From Alveolar Diffuse Atrophy to Aggressive Periodontitis: A Brief History

Esra Guzeldemir, DDS, PhD* and Dr. Hilal Ulu Uygur*

*From the Baskent University, Faculty of Dentistry, Department of Periodontology
Ankara, Turkey

Technologic advances in mechanics, electronics, physics, chemistry, and computer science have contributed to advances in dental medicine. Periodontology is not only a clinical science but is also directly related to the basic sciences. Research is conducted in laboratories rather than in clinics now. During the last century, aggressive periodontitis has received attention from numerous researchers because of its multifactorial features. This paper explores the long scientific journey of aggressive periodontitis, beginning with its first definition as alveolar diffuse atrophy. Perhaps in the future, “alveolar diffuse atrophy” will be referred to by another name or term. However this journey will never end.

Aggressive periodontitis (AgP) is a complex multifactorial periodontal disease, its progression and severity of which are determined by immunologic, microbiologic, environmental, and genetic risk factors: age, sex, and race and by the interactions of those elements. Although the clinical and radiological characteristics of AgP have been clearly defined, its genetic and immunologic features remain unclear.

As in all research fields, dental science evolves by means of technologic advances and improved instrumentation. Advances in technology made over the last 5 decades have contributed to the improvement in all dental sciences. A review of the English-language literature reveals only case reports of AgP from the 1920s to the 1940s. The first reports of AgP comprise only the clinical aspects of the disease, radiographic evaluations, and predictions about progression rather than etiologic factors and treatment options. In 1923, Gottlieb described a 22-year-old man with a fatal case of influenza as having “diffuse atrophy of alveolar bone”, a condition thought to be a manifestation of a systemic disease. However, that case report was the first to present AgP, the pathologic mechanism of which is described as an enlargement of the periodontium resulting from severe alveolar bone loss and the destruction of collagen fibrils in the periodontal ligament. No gingival involvement was observed in the patient described, a finding that proved to be a distinctive feature of the diagnosis.

In 1928, Gottlieb realized that AgP was characterized by the destruction of continuous cement formation, which resulted in disorganized and irregular periodontal ligament fibrils. He referred to the pathologic process as “deep cementopathy” and thought that alveolar bone resorption and periodontal pocketing occurred as a result of a physiologic response to a
foreign substance (root cement).17

Wangensteen and associates established that inflammation and pocket formation occurred even if the gingival form and shape appeared healthy. That observation was the first accurate description of AgP according to Saxen.

Thoma and Goldman contributed new information to the description of AgP in 1940 when they described a condition called "periodontitis," the primary characteristic of which are the pathologic migration of auxiliary incisors and the pathologic eruption of teeth. One year later, in 1941, Miller and colleagues evaluated 35 individuals who ranged in age from 14 to 30 years. Those investigators endeavored to establish the relationship of systemic factors to the pathogenesis of periodontitis. To that end, various laboratory analyses were performed. Periodontitis was described as "occurring at an early age and causing severe alveolar bone destruction."

"Periodontosis" was the term used by Urban and Weinmann in 1942 to describe a degenerative (rather than inflammatory) disease that usually affects females aged from 10 to 25 years. According to those authors, periodontosis is characterized by 3 histopathologic stages.

In 1950, the American Academy of Periodontology (AAP) published a dictionary of terms in which periodontosis was described as a secondary epithelial proliferation with or without the presence of a secondary gingival disease. The definitions further states that periodontitis is characterized by the pathologic migration of teeth and tooth loss, that it arises from one or more periodontal tissues, and that it causes the degenerative non-inflammatory destruction of the periodontium. "Periodontosis" was the term used to refer to the inflammatory involvement of the periodontium.

In 1952, McClain postulated that hypoplastic first molars and incisors are a result of a serious disease or vitamin D deficiency in early childhood. He further stated that because these hypoplastic teeth erupt into the oral cavity, they are exposed to collagenous tissues to a greater degree than are other teeth, a process during which alveolar bone becomes progressively thinner.

In 1953, Glickman evaluated changes in radiographic images that are summarized as follows: enlargement of the periodontal space, the discontinuity or absence of lamina, vertical bone loss, and malformation of the tubercular bone.

The findings of Thoma and Goldman were supported in 1956 by Yount and Belting, who described the clinical appearance of the disease in detail. Yount and Belting reported no periodontal pocket formation, even in the presence of vertical bone loss. The clinical characteristics of the disease were described as pink, tight gingival tissue and a maximal amount of calculus. Even when periodontal pockets were present, the gingiva remained pink, and the accumulation of calculus might have been caused by pus formation. Usually, no cavities were seen in these patients. In the World Workshop in Periodontics in 1966, "periodontosis" was excluded from periodontal terms.

The term "juvenile periodontitis" (JP) was used in the French literature by Chaptut and colleagues in 1969, and also in the English literature in 1969 by Butler. Bae, however, referred to JP as "periodontosis," a disease of the periodontium that is characterized by the destruction of alveolar bone in more than 1 tooth in otherwise healthy young adults. Bae described 2 different types of progressive periodontosis, one limited to molars and incisors, and the other a generalized form of the disease. Most of the teeth are involved in patients with this disorder. It was not possible to attribute the excessive destruction noted to the effects of local irritants. Bae classified periodontosis by its clinical features. He noted that periodontosis was diagnosed in young individuals but did not affect the primary dentition (especially in females), that the pattern of alveolar bone loss differed from other types of alveolar bone loss on radiographic examination, that consanguinity was often a feature of the medical history of patients with periodontosis, that local etologic factors did not cause deep periodontal pockets, and that the disease was progressive. Bae suggested that the term "periodontosis" was more accurate than "juvenile periodontitis," 1979.

In 1977, Socransky and Bae described types of periodontosis that is characterized by generalized, acute, and rapid progression.

In a case series by Fournel in 1972, the term "periodontal syndrome" was used. Fournel claimed that periodontal syndrome did not differ in men and women, and he reported an autosomal recessive genic transmission of that disorder. Regression analysis in family studies revealed that the X chromosome is responsible for this transmission. In contrast to the theory of Fournel, the transmission of periodontal syndrome has also been defined as being autosomal dominant (Metcalf and colleagues, 1979). In 1979, Hordmand and Frandsen reported on 156 individuals with JP. Those investigators classified JP by the type of alveolar bone loss. First molars and/or incisors were involved in type I JP, and fewer than 14 teeth were involved in type II. Type III, the generalized form of the disease, involved 14 or more teeth.

Over the years, JP has attracted the attention of numerous authors. In the 1980s, the clinical consensus
characterized JP as a degenerative and non-inflammatory disease. Page and Baal (1985) reported that JP is limited to certain regions of its regional specificity. They theorized that the discontinuity of the abnormal deposition of root cement in certain areas of dentition enables microbial invasion and results in destructive periodontal attachment and the destruction of periodontal tissues without inflammation. Genc defined JP as a destructive disease of two different forms: either localized or generalized.

It is important to have a common scientific language for international communication in science. For this purpose, the First European Workshop in Periodontology and the World Workshop in Clinical Periodontics were organized, and during those events, periodontal disease classification was discussed. JP was evaluated under the heading of "early onset periodontitis" that is localized or generalized. The knowledge of the pathogenesis, inflammation, and disease progression of JP that was summarized from the 1960s to the 1980s was used to establish that classification. However, in the 1990s researchers realized that there were some missing points in the classification of periodontal disease. First in the European Workshop on Periodontology in 1993 and then in 1996 under the supervision of American Academy of Periodontology (AAP), those emissions were discussed, and another classification was accepted during the AAP Workshop for the International Classification of Periodontal Diseases in 1999. The term "juvenile periodontitis" was changed to "aggressive periodontitis" to exclude age as a criteria for diagnosis and to emphasize the aggressive destruction caused by the disease.

In 2002, two types of AgP were described in a review by Mombelli and colleagues. The clinical diagnoses were termed "secure AgP": "insecure AgP" or "uncertain AgP" according to a re-evaluation of previously published articles.

The etiology of periodontal diseases was initiated in the golden age of microbiology (1880-1920). Over the years, hundreds of papers were published which contributed to define the developing of periodontal diseases. Hypothesis of "diffuse alveolar atrophy" that basically defined the description of the infectious nature of the disease and then "trauma from occlusion and some combination of these factors, such as smoking, bacterial invasion, medically compromised patients, were thought to be affected of the disease progression. During the last era, the concepts regarding the microbiological etiology of the periodontal disease were changed. Around the seventies, researchers demonstrated that microorganisms obtained from lesion sides of JP adult periodontitis and healthy individuals differed from each other. In early studies, culture techniques were usually used for identification of susceptible bacteria. The World Workshop in Periodontology (1996) designated A. Actinomycetemcomitans (a.a.), P. gingivalis and B. forsythus as periodontal pathogens. In these days, an immunological response was suggested that has a value in defining periodontal pathogens. Technical developments such as checkerboard DNA-DNA hybridization, DNA probe, PCR and FLP allow to assessment of specific microorganisms, by this way risk of periodontal disease progression, presence of an organism could be assessed. Aa was found strongly associated with LAgP. Aa has the ability to invade human gingival epithelial cells, human vascular endothelial cells and induce apoptotic cell death. Elevated levels of antibody in sera and saliva were detected in LAgP patients. The acceptance of the key of Aa in LAgP is allowed to determine possibilities of the treatment and prevention from LAgP.

Discussion

The goals of this review were to demonstrate the difficulties in classifying a disease while acknowledging its most essential aspects and to emphasize those efforts to classify diseases more accurately will never end. After World War II, medical and dental research accelerated. Technologic advances in mechanics, electronics, physics, chemistry, and computer science have contributed to advances in dental medicine. Periodontology is not only a clinical science but is also directly related to the basic sciences. Research is conducted in laboratories rather than in clinics now. Technical advancements in microbiology and immunology will change the perspectives in prevention and treatment of the AgP.

Perhaps in the future, "alveolar diffuse atrophy" will be referred to by another name or term, or perhaps that disease will diagnosed in utero and will be controlled before birth. Genetic research may allow gene therapy in practice. Perhaps advances in nanotechnology will play a major role in the diagnosis and treatment of periodontal diseases. Perhaps, researchers will use techniques as yet unknown to detect early symptoms of disease and destroy diseased cells without injury to healthy cells. Perhaps aggressive periodontitis will no longer exist with intervention and changing the activities of polymorphonuclear leukocytes.

Many researchers have devoted their life to such purposes. Ideally, each scientific contribution would result in a better quality of life for every individual and a less expense for governments. The whole is indeed the sum of its parts.
References