Aggressive Periodontitis–Multifactorial Disease: A Review

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Abstract

Aggressive periodontitis is periodontal destruction that becomes clinically significant around adolescence or early adulthood and whose phenotype is determined by genetic and environmental influences on the affected individuals. Actinobacillus actinomycetemcomitans is considered a major etiologic agent of aggressive periodontitis. About 45% of the adult population in India has periodontitis.

The purpose of this review is to highlight the current etiological and therapeutic concepts of aggressive periodontitis. We need advanced diagnostic techniques to learn about current disease activity and rate of progression. We also require strategies to keep the disease under control with proper maintenance regime and prevent tooth loss, because it can result into complicated prosthetic rehabilitation in a very young patient. This paper also reviews clinical, microbiological, immunological, and genetic aspects of pathogenesis of aggressive periodontitis, as well as diagnostic criteria of the disease.

Key words : Aggressive periodontitis, Prevalence, Actinobacillus actinomycetemcomitans.

Introduction

Periodontitis is inflammation of the periodontium that extends beyond the gingiva and produces destruction of the connective tissue attachment of the teeth. It is no longer considered a single disease and is now considered to exist in three primary forms: Chronic, Aggressive, and as a Manifestation of systemic diseases[1]. Aggregatibacter actinomycetemcomitans is considered a major etiologic agent of aggressive periodontitis (AgP). Other periodontopathic bacteria such as Porphyromonas gingivalis are also suspected of participating in aggressive periodontitis although the evidence to support this is controversial[2].

Aggressive periodontitis (formerly known as early onset periodontitis) is periodontal destruction that becomes clinically significant around adolescence or early adulthood. The disease has been classified into two types: Localized and Generalized. Other terms found in the literature that have been used to describe aggressive forms of periodontitis include juvenile, localized juvenile, generalized juvenile, rapidly progressive, severe, and prepubertal periodontitis. In a national survey of the oral health of U.S. school children, three case definitions were used for aggressive periodontitis, as follows:

i. Localized aggressive periodontitis: At least one first molar and at least one incisor or second molar and two or fewer canines or premolars had >3 mm of attachment loss.
ii. Generalized aggressive periodontitis: Criteria for localized disease were not met, four or more teeth had >3 mm of attachment loss, and at least 2 affected teeth were second molars, canines, or premolars[3].

Epidemiology of Aggressive Periodontitis

Most studies from the U.S. and other countries that have examined localized aggressive periodontitis in adolescents have reported prevalence estimates below 1%. In the U.S., the prevalence of localized and generalized aggressive periodontitis were estimated to be 0.53% and 0.13% respectively, another 1.61% of the adolescents had incidental attachment loss-(attachment loss that did not fit the study's case definitions for localized or generalized disease). Although these prevalence estimates are small, together they represented almost 300,000 U.S. adolescents at the time of the study (1986-87)[4].

Estimates of the prevalence of juvenile periodontitis vary greatly. Recent epidemiological studies of Nordic populations reported prevalence rates of 0.2% for Norway, 0.1% for Finland and 0.1% for Denmark[5]. Two studies of populations in Nigeria reported prevalence rates of 0.75% and 0.8% in children 12 to 20 years old. A prevalence of 3.7% was estimated for children 15-16 years old in Brazil[6].

Cianciola et al reported juvenile periodontitis among 9.8% of insulin-dependent diabetes mellitus patients and 1.7% of non-diabetic controls at an outpatient dental clinic of a large children's hospital[7]. Very few longitudinal studies conducted in the United Kingdom followed 167 subjects from ages 14 to 19. During the 5 years of follow-up, the percentage of subjects with attachment loss on one or more of the examined teeth increased dramatically from 3% to 77% for attachment loss of >1 mm and from 0% to 14% for attachment loss of >2 mm. By age 19, 31% of the sites examined had attachment loss of >1 mm, and 3.1% had attachment loss of >2 mm. The teeth most commonly affected were the maxillary first molars and the mandibular central incisors7. A review of the available longitudinal studies of periodontitis in childhood and adolescence concluded that subjects with signs of periodontal disease at a young age are likely to have further periodontal deterioration. Progression is more extensive at initially infected sites and among subjects of low socioeconomic status[8].

Why Do Patients Have Aggressive Periodontitis, and What Puts Them at Risk?

Microbiologic etiologic factors

Aggressive periodontitis is an infectious disease and its pathogenesis is somewhat similar to that of chronic periodontitis[9].

The search for the microbial causal agent(s) has endured for decades, and it remains inconclusive. Recent advances in microbiologic detection methods and genome mapping have considerably increased our knowledge of the composition of sub gingival biofilms in health and in various forms of periodontal diseases. Previous findings that Aggregatibacter actinomycetemcomitans plays a primary role in the pathogenesis of all types of aggressive periodontitis may not be supported by more recent findings from studies using more sensitive molecular microbiologic methodologies. The current understanding is that aggressive periodontitis contains a mixed microbial flora, with a wide heterogeneity in the types and proportions of microorganisms recovered from patients with the disease, and there are significant differences between different geographic regions and ethnicities[10].

There is evidence that the A. actinomycetemycomitans – JP2 clone may play an important role in the development of aggressive periodontitis[11]. The role of novel and not-yet-culturable bacteria in the pathogenesis of aggressive periodontitis is not known. It has also been hypothesized that herpes-viruses, including Epstein-Barr virus and cytomegalovirus, may impair the local host defenses, and hence may increase the aggressiveness of periodontopathic bacteria and thereby contribute to the etiology of aggressive periodontitis .[12]

Host response

The host response to microbial infection plays an important role in the susceptibility to aggressive periodontitis. To that extent, a prevailing view has been that chemotactic defects in neutrophils are a major etiologic factor in the Etiopathogenesis of this disease. However, a critical review of the significance of host responses in aggressive periodontitis suggests that neutrophil dysfunction is not a major cause of this disease in non-syndromic individuals[13].

In a competent host, the innate and inflammatory immune systems provide early responses and contribute to protect the host from potentially pathogenic microorganisms. In contrast, the adaptive immune system includes the humoral and cellular immune responses, and it plays a role during the later stages of infection. The type of immune response mounted by the host to infection will depend on the type of microorganisms, and because the microbial infection in aggressive periodontitis is a mixed infection, the immune response may be complex and involve multiple mechanisms. Diagnostic methods of this disease, based on a specific microbiologic or immunologic profile, currently do not exist.[14]

Genetic risk factors

Cases of aggressive periodontitis tend to show a familiar pattern, and this suggests a role for genetic factors in the pathogenesis of this disease[15].

Syndromic periodontal diseases are associated with monogenic diseases, and the genetic bases for these diseases have been identified. However, nonsyndromic periodontal diseases seem to have different, more complex, genetic mechanisms. Insofar, available date suggest that aggressive periodontitis is caused either by mutations in a few major genes or by multiple small- effect genes, and there is also evidence of gene- gene and gene-environment interaction effects. The genetic association study approach is useful for the identification of genetic variants that could affect susceptibility to complex diseases, such as aggressive periodontitis. Most association studies have focused on the abnormal effects of various gene polymorphisms on host responses, including the chemo taxis and functions of neutrophils, chemotaxis and inflammatory mediators, and on other genes, such as the vitamin D receptor gene. Many of these studies have yielded inconsistent findings. This may be attributed to multiple reasons, including relatively small study samples, inadequate phenotypic characterization of cases and controls, and variability of the genetic profiles among different populations. In addition, as most studies focuses on only one or a few genes, the study design may not have been adequately powered to detect potentially small effects. Most recent genetic studies have documented father-to- son trans-missions of aggressive periodontitis, which is not consistent with the X- linked mode of transmission that was previously the accepted mode of inheritance of the disease. Rather, an autosomal mode of inheritance is now considered more valid. Genetic markers have the potential to be implemented as screening tools to identify subjects at risk for developing aggressive periodontitis. This approach may significantly enhance treatment outcome through the early detection and treatment of patients, as well as using future approaches based on gene therapy.[16]

Conclusion

The prevalence of aggressive perio-dontitis is much lower than chronic periodontitis, the management of aggressive periodontitis is more challenging compared to that of chronic periodontitis because of its multiple risk factors. Further understanding of the etiology, risk factors, pathogenesis, and host immune response in aggressive periodontitis along with advances in regenerative concepts, tissue engineering, and gene therapy is needed for formulating better management protocols in the treatment of generalized aggressive periodontitis.

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