AGGRESSIVE PERIODONTITIS: A DIAGNOSTIC DILEMMA

Sangeeta Singh¹*, A K Shreehari²

ABSTRACT

BACKGROUND: Aggressive periodontitis (AgP) by definition is a group of rare, very destructive forms of periodontitis, usually manifests itself at an early age and shows a marked tendency to aggregate in families. A variety of factors, such as microbial, environmental and behavioral have been suggested as risk factors of Aggressive periodontitis.

METHODS: Ten cases with clinical and radiological features suggestive of Aggressive Periodontitis are discussed in this paper. In two cases, anaerobic microbial analysis was carried out and a PCR was done.

RESULTS: Out of the 10 cases, only 2 cases could be supported with a microbial evaluation. The remaining 8 cases were clinically and radiologically suggestive of Aggressive Periodontitis.

CONCLUSION: Aggressive Periodontitis is a rare but severe form of periodontitis and it is important to distinguish it from a more common form, Chronic Periodontitis. Despite well-defined criteria laid down for classifying these conditions, practical application of the same is not feasible in all cases. More research is needed to find simpler diagnostic tools to distinguish and diagnose Aggressive Periodontitis.

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INTRODUCTION

Periodontal diseases affect 5-30% of the adult population and are considered to one of the most common bacterial infections in humans \(^1\). A huge data is available from studies that have investigated the etiopathogenesis of periodontal diseases. Although a significant subset of the population appears to be susceptible to Periodontitis, there are also individuals who seem to be more resistant to more severe form of disease \(^2\). The criteria considered for distinguishing chronic and aggressive forms of Periodontitis initially focuses on the amount and pattern of periodontal destruction, patients age & medical status. Aggressive Periodontitis is characterized by a significant destruction of the attachment apparatus in a short span of time and commonly occurs at a young age. Severe destruction occurs at an early age either due to expression of highly virulent causative organisms or high levels of susceptibility or a combination of both.

The diagnosis of Aggressive Periodontitis is difficult to make and often challenging. The 1999 world workshop of AAP introduced the term ‘Aggressive Periodontitis’ and presented the criteria for diagnosis of this disease. However the practical application of the same is difficult and most of the times we come across cases which appear clinically as AgP but do not fit into the laid down criteria.

This study was carried out with the aim to evaluate the efficacy of current diagnostic criteria for Aggressive Periodontitis and the objectives of the study were to evaluate the clinical, radiological, microbiological & molecular diagnostic criteria for Aggressive periodontitis in 10 cases of Periodontitis.

MATERIALS & METHODS:

All the cases discussed here were informed about the records and need for diagnostic analysis. An informed consent was obtained. The necessary clearance was taken from the ethical committee of the institute i.e Armed Forces Medical College, Pune. The cases were evaluated clinically and radiographically for the diagnostic features \(^3\). The primary and secondary criteria for diagnosis of Aggressive Periodontitis were evaluated \(^4\).

The primary criteria as described by Lang et al were (a) Rapid attachment loss, (b) Negative medical history and (c) Positive family history. The secondary criteria considered were: (a) Inconsistency of the low amounts of present etiological factors (i.e., plaque) and the observed inflammation (b) Strong colonization by...
Aggregatibacter actinomycetemcomitans and, in some populations, Porphyromonas gingivalis

(c) Immunological differences that do not entail the diagnosis of periodontitis as a manifestation of systemic disease (d) Hyper responsive macrophages (e) Abnormalities of neutrophil function. To evaluate these, clinical periodontal charting was done. An OPG was taken. It was not feasible to carry out any further investigations in most of the cases due to non-availability of laboratory. In two cases, which reported to our college, aerobic & anaerobic culturing was carried out. The anerobic culturing was done using the selective media, Dentaid [5]. Fig 1,2. Gingival crevicular fluid (GCF) samples were collected for microbiological analysis. A multiplex polymerase chain reaction was put up using primers for three main periodontal pathogens; Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis & Tannerella forsythensis on GCF samples [Fig 3].

RESULTS:

All the 10 cases were diagnosed as Aggressive Periodontitis, out of which 2 were cases of Localized Aggressive Periodontitis (LAP) and 8 were Generalized Aggressive Periodontitis (GAP) [Fig 4 – 10]. In the cases where microbiological analysis was carried out, presence of Aa could be ascertained using anaerobic culture. The PCR was positive for Pg and Tf, but Aa was not detected. The arc shaped pattern is a classic feature of Generalised aggressive periodontitis. However, in Localised aggressive periodontitis, only the involved teeth show a vertical bone loss which is evident in the cases discussed. The radiological features are one of the secondary features of this disease. A summary of the clinical, radiological and microbiological features of all the cases is presented in Table 1.

DISCUSSION:

Aggressive periodontitis (AgP) is a rare, destructive disease which is often accompanied by severe and rapid loss of periodontal attachment and may be more common in children and adolescents. In young individuals, the onset of these diseases is often circumpubertal. The primary features of AgP include a history of rapid attachment and bone loss with familial aggregation. Reported estimates of the prevalence of Aggressive Periodontitis forms in geographically diverse adolescent populations range from 0.1 to 15% [6].
<table>
<thead>
<tr>
<th>Case no</th>
<th>Age/Sex</th>
<th>Family history</th>
<th>Medical history</th>
<th>Local factors</th>
<th>Bone loss</th>
<th>Teeth involved</th>
<th>Anerobic culture</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39/M</td>
<td>Sibling</td>
<td>Negative</td>
<td>Minimal</td>
<td>Angular</td>
<td>27,46,36</td>
<td>Positive</td>
</tr>
<tr>
<td>2</td>
<td>34/M</td>
<td>Unknown</td>
<td>Negative</td>
<td>Minimal</td>
<td>Angular</td>
<td>17,26,27,37,46,47</td>
<td>Positive</td>
</tr>
<tr>
<td>3</td>
<td>38/M</td>
<td>Unknown</td>
<td>Negative</td>
<td>Present</td>
<td>Angular</td>
<td>Most</td>
<td>Not done</td>
</tr>
<tr>
<td>4</td>
<td>42/M</td>
<td>Mother, One sibling</td>
<td>Negative</td>
<td>Present</td>
<td>Angular</td>
<td>Most</td>
<td>Not done</td>
</tr>
<tr>
<td>5</td>
<td>32/M</td>
<td>Not Clear</td>
<td>Negative</td>
<td>Abundant</td>
<td>Horizontal &amp; Angular</td>
<td>Most</td>
<td>Not Done</td>
</tr>
<tr>
<td>6</td>
<td>30/M</td>
<td>Sibling</td>
<td>Negative</td>
<td>Minimal</td>
<td>Angular</td>
<td>25,46,36</td>
<td>Not done</td>
</tr>
<tr>
<td>7</td>
<td>34/M</td>
<td>Mother</td>
<td>Negative</td>
<td>Present</td>
<td>Angular</td>
<td>Most</td>
<td>Not done</td>
</tr>
<tr>
<td>8</td>
<td>36/M</td>
<td>Not Clear</td>
<td>Negative</td>
<td>Abundant</td>
<td>Angular</td>
<td>Most</td>
<td>Not Done</td>
</tr>
<tr>
<td>9</td>
<td>31/M</td>
<td>Unknown</td>
<td>Negative</td>
<td>Minimal</td>
<td>Angular</td>
<td>Most</td>
<td>Not done</td>
</tr>
<tr>
<td>10</td>
<td>43/M</td>
<td>Sibling</td>
<td>Negative</td>
<td>Present</td>
<td>Angular</td>
<td>Most</td>
<td>Not done</td>
</tr>
</tbody>
</table>

Table 1: Summary of primary & secondary criteria in the 10 cases

Fig 1: Case1 diagnosed based on clinical, Microbiological & Radiographic picture

Fig 2: Microbiological analysis: Anerobic culture using Dentaid medium (Selective for Aa)
Fig 3: Multiplex PCR put up for Aa, Pg & Tf

Fig 4: Case2: Clinical & Radiographic evaluation

Fig 5: Case3: Clinical & Radiographic evaluation

Fig 6: Case4: Clinical & Radiographic evaluation

Fig 7: Case5 Clinical evaluation

Fig 8: Case 6

Fig 9: Case 7

Fig 9: Case 8

Fig 10: Case 9, 10
“For surveillance purposes, there seems to be no reason for separating Chronic & Aggressive Periodontitis” [7]. Page made this statement in the year 2007 where the authors have summarized the case definition for population-based surveillance. According to them, for surveillance alone there is no need to differentiate between Chronic and Aggressive Periodontitis.

So, what lies in a name? Though rare, this condition needs to be diagnosed and distinguished from the more common Chronic Periodontitis. The need for this is emphasized since the approach as well as the line of treatment differs significantly in both the conditions. There is a distinct pattern of bone loss with typical arc like bone loss pattern in GAP and isolated vertical osseous defects in LAP. The bone loss pattern in Chronic Periodontitis is more or less horizontal. Based on the understanding of the destruction pattern, treatment differs in these cases. Debridement alone is sufficient in most of the cases of Chronic Periodontitis whereas the defects commonly associated with both LAP and GAP requires regenerative osseous surgery. Vertical bone loss is a common feature of Aggressive periodontitis and not Chronic periodontitis. Only in presence of trauma from occlusion the bone loss pattern becomes vertical in cases of Chronic periodontitis. The flora is more pathogenic and penetrates the soft tissues in cases of Aggressive Periodontitis and therefore antimicrobial therapy is an essential adjunct in all cases of AgP. But in Chronic Periodontitis unless it is the severe form, antimicrobial therapy is not required [8,9]. The underlying mechanism of periodontal disease initiation and progression is still being researched around the world despite significant advances in the field of periodontology with focused research on biomarkers, introduction of chairside diagnostic kits, The World workshop of Periodontics, 1999 established and defined the criteria for classification of periodontal disease. Lang further established the primary and secondary diagnostic criteria for Aggressive Periodontitis. However the practical application of these criteria in day-to-day practice is not always feasible. In such cases, it becomes difficult to diagnose the cases based on these defined criteria. In the cases presented, the microbial and molecular methods helped in supporting the diagnosis in two cases. This was possible since the cases reported to AFMC, Pune where the latest state of art equipment for microbiological and molecular studies are available. However it is not always feasible
to carry out these studies in every dental centre especially the ones located in peripheries where resources are limited and practical difficulties limit the scope of investigations. In such cases, one has to rely on clinical and radiological investigations, alone which are not sufficient at times for a definite diagnosis of AgP.

One of the most active areas of periodontal research is concerned with the search of diagnostic tests of periodontal disease activity [10]. Potential biomarkers of disease activity would need to undergo extensive and careful basic research investigation before undergoing clinical evaluation. Once these biomarkers can be definitely proven to be linked with different forms of Periodontitis, chairside diagnosis of AgP can become a reality in the future. However till such time we have to rely on the clinical presentation of the patient along with radiographic evaluation of the osseous architecture, microbial and molecular analysis wherever feasible and last but not the least on our past clinical experience with similar cases. Once diagnosed we need to formulate a definite treatment plan taking into consideration the rapid rate of progression of destruction in Aggressive Periodontitis. This will not only ensure timely intervention to control the disease but also considering the tendency for familial aggregation [11]. In such cases patients can be motivated to get the family members evaluated so that if the disease process has started it can be timely controlled before significant damage occurs. Aggressive periodontitis though rare, when encountered in clinical practice, it poses a challenge since the criteria defined in literature do not necessarily apply in all such cases. This article discusses these problems and the need to define guidelines, which are more practical and can be used in areas where advanced investigative facilities are unavailable. The cases have been presented to put forth clinical experiences and difficulties when such challenging cases are seen in peripheral locations where diagnostic facilities are minimal and one has to rely on previous experiences with a few seen cases. Global authorities lay down the diagnostic criteria and individual clinicians can only put forth challenges with the same. The AAP task force is working on the revision of classification and hopefully will define the guidelines [12].

CONCLUSION:

Aggressive Periodontitis continues to be an enigma as far as diagnosis and formulating a definite treatment plan is concerned. In future, development and application of rapid and simple diagnostic tests based on host salivary and immune factors may help in early detection, timely intervention, decrease the need for more aggressive form of therapy and improve the response to therapy.
Declaration of Conflicting Interests:

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES:


