

A CASE REPORT ON AGGRESSIVE PERIODONTITIS

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Abstract

Aggressive periodontitis which is an uncommon condition is characterized by severe loss of attachment and destruction of alveolar bone around one or more permanent teeth during the period of pubescence. Aggressive periodontitis have localized and generalized forms. It is currently believed that combination of bacteriologic, immunologic and hereditary factors are of major importance in the etiology of this disease. The case was of a 12 - year old male and his clinical and radiographic findings were typical for generalized aggressive periodontitis. Treatment consisted of thorough training in techniques of plaque control, scaling and root planning and administration of tetracycline 250 mg every six hours for three weeks, as well as combined surgery and antibiotic therapy. But both failed to cure or control the disease mostly because of the non-cooperation with the oral hygiene instructions.

Keywords: Localized Aggressive Periodontitis, Periodontitis, Generalized Aggressive Periodontitis, Radiographic Findings

1. Introduction:

Aggressive Periodontitis is an uncommon condition characterized by severe loss of attachment and destruction of alveolar bone around one or more permanent teeth in otherwise healthy adolescent. The disease has a predilection for first molars and incisors and when limited to these teeth is termed localized aggressive periodontitis. A generalized form of aggressive periodontitis has been described in which there is severe tissue destruction around many teeth. The generalized form may be preceded by localized aggressive periodontitis or arise spontaneously. Localized aggressive periodontitis was described by Gottlieb as a chronic, degenerative, non-inflammatory disease of the periodontal tissues, which he referred to as "diffuse atrophy of alveolar bone". Aggressive periodontitis becomes apparent about the time of puberty, usually between the ages of 10 and 15. The disease progresses rapidly at the mesial or distal surfaces of one or more first permanent molars or distal surfaces of one or more first permanent molars, and in most instances there is additional involvement of one or more incisors¹. As the disease progresses, the affected teeth may become increasingly mobile, with labial movement and spacing of incisors. Bleeding on probing of the periodontal pockets is also evident, reflecting ulceration of the cervical epithelium. Histological examination shows numerous areas of chronic inflammation

containing poly-morph nuclear leukocytes, lymphocytes and large numbers of plasma cells. Unlike ordinary periodontitis which usually progresses at a slow rate, aggressive periodontitis progresses rapidly. And this destructive form of periodontal disease frequently remain undetected in young individuals, until increased tooth mobility, drifting, and spacing of teeth, abscess formation occurs. Christersson suggest that the therapy should be directed at the total elimination of A. actinomycetemcomitans from the subgingival and supragingival plaque, from the subgingival microflora and from the periodontal soft tissues². They suggest closed curettage and surgical curettage in conjunction with scaling and root planning. Adjunctive antibiotic treatment of periodontally diseased patients is beneficial. One gram a day of systemically administered tetracycline produces a crevicular fluid concentration which is 10 to 20 times that of the blood. This concentration is effective against most of the periodontally pathogenic organisms³.

2. Case History:

A 12 - year old male is discussed here. The patient's medical history was unremarkable. The clinical features are typical of aggressive periodontitis in its most active stage of progression. Both the attached and marginal gingival were fiery red, acutely inflamed. Pus

was oozing from around many teeth. In spite of this, the patient had no carious lesions and no restorations. The radiographs revealed almost total loss of the alveolar bone of Radiographic measurements indicated that all of the teeth were affected. The mean pocket depth was about 7 mm. Complete course of laboratory tests, including glucose tolerance, urinalysis and routine blood tests were normal. Culture for *Actinobacillus actinomycetemcomitans* was negative. Neutrophil chemotaxis was normal but monocyte chemotaxis was significantly suppressed. Treatment consisted of through training in techniques of plaque control scaling and root planning and administration of tetracycline 250 mg every six hours for 3 weeks. The response to treatment was not good, and mobility could not be controlled. So were extracted. Following the completion of this phase of treatment, the patient was placed on recall, but he failed to reappear. He was not seen again until approximately 4 months later, at which time it was noted that he had ceased tooth brushing and disease was again active. Oral hygiene was poor and mobility was present. Hemorrhage had occurred from the granulation like tissue. Soft tissue specimen adjacent to the teeth sent for histopathological examination with the provisional diagnosis of Aggressive periodontitis. Microscopic description was Chronic, extensive inflammatory condition with numerous plasma cells, covered by non-keratinizing squamous, hyperplastic, pseudoepithelial hyperplasia. Flap curettage was done around all remaining teeth. 250 mg tetracycline 4 times daily for 2 weeks administered again. Partial denture fitted to replace the missing teeth.

3. Discussion:

During the period of pubescence, the most common form of periodontal disease is an inflammatory hyperplastic gingivitis associated with poor oral hygiene, plaque and supragingival calculus. Untreated, the disease usually progresses into a periodontitis. In the vast majority of these latter cases, both supra and subgingival calculus is present. This is easily discernible clinically and in roentgenographs. In pockets which are greater than 6 mm. In depth, subgingival calculus is invariably present. In aggressive periodontitis however, the gingiva in the early stages, is most frequently normal in appearance^{4, 5, 6}. Patients, whose oral hygiene is

poor, have obvious plaque and supragingival calculus. In these instances gingival inflammation is present. It is also common to see some signs of gingival inflammation clinically in the very advanced cases. In the vast majority of cases of aggressive periodontitis, however, one is left with the clinical impression that the amount of periodontal destruction observed is not commensurate with the amount of local irritants which can be found⁷. Vertical loss of alveolar bone about the first molars and one or more incisor teeth in an otherwise healthy adolescent is a diagnostic sign of periodontosis. The pattern of bone loss is usually described as an arch-shaped loss of alveolar bone extending from the distal surface of the second bicuspid to the mesial surface of the second molar. The bone loss in the posterior regions occurs bilaterally and the right and left sides are generally mirror images of each other. The degree and the shape of the bone loss are generally dependent upon whether the lesion is diagnosed in an early or advanced stage⁸. As the terminal stage of the disease, the bone loss is no longer vertical in nature but it assumes a horizontal shape. In the case presented, all these clinical and radiographic signs were present. Antibiotic therapy with scaling and root planning as well as surgery and antibiotic therapy was performed. But both failed to cure or control to disease, mostly because of the non-cooperation with the oral hygiene instructions^{9, 10}.

Conclusion:

Although various advanced diagnostic and treatment modalities have emerged in the management of aggressive periodontitis, the conventional techniques retain their popularity to date. A case of aggressive periodontitis was reported which was diagnosed clinically and radiographically and treated accordingly.

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