# JUVENILE PERIODONTITIS *(A CASE REPORT)*

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*SUMMARY: In this article, juvenile periodontitis, which is an uncommon condition, characterized by severe loss of attachment and destruction of alveolar bone around one or more permanent teeth during the period of pubescence, was discussed with a case report. Juvenile periodontitis have localized and generalized forms. It is currently believed that a combination of bacteriologic, immunologic and hereditary factors are of major importance in the etiology of this disease. Our case was a 10 - year old female and her clinical and radiographic findings were typical for generalized juvenile periodontitis. Treatment consisted of thorough training in techniques of plaque control, scaling and root planing 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.*

*Key Words: Generalized juvenile periodontitis, localized juvenile periodontitis, periodontitis.*

## INTRODUCTION

Juvenile 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 juvenile periodontitis (1). A generalized form of juvenile periodontitis has been described in which there is severe tissue destruction around many teeth. The generalized form may be preceded by localized juvenile peridontitis or arise spontaneously (10).

Localized juvenile periodontitis was described by Gottlieb (10) as a chronic, degenerative, noninflammatory disease of the periodontal tissues, which he referred to as "diffuse atrophy of alveolar bone".

Juvenile periodontitis becomes apparent about the time of puberty, usually between the ages of 10 and 15 (23). 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 incisors. As the disease progresses, the affected teeth may become increasingly mobile, with labial movement and spacing of incisors (6). Bleeding on probing of the periodontal pockets is also evident, reflecting ulceration of the crevicular epithelium. This has been confirmed by histologic examination of localized juvenile periodontitis lesions (14, 20).

Localized juvenile periodontitis tends to occur among members of the same family (2,3,17) and although an X-linked inheritance has been suggested (7,16) both sexes are affected and an autosomal recessive pattern of inheritance seems more likely 13.

Many researches have been done in identifying the microorganisms in localized juvenile periodontitis and their pathogenic potential (12, 18). Recent evidence suggests

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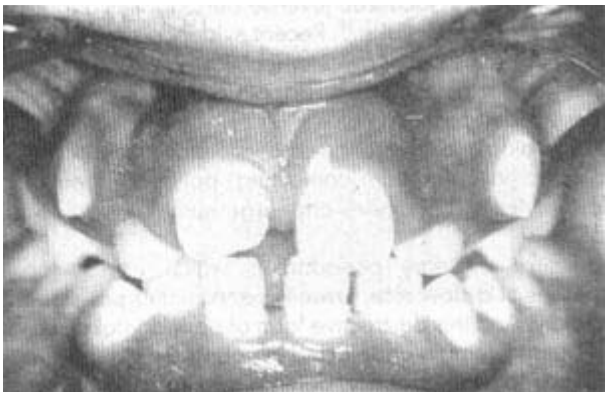
that Actinobacillus actinomycetemcomitans, a gram negative facultative anaerobic rod, plays a dominant role in the disease process (11,19).

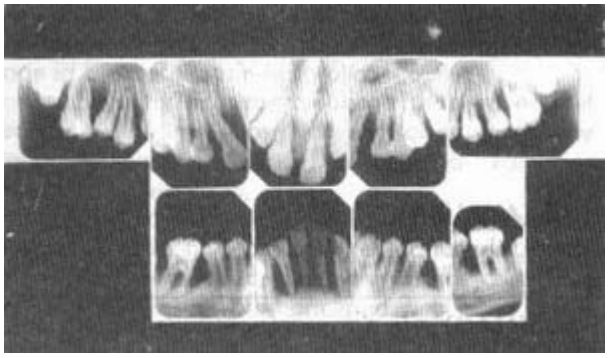
Histologic examination shows numerous areas of chronic inflammation containing polymorphonuclear leukocytes, lymphocytes and large numbers of plasma cells.

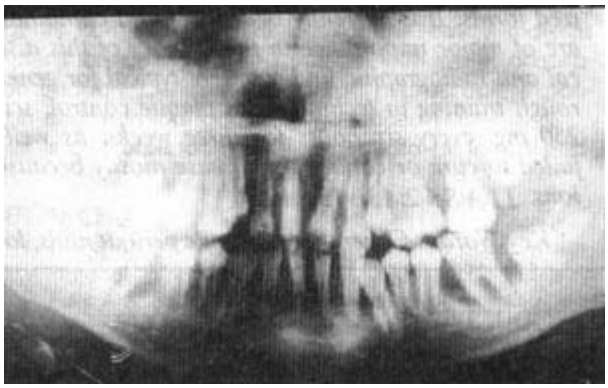
Unlike ordinary periodontitis which usually progresses at a slow rate, juvenile periodontitis progresses rapidly. And this destructive from of periodontal disease frequently remain undetected in young individuals, until increased tooth mobility, drifting, and spacing of teeth, abscess formation occurs. This makes the treatment so difficult.

A brief understanding of the etiologic agents in juvenile periodontitis may help clinicians provide a more efficient and effective therapy. A. actinomycetemcomitans is the mostly found organism in the mixed anaerobic pocket microflora of diseased sites in juvenile periodontitis. Culture studies of sulcular epithelium have comfirmed the histologic impression that, A. actinomycetemcomitans, invades and thrives within the periodontal soft tissues (4,8). So, Christersson *et al.* (5) 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 tissues. They suggest closed curettage and surgical curettage in conjunction with scaling and root planing.



Adjunctive antibiotic treatment of periodontally diseased patients is beneficial (22). One gram a day of systemically administered tetracycline produces a crevicular fluid concentration which is 20 to 10 times that of the blood (9). This concentration is effective against most of the periodontally pathogenic organisms (21).

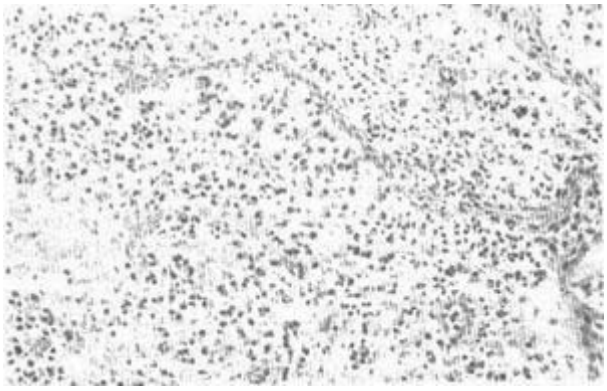




CASE REPORT

A 10 - year old female, was referred by her dental practitioner for a periodontal opinion. The patient's medical history was unremarkable.

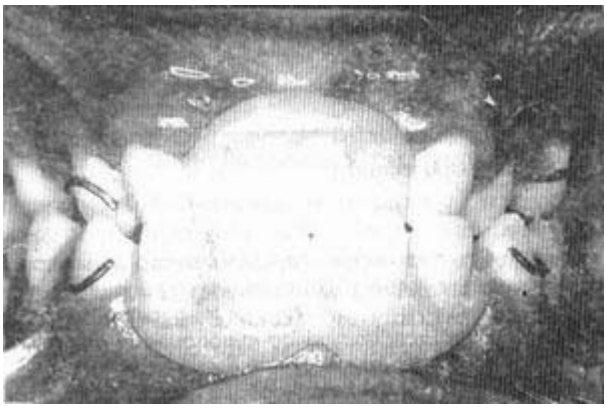
The pretreatment clinical photographs and radiographs are shown in Figures 1, 2,3. The clinical features are typical of juvenile periodontitis in its most active stage of progression. Both the attached and marginal gingiva were fiery red, acutely inflamed. Pus was oozing from around many teeth. In spite of this, the patient had no carious lesions and no restorations. The radiographs

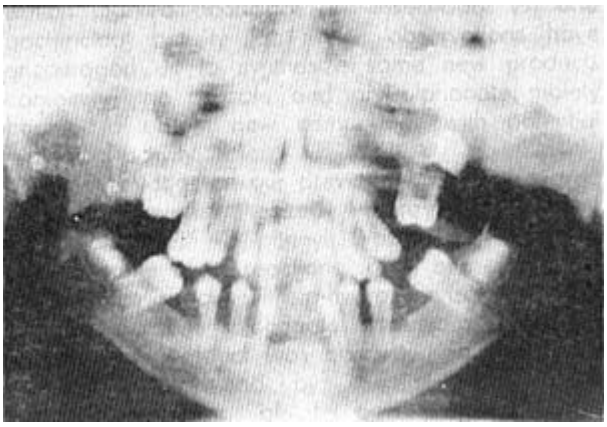


revealed almost total loss of the alveolar bone of $\frac{621|126}{621|126}$

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 chermotaxis 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 $\frac{1|6}{621|126}$ were extracted.

Following the completion of this phase of treatment, the patient was placed on recall, but she failed to reappear. She was not seen again until approximately 1 year later, at which time it was noted that she had ceased tooth brushing and her disease was again active. Oral hygiene was poor $\frac{7-2|2-57}{75-3|3-57}$ was present $\frac{642|245}{|}$ were severely mobile. During extractions of $\frac{64|}{|}$, significant hemorrhage had occurred from the granulation like tissue. Soft tissue specimen adjacent to the teeth sent for histopathological examination with the provisional diagnos of Juvenile periodontitis. Microscopic description was "Chronic, extensive inflammatory condition with numerous plasma cells, covered by non-keratinizing sguamos, hyperplastic, pseudoepithelial hyperplasia. Histological appearance is supporting clinical impression that of Juvenile Periodontis", (Figure 4).

$\frac{2|2\ 45}{|}$ were extracted over a period of time and flap currettage 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 (Figure 5). By the time of this paper was written the patient still had poor oral hygiene, upper %35, lower% 38 inflammation and extensive bone loss (Figure 6).

## 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 juvenile periodontitis however, the gingiva in the early stages, is most frequently normal in appearance. Patients, whose oral hygiene is poor have obvious plaque and supragingival calculus. In these instances gingival inflammation is present. It is also common to seen some signs of gingival inflammation clinically in the very advanced cases. In the vast majority of cases of juvenile 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 found (2).

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 bilat-

erally and the right and left sides are generally mirror images of each other (15). The degree and the shape of the bone loss is generally dependent upon whether the lesion is diagnosed in an early or advanced stage. 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 noncooperation with the oral hygiene instructions.

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