CASE REPORT

Generalised Aggressive Periodontitis: A Case Report and Review of the Literature.

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ABSTRACT

Background: Generalized aggressive periodontitis is a rare periodontal disease characterized by generalized loss of alveolar bone usually affecting young individuals and may result in early tooth loss and psychological effects. Severity of the periodontal effects vary among patients and depends on duration before presentation, and may also determine the options and outcome of treatment.
Objectives: A case report of generalized aggressive periodontitis managed in our centre and review of available literature on the subject in English language.
Case: *A* 32-year-male who presented with 18months history of mobile, mal-aligned teeth and bad breath at the oral diagnosis clinic of the University of Maiduguri Teaching Hospital, Maiduguri.
Extra-oral examination revealed bilaterally enlarged submandibular lymph nodes- firm, mobile and not tender. Intra-orally, the patient had a full complement of teeth except the lower right first premolar. Oral hygiene was fair but with presence of suppuration from the gingiva around the lower incisors and the lower right first molar.
Conclusion: Clinical outcome of treatment is better with early diagnosis. A consensus on case definition is needed for epidemiological studies. The case presented demonstrated the clinical and radiological features necessary for diagnosis of the condition.

KEYWORDS: Aggressive periodontitis, generalized aggressive periodontitis, bone resorption, genetic predisposition, quality of life

Introduction

The periodontal tissues refer to both hard and soft tissues that surround and support the root of the teeth and include the gingiva, cementum, periodontal ligaments, and alveolar bone. Periodontitis, an inflammatory condition of the periodontal tissues resulting from host response to bacterial infection associated with dental plaque may be seen in all groups of patients of both gender. It is characterized by destruction of the periodontal tissues including resorption of the alveolar bone. Of particular interest is a distinct group referred to as aggressive periodontitis (AgP) that, in most cases, affects people who are otherwise systemically healthy.[1] This condition is believed to have a familial tendency and most importantly, a rapid rate of loss of periodontal soft tissue attachment and alveolar bone destruction which is inconsistent with the bacterial plaque present.[1,2] AgP can occur in a localized or generalized form defined by the number of teeth affected. Generalized aggressive periodontitis (GAgP) appear to be the most severe form of all periodontal disease and is characterized by "generalized interproximal attachment loss affecting at least three permanent teeth other than first molars and

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incisors".[3] Although uncommon, the prevalence of GAgP remains unknown mostly as a result of variations in case definition.[4,5] It is generally known to have a racial predilection with blacks more commonly affected. The aetiology of this disease is multifactorial- microbiologic, immunologic, and environmental/ behavioral risk factors in a background of genetic predisposition.[3] Treatment modality is dependent on severity at time of presentation and the outcome is greatly improved by early diagnosis. The aim of this paper is to present a case managed in our center and a review of literature.

Prior to 1999, several terminologies had been used to describe AgP based on popular opinion concerning its etiology, age of onset and clinical presentation of the diseased periodontal tissues. The term "periodontosis" had been in use, as proposed by Orban and Weinmann.[6] It was proposed based on the belief that the rapid periodontal tissue destruction in the affected young adults was not inflammatory in origin but rather degenerative. But with increasing evidence of an inflammatory etiology, Butler in 1969,[7] used the term juvenile periodontitis (JP) to replace periodontosis. The introduction of the term "juvenile" was to categorize the age group of patients presenting with the condition, as opposed to chronic periodontitis (CP) that is seen in older people. The American Academy of Periodontology (AAP) at a workshop in 1989 reviewed this term and replaced it with "early-onset periodontitis" with sub-classification "rapidly progressive periodontitis".[8] The use of these terminologies (early-onset periodontitis and rapidly progressive periodontitis) was based on the age of onset as well as the rate of progression of the disease. The terminologies remained in use for the next 10 years but not without criticism. According to Armitage,[9] the criticisms were mainly due to the classification's reliance on age of onset and rate of progression. They argue that these term presupposes that the clinician knows exactly when the disease started and the rate at which it is progressing, things that cannot be ascertained clinically. These concerns prompted a review of the terminologies by the AAP in 1999, and the use of "aggressive periodontitis" as the new term with localized aggressive periodontitis (LAgP) and generalized aggressive periodontitis (GAgP) as subtypes.[9] The new term "aggressive" was to lay emphasis on the rapidity of periodontal tissue destruction rather than on the age of onset as it was with the previous classification. Although these new terms have remained in use, discussions are still ongoing on the appropriateness of the term "aggressive" by those who believe that the condition has several similarities with CP, and may actually be a severe form of this condition.[10-12]

There is paucity of data on the prevalence of GAgP. This is however not due to lack of prevalence values but rather due to the varying criteria used in the different studies for identification of the cases, that is, a problem with case definition.[4] The case definition for the different prevalence studies is affected majorly by the age ranges included in the studies, and consequently the overlap between cases of severe CP and AgP. According to the AAP definitions of 1999, GAgP is suggested to "usually affect persons under 30 years of age, but patients may be older".[1] At the same time it was suggested that CP may affect persons under 30 years of age producing severe periodontal tissue destruction in the presence of a commensurable amount of bacterial plaque deposit. Thus the dilemma and the great variability in the global prevalence rates of the disease from epidemiologic studies. As a result, the global prevalence of AgP and GAgP remains elusive due to lack of proper case definition.[4] Prevalence values of GAgP ranging from 0.1% to 2.6% have been reported

among different populations.[13-15] Demmer and Papapanou[4] in their review concluded that these prevalence values to be an over estimation of the real prevalence due to overlap of cases of CP and GAgP, caused by the 1999 AAP definition. However, despite the problem of case definition and global variation seen in the prevalence estimates, the available data consistently confirms that is generally rare, most prevalent in Africa and in populations of African descent, and commoner in men.[4,13]

GAgP is a multifactorial disease that results from an interplay of genetic, microbiologic, immunologic and environmental/behavioral factors. The major underlying factor has been determined to be a genetic predisposition to an exacerbated response by the affected person to certain pathogenic bacteria.[3] The familial aggregation of the condition has been linked by several studies to transmission of gene polymorphisms that alters host susceptibility and immune response.[2] Researchers have investigated the association between polymorphisms of gene clusters that code for IL-1, IL-4, IL-10, TNF-α, Fc γ and α, Human Leucocyte Antigen (HLA), matrix metalloproteinase (MMP) 1 and 3, Lactoferrin and Calprotectin, and immune receptors.[16-19] Although the results of these studies did not show clear associations between the polymorphisms and the two forms of AgP, IL-10 promoter and Fc γ polymorphisms are believed to be associated with the generalized form.[20,21] Fc γ polymorphisms can lead to modulation of neutrophil superoxide production and predispose to GAgP.[22] Another important observation from these genetic studies is the ethnic variations in the type of gene polymorphism associated with AgP. Some polymorphisms may be associated with periodontitis in certain ethnic groups, but not in others.[16,18,19] It is however agreed that the chance of developing AgP is related to the number of genetic risk factors an individual has inherited.[23] In this setting of gene polymorphism, the individual becomes genetically predisposed to a combination of malfunctioning neutrophils, macrophages, fibroblasts, epithelial and dendritic cells, with the neutrophils and macrophages at the center of these malfunctions.[24-28] Microbial triggers set off the malfunctioning defense cells resulting in increased adhesion, reduced chemotaxis, increased superoxide and nitric acid production, reduced phagocytosis, elevated levels of prostaglandin E2, IL-1 and macrophage inflammatory protein (MIP), with resultant periodontal tissue destruction[25-28] Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis have been implicated to play the key role in initiating and perpetuating this "hyper-inflammatory" state that contribute to the extensive and rapid damage of the periodontium.[11,29] In a recent study, Hwang et al.[30] isolated Treponema Denticola and Campilobacter rectus from subgingival plaque of GAgP, and also detected significantly higher levels of IgG in the serum of the subjects compared to subjects with LAgP and CP. They thus suggested that these organisms may play similar roles in GAgP as Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis.

Patients' complaints on presentation usually borders around the effect of the condition on function, esthetics, pain and less commonly psychological effects. GAgP has generalized attachment loss affecting at least three permanent teeth in addition to the incisors and first molar in individuals younger than 30 years of age or older.[1] With this generalized attachment loss due to periodontal tissue destruction, complaints can include: increasing mobility of the teeth and spacing which may affect function and esthetics; pain and bleeding due to inflammation; dentine hypersensitivity due to exposed dentine following root exposure; bad breadth due to suppuration from the infected periodontal

pockets; and tooth loss. The history may suggest presence of similar symptoms in other family members.

Examination usually reveals minimal bacterial plaque, and occasionally an inflamed gingiva but the presentation in most cases is that of a gingiva that is free of inflammation. Tooth migration, spacing, tooth mobility, gingival recession and root exposure, pus discharge and deep periodontal pockets are all findings that become obvious on intraoral examination. Radiographic finding may show generalized bone destruction ranging from mild crestal bone resorption to severe and extensive alveolar bone destruction depending on the severity of the disease. The defects may be a combination of vertical and horizontal defects involving all incisors, first molars and at least three other permanent teeth.

The aims of treatment are to halt the progression of the disease and preserve the dentition and to eliminate contributory factors. The loss of the whole dentition may however be inevitable in some cases. The treatment regimen which is the same as for CP is therefore dependent on the severity and extent of the periodontal tissue destruction. Essentially, treatment of GAgP consists of a non-surgical phase to be followed by surgical therapy and subsequently supportive periodontal therapy for maintenance. Non-surgical therapy may suffice in mild cases without advanced periodontal tissue loss. Most cases however require surgical therapy as well as prosthetic rehabilitation due to lost teeth. Whatever the treatment regimen used, it is important for the patient to know their role in maintaining the periodontal health which is this case is for a lifetime.[31]

Mechanical plaque removal by full mouth supragingival and subgingival scaling and root planing remains the first line of treatment in the non-surgical therapy. This is to eliminate or reduce the microbial etiologic agents. The success of this treatment is dependent on adequate patient education on maintenance of oral hygiene and requires instructions and reinforcement.[31] Chemical adjuncts like 1% povidone iodine and chlorhexidine 0.12% or 0.2% have been suggested as mouth rinses to further help reduce microbial load.[32] The use of systemic antimicrobials as a single-agent therapy with doxycycline or minocycline, tetracycline, metronidazole, azithromycin, clindamycin or combinations of metronidazole and amoxicillin or metronidazole and ciprofloxacin has been documented and recommended in the treatment of GAgP.[33-35] In a systematic review by Haffajee et al.,[36] it was concluded that systemically administered antibiotics with or without scaling and root planning and/or surgery produced better clinical results when compared to these therapies alone without antibiotics.

Surgical removal of bacterial plaque is usually reserved for particularly deep periodontal pockets (>7 mm) or sites that are resistant to non-surgical debridement. Such deep pockets require flap procedures to increase visibility and enhance complete debridement of the periodontal pockets. The open flap debridement may be done alone or in combination with resective or regenerative procedures.[37] Regenerative surgical procedures may be required to replace destroyed periodontal soft and hard tissues. Bone replacement grafts to augment the lost alveolar bone with the use of autografts, allografts, xenografts or alloplastic materials have been studied with promising results.[37-39] This is to the alveolar bone support for the remaining teeth as well provide bulk for needed implant supported prosthesis where necessary.[36,38] Although autograft remain the best option, obtaining it in large quantities needed for GAgP remains a challenge.[31] Guided tissue regeneration (GTR), a barrier technique that use membrane barriers,

excludes gingival connective tissue from the healing wound and has been documented to produce better results when used in conjunction with bone grafting.[40]

It is important to state that patients with GAgP will not only require management by a periodontologist to control infection and arrest the progression of the disease, but also the services of other specialists. The management should therefore incorporate a multidisciplinary approach to attend to the functional, esthetic and psychological problems of the patient. Prosthetic rehabilitation with removable dentures or implant retained prosthesis have been used in patients with GAgP to replace lost teeth. Contrary to previous opinion, Al-Zahrani[41] in a systematic review of the literature showed that implant-supported prosthesis in peridontally well-maintained patients had survival rates of over 10 years.

Case Report

A 32-year-old, male, presented with eighteen (18) months history of mobile, mal-aligned teeth and bad breath with associated tooth loss, occasional pus discharge and esthetic concern. No history of cigarette and no family history agp otherwise healthy looking young man, bilaterally enlarged submandibular lymph nodes-firm, mobile and not tender. Intra-orally, the patient had a full complement of teeth except the lower right first premolar with fair oral hygiene and no obvious inflammation of the gingiva but suppuration around the lower incisors and the lower right first molar (Figure 1).

Fig. 1: Clinical photograph demonstrates pus on gingiva buccal to lower incisors

There was gingival recession with root exposure involving all standing teeth except the upper incisors, with the lower right canine most affected (Miller's class IV gingival recession). Subgingival deposits of calculus and periodontal attachment loss was noticed on periodontal probing, and the deepest probing depths of between 6mm and 7.5mm recorded buccally at more than three sites. Marked mesial drifting of the lower right molars, proclination of the upper right lateral incisor, migration of the other affected teeth, and extrusion of the lower left first premolar were recorded (Fig 2 and fig 3). All the teeth were mobile with grade III mobility recorded for nine teeth including two of the lower incisors.

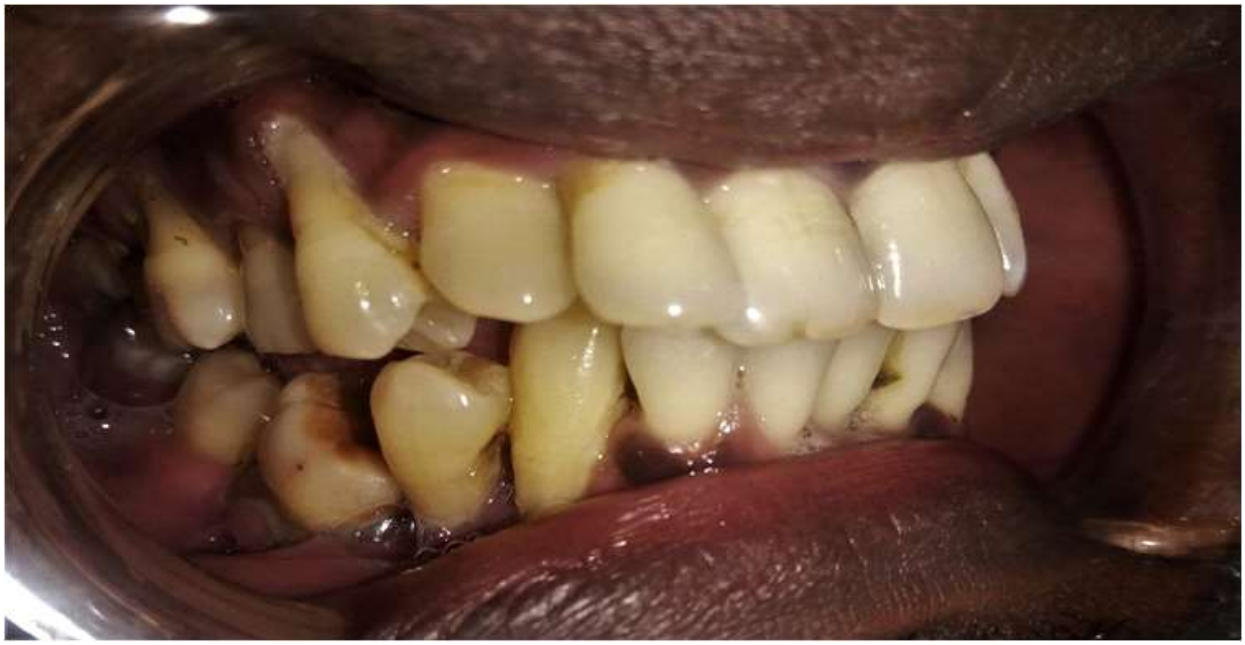

Fig. 2: Demonstrates severe gingival recession and migration

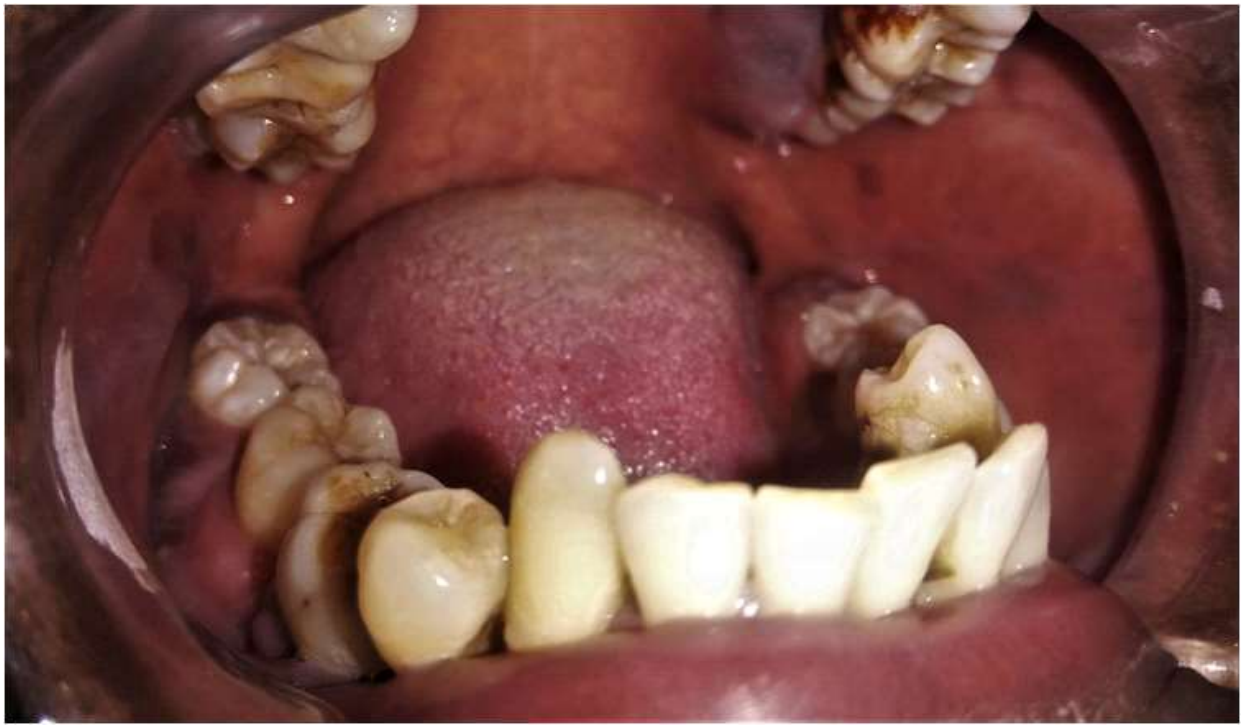

Fig. 3: Intraoral view showing supra-erupted lower right premolar and migration of the lower right posterior teeth

Radiographic examination using the orthopantomogram showed generalized distribution of alveolar bone loss which was severe around the molars, premolars and the lower canines (Figure 4). Both angular and crescent or saucer-shaped bone defects were identified on the radiograph, the angular bone loss signifying ongoing bone destruction.

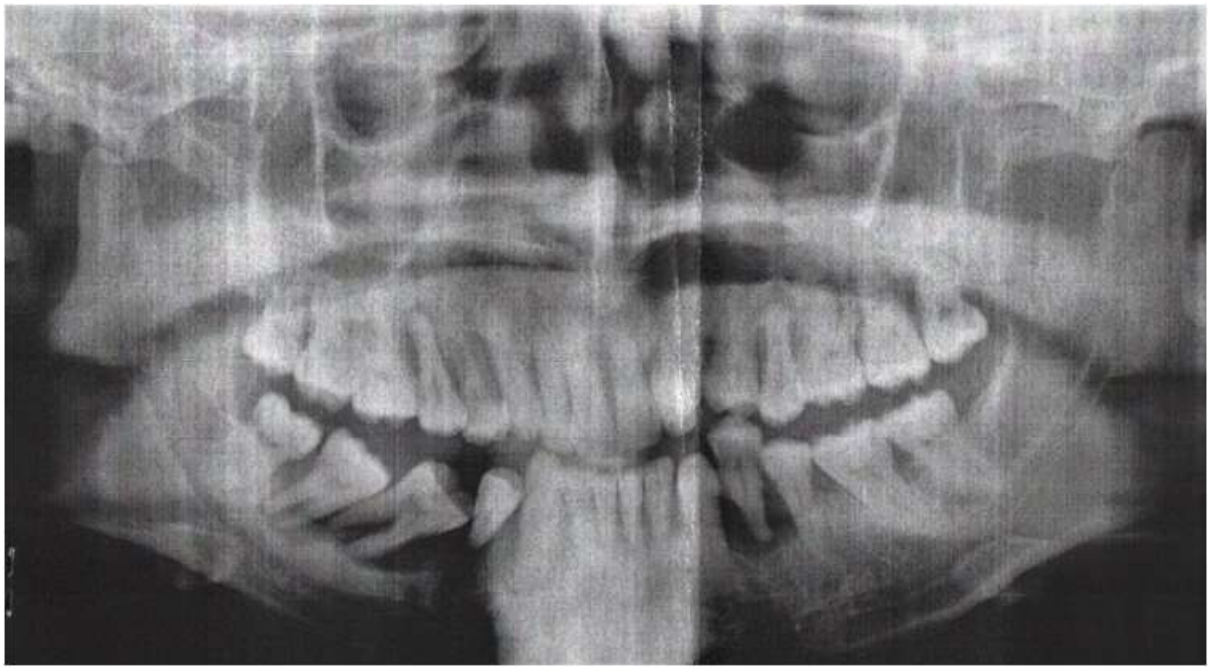

Figure 4: Orthopantomogram showing generalized severe bone loss

Routine blood investigations including full blood count and fasting blood glucose level were within normal limits.

A diagnosis of GAgP was made based on the patient's age, severity and rate of bone loss involving all the incisors, all first molars and at least three other teeth (in this case all other standing teeth), and lack of systemic disease. Patient received full mouth supra and subgingival scaling and was placed on a 21 days systemic course of oral doxycycline 100mg daily and 12 hourly mouth rinse with 0.12% chlorhexidine for 2 weeks. Patient did very well however declined the planned extraction of the worse affected teeth.

Discussion

In accordance with the 1999 recommendations of the AAP, a diagnosis of GAgP can be made based on history, intra-oral findings and radiographic features.[1] In the index case, the diagnosis was based on: the level of periodontal support destruction that has occurred in relation to the age of the patient; the absence of systemic disease; severity of periodontal attachment loss inconsistent with amount of bacteria plaque; as well as a radiographic evidence of past and ongoing alveolar bone destruction around more than three permanent teeth in addition to all the first molars and incisors. The absence of a microbiological diagnosis of the predominant bacterial species, and lack familiar aggregation did not negate the diagnosis.

The prognosis of the condition depends majorly on early diagnosis and multidisciplinary management. Due to its rapid rate of progression, it is desirable for the periodontologist to be on this team. It is however important for the general dental practitioner to be able to make an early diagnosis, as it is possible to label it as a case of CP if the age at presentation does not raise the clinician's suspicion. Early diagnosis can help to prevent progression of the disease, thereby avoiding the possibility of advanced tissue destruction and alveolar bone loss. This patient presented rather late and the prognosis for retaining most of the teeth was quite poor. It is important to have a high index of suspicion, and for early and accurate diagnosis to be

made by general dental practitioners to forestall tooth loss and other sequelae associated with the condition. The patient in the presented case had sought treatment at other dental clinics but this diagnosis (GAgP) was not made until his self-referral to our clinic. The eventual loss of his teeth, especially those severely affected will further affect him psychologically, thus making a multidisciplinary approach to management necessary. Studies have shown the negative impact of GAgP and tooth loss on the quality of life (QoL) of sufferers.[43-44] The need for psychotherapy in these patients was stressed by Dosumu *et al*.[45] as a way to help restore their ability to socialize in their environment. The goals of treatment in this patient apart from psychological rehabilitation, was to arrest the progression of the disease in an attempt to preserve the dentition in comfort and function. Part of restoring function included extractions of the worst affected teeth to be followed by prosthetic replacement after a period of periodontal maintenance. Non-surgical debridement was successfully completed by the second appointment. Patient however objected to extraction of any of the teeth, and this may possibly be the reason he failed to show up for recall visits.

Early diagnosis remains the most important determinant of a successful management outcome of GAgP. Diagnosis at a late stage with severe alveolar bone loss confer poor prognosis for retention of the dentition. In either case, an individualized oral hygiene maintenance regimen is required.

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