely poor prognosis was given for the remaining teeth.

Treatment options were discussed (Table 1) and the patient expressed a keen interest in an implant-supported prosthesis as he felt a removable option would be undesirable. It was decided to transition the patient to an edentulous state and proceed towards the final goal of an implant-supported prosthesis using a standard Bränemark two-stage submerged protocol. This would involve placement of the implants and complete wound closure, with subsequent re-entry after 3 months of healing for second stage surgery to place healing abutments. These cylindrical abutments produce a trans-mucosal cuff of tissue that allows prosthesis and implants to be linked. The patient was to be rendered edentulous for a period of months prior to placement to allow a substantial decrease in the periodontopathic flora that would have otherwise been present in the dentate state.33
up impression copings remained in the impression after removal from the mouth, unlike the closed tray approach. To ensure no further movement of the implants relative to each other, the open tray impression copings were initially attached to the implant analogues on the preliminary cast and linked with Pattern Resin (GC Pattern Resin, GC United Kingdom Ltd, Bucks, UK) (Figure 5). These were subsequently sectioned longitudinally 24 hours before the master impression was made (Figure 6) to allow for the associated shrinkage of cold cure acrylic resins. A custom tray with windows to accommodate the open tray impression copings was fabricated from visible light cured custom tray material (Triad, Dentsply International, York, USA).

The sectioned impression copings were then transferred to the patient’s mouth and positive seating confirmed radiographically. The copings were then re-attached to each other intra- orally by beading GC Pattern Resin (GC United Kingdom Ltd, Bucks, UK) on to the adjacent sectioned surfaces. By joining and sectioning these copings out of the mouth on a preliminary cast, the majority of the resin shrinkage occurs out of the mouth and any distortion due to the polymerization of the beaded portion of acrylic is negligible. Once set, an impression using a combination of regular and heavy bodied polyvinylsiloxane (President, Coltene, Switzerland) was made (Figure 7). 3i implant analogues (Biomet 3i Inc, Palm Beach Gardens, FL) were attached to the copings and a soft tissue master cast was poured up using synthetic gingival material (Gingifast, Zeramex Materials, Germany) and type IV dental stone (Jade Stone, Whipmix Corp Ltd, KY, USA)(Figures 8a and b). By splinting the copings together, this technique allows a more accurate transfer of the position and angulation of the implants to be made, which is of the utmost importance in avoiding sectioning and soldering or costly remakes of the metal framework at a later stage.

Temporary cylinders were incorporated into a Triad base (Triad, Dentsply International, York, USA) to give stable screw-retained bases for the wax occlusal rims. The rims were then used to make facebow records (Whipmix arbitrary bow, Whipmix Corp Ltd, KY, USA), interocclusal registration and record centrelines and smile lines as for complete denture construction (Figure 9). The casts were mounted on a 2240 Whipmix Articulator (Whipmix Corp Ltd, KY, USA) and the denture brought to try-in stage and initial approval of the set-up obtained from the patient. Putty (President, Coltene, Switzerland) facial indices of the try-in dentures were made to seat directly on to the master cast, which was notched in the land portion of the cast to ensure reproducible seating of the matrices. These indices served as a guide for placement of the wax teeth on to the framework at a later stage (Figure 10). Procera CAD-CAM titanium frameworks (Nobel Biocare AB, Göteborg) were milled and tried into the patient’s mouth and accuracy and passivity of fit confirmed with periapical radiographs taken using the paralleling technique and the one screw test (Figures 11, 12 and 13). This screw test, as advocated by Jemt, involves screwing one retaining screw into the distal most abutment and examining visually and radiographically whether any lift-off of the prosthesis occurs at the other abutments.
Acrylic denture teeth (Genios Degudent, Dentsply International, York, USA) were then set-up on the metal framework and the aesthetics and occlusion defined following standard prosthodontic procedures (Figure 14). The gingival contours of the wax were festooned to simulate root eminences and the prosthesis finished. The acrylic and metal framework was contoured to facilitate cleaning by raising the tissue surface above the gingiva in the mandibular arch and by ensuring only light tissue contact with convex polished surfaces in the maxillary arch, thus also preserving a palatal seal, which is vital for adequate phonetic function. The patient was subsequently instructed in hygiene procedures with both an interdental brush (TePe Munhygienprodukter AB, Sweden) and a water jet cleaning device (Water Pik Inc, Waterpik Technologies, Fort Collins, United States).

The processed prosthesis was returned and any occlusal discrepancies due to processing errors in the application of the acrylic to the framework corrected intra-orally using shim stock and articulating paper (Figure 15). A composite-based staining kit (Gradia, GC GRADIA gum shades, GC United Kingdom Ltd, Bucks, UK) was used to enhance the gingival architecture subtly by blending the root eminences of the acrylic and the underlying denture teeth, intensifying the hue between root eminences and colouring the interdental papillae (Figure 16). The prosthesis was then returned to the patient’s mouth, inserted and torqued down to 20 Ncm using a manual torque driver. The patient was again instructed as to the hygiene procedures necessary. The access holes were provisionally sealed and the patient was reviewed at one week, one month and six months.

After 6 months, the patient returned and the prosthesis was removed and the condition of the peri-implant tissues assessed (Figure 17) and bone levels around the implants (now in place 18 months) assessed radiographically (Figure 18). All implants showed stable bone levels and no complications or patient complaints were reported. The prostheses were re-inserted and the screws tightened to 30 Ncm of torque. The access holes were now definitively sealed with PTFE (PolyTetrafluoroethylene, Enflo Canada Ltd, Grand Falls, Canada) tape and composite resin (Herculite XRV, Kerr Corporation, Orange, CA, USA) (Figure 19). Annual follow-up appointments were arranged thereafter.

**Discussion**

When diagnosed early, treatment...
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of aggressive periodontitis can involve both surgical or non-surgical therapy and anti-microbial therapy, with an associated extensive supportive therapy regimen. However, when late presentation occurs, as described here, treatment options are more limited. Treatment difficulties are confounded by the fact that most presentations occur at a relatively young age. Situations, such as these, where the patient has to be rendered edentulous, can be psychologically and emotionally challenging for the patient. This is reinforced by the fact that, in general, these patients are young, fit and healthy and never imagine being faced with the possibility of losing all their natural teeth at such a young age.

Implant-supported prostheses have been successfully used in the management of edentulous patients for many years. Their placement in previous sites of aggressive periodontitis is a more recent phenomenon.38

There are very few robust studies examining the difference between implant survival in healthy and periodontally challenged patients. A recent review,39 of implant therapy in partially edentulous periodontally compromised patients, found only 13 papers that dealt with this matter and only four of them were deemed of sufficient quality to be reviewed. Two of these studies34,40 compared the success rate of implants in healthy and periodontally challenged patients, with Karoussis’ study being the only 10-year prospective paper comparing these two patient groups. This would therefore be regarded as the highest form of evidence available for assessing this situation. Karoussis’ study showed survival rates of 96.5% (out of 91 implants) in the non-periodontal disease group and 90.5% (out of 21 implants) in periodontitis patients after 10 years.40

Hardt et al showed similar results after 5 years, with survival rates of 97% in non affected patients (out of 92 implants) and 92% in those with periodontal disease (out of 100 implants).44 Of the two remaining studies, Leonhardt41 followed 54 implants being placed in patients previously treated for advanced periodontitis with a reported survival rate of 94.7%.

These three studies show that
periodontally challenged patients can be successfully and predictably restored with dental implants.

The fourth study, by Mengel et al., was a prospective study, with 3- and 5-year results that followed the progress of 10 partially dentate patients previously treated for generalized aggressive periodontitis and chronic periodontitis and restored with implant-supported prostheses. They compared gingival health, bone loss, microbiological flora and survival of the implants. A total of 36 implants were placed in the aggressive periodontitis group, with two early failures and two ‘sleeping’ implants, resulting in an overall success rate of 88% and survival rate of 94.4%. The prosthesis success rate was 100% after 5 years. Twelve implants were placed in the chronic periodontitis group and, after 3 years, implant success and prosthesis survival were both 100%.

Another 5-year prospective study, not included in Van der Weijden’s review, also examined implant restorations in patients susceptible to periodontitis. They found that bone loss in the first year and thereafter was small (0.41 mm over 5 years) and no difference existed between machined and rough surface designs.

Despite the paucity of literature and the relatively small sample sizes in the quoted studies, it would seem that restoration of an orally debilitated patient with implant-supported prostheses is a valid and predictable treatment, even in periodontally compromised patients. There does appear to be a slightly increased risk of implant failure in these patients, but more clinical studies of longer duration will be needed to confirm this trend.

However, the success rates quoted above were all from studies conducted on partially dentate patients, and it remains to be seen if better success rates are achieved with patients, such as the one presented here, who had his periodontopathic burden effectively eliminated by rendering him edentulous.15 After connection to an implant prosthesis, however, some of the original flora return to colonize the oral environment, although in greatly reduced numbers.44 It would appear that, over the short term, these levels remain low, but longer observation periods are again needed to confirm this.

The choice of prosthesis for this patient was based primarily upon the patient’s age and preference for a fixed option, but also upon the amount of residual bone and interocclusal space present. Accurately positioned implants are also a prerequisite for this type of prosthesis, and a surgical stent greatly facilitates their correct placement relative to the final tooth position. The use of a hybrid acrylic-metal superstructure combines the strength and rigidity of the metal with the versatility and ease of adjustment and repair of acrylic. No aesthetic loss occurs with this type of prosthesis, provided correct tooth type and shape are selected and attention is paid to the finishing details. The alternative for this type of fixed restoration is a porcelain fused to metal (or zirconia) superstructure. This approach is both technically more demanding, in terms of compensating for such a large amount of porcelain contraction during firing, and financially more demanding, in terms of laboratory bills and maintenance costs.

The use of an all-in-one CAD-CAM titanium framework has been shown to be more accurate than the conventional casting processes previously used to fabricate such large structures.45 Therefore, when sufficient attention to detail is paid to making the definitive impression and generating the master cast, costly remakes and time consuming sectioning of the frameworks are now rarely indicated. The consequences of ill-fitting implant prostheses go beyond the fiscal, as it has been shown that an increased number of biological (bone loss and soft tissue inflammation) and mechanical (screw loosening and fracture) complications occur when prostheses don’t accurately fit the mating surfaces of their opposing implants.46

The benefits of well constructed fixed implant prostheses are apparent from both a functional and psychological viewpoint. This has been borne out in many oral health impact studies, where a patient’s (particularly younger age groups) preferences for fixed as opposed to removable implant prostheses have been evident.44 Once a patient is correctly instructed, this type of prosthesis can be hygienically maintained by the patient and a regular annual recall can ensure both continued compliance and absence of complications.

Conclusion

This report outlines some of the current thinking on aggressive periodontitis and shows some of the difficulties which may be encountered in dealing with the patients usually affected by this insidious disease. Future advances in detection and early treatment may help in slowing the disease progression but, in advanced cases, such as described in this case report, the fixed implant-supported option may be the treatment of choice.

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References


