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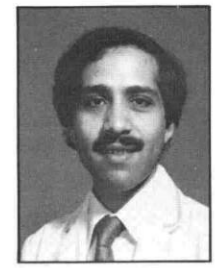

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Localized juvenile periodontitis (periodontosis)

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Abstract

A discussion of the recent information regarding the terminology, clinical manifestations and epidemiology of the periodontal disease termed Localized Juvenile Periodontitis (LJP) is presented in the context of newer scientific advances. Emphasis is given to the microbiological and immunological parameters associated with the etiology of the disease previously referred to as Periodontosis. Newer therapeutic modalities including adjunctive antimicrobial therapy appear to offer promise in the treatment of this highly destructive disease.

Introduction

Interest in the rapidly destructive form of periodontal disease referred to as "periodontosis" has greatly increased since 1973 when the suggestion of a unique bacterial etiology was made. In preliminary findings Newman et al.[1] reported that gram negative rods predominated at the forefront of molar incisor lesions in patients diagnosed as having "periodontosis." In that study, a sampling device which permitted bacterial sampling at the apical zone of the lesion was combined with newly developed methods[2] of increasing the viable recovery of plaque bacteria. Their report stimulated great interest among microbiologists, immunologists and clinicians since it suggested that bacterial specificity may be an important etiologic factor in this disease.

In the last seven years a major world-wide research effort has taken place in all areas of study. Particularly important has been the recognition that the rapid periodontal breakdown which occurs in "periodontosis" may also occur during periods of exacerbation in more common adult forms of periodontitis.[3,4] Understanding more about these periods of disease activity may provide the basis to unraveling key features associated with destructive periodontal diseases in man. Since "periodontosis" can be sharply defined it may be considered as an ideal model in which exacerbation and bone destruction can be more accurately predicted and studied.

The following discussion will highlight recent major areas of research activity which have provided the basis for our current understanding of this important periodontal disease. Many reviews have been published.[5,6,7]

Terminology

Even though a more clear understanding of the etiology and pathogenesis of this disease currently exists, the terminology used to describe this entity has become more confusing. Confusion may result from a failure to clarify and correlate the clinical descriptors with research findings. These findings have clearly demonstrated that specific microbiologic, immunologic and histologic features can be associated with this disease process (Table 1). It is suggested that these findings become incorporated into, and become part of, the diagnostic features and then become the basis for classification of the disease process. Table 2 lists some current terminology. The author prefers the differentiation of the molar-incisor type of bone loss from the generalized involvement since this condition can be distinctly separated on the basis of clinical criteria in moderate and advanced cases. *Localized Juvenile Periodontitis, (LJP),* is the suggested term for the molar incisor involvement. Generalized terms can be referred to as *Juvenile Periodontitis, (JP).* As with any classification system there are always exceptions. In a recent study Hormand and Frandsen[8] described three types of bone loss in patients with JP. Type I included first molars and/or incisors. Type II included first molars and/or incisors and some additional teeth (less than 14 involved teeth) and Type III patients were generally involved. The authors referred to all three types as JP.

In 1977 Sugarman and Sugarman[9] argued that the term *Precocious Periodontitis* be used to describe both localized and generalized forms. The authors argued that this terminology more clearly describes the clinical manifestation of the disease process. In 1978 Baer and Kaslick pointed out that the term *"Periodon-*

Table 1. Possible etiologic factors associated with LJP.

Subgingival Plaque
Gram Negative Rods
PMN Dysfunction
Cell Mediated Deficiency
Altered Cementum

tosis" was never intended to mean "degenerative." They suggested the etymological derivation was from the Greek and properly defined the term *"Periodontosis"* as meaning "an abnormal or diseased condition of the periodontium." It is clear that confusion over terminology and definitions exist. The following discussion may help to clarify the confusion and provide the basis for agreement.

Definition — Clinical Features

Age of Onset: The circumpubertal period appears to be the primary time when the disease process manifests.[11,12] In some cases however, bone destruction can be documented to occur in the mid-teen years. LJP as described in this article, does not occur in the prepubertal child, it is a disease of the immediate preteen and teenage years. If rapid or unusual periodontal destruction occurs in young children it may be simply termed, *Periodontitis.*

Clinical Manifestations: Table 3 summarizes the major clinical manifestations associated with LJP. The key features of early and moderate forms of the disease are unlike the adult forms of periodontitis of similar involvement. Particularly important is the lack of those clinical signs which result from an acute inflammatory response such as: gingival erythema, edema, and bleeding adjacent to involved teeth. When poor oral hygiene is present plaque and calculus may induce gingival inflammation. Later clinical findings (Table 4) are more typical of "inflammatory" chronic periodontitis. In these cases roentgenographic evidence of bone loss and periodontal probing can be used to confirm the diagnosis of LJP.

Table 3. LJP: Early clinical findings.

Age	Circumpubertal
Systemic	None
Bone Loss	Molar — Incisor — Bilateral and Symmetrical
Onset and Course	Insidious + Rapid
Gingiva	Little Clinical Inflammation
Plaque	Small Amounts
Microbiology	Characteristic Gram Neg. Flora
Other	Regional Lymph Nodes
	Familial Pattern
	No Primary Teeth Involvement
	PMN Dysfunction

Table 2. Current terminology.

Periodontosis
Juvenile Periodontitis
*Localized Juvenile Periodontitis
Idiopathic Juvenile Periodontitis
Precocious Periodontitis
Gottlieb Syndrome
Destructive Juvenile Periodontitis

* Preferred Term

Many patterns of bone loss may occur among involved teeth. Maxillary first molars and incisors are affected to a higher degree than their mandibular counterparts.[8] There are numerous studies which have documented the bilateral (cross arch) symmetry of bone loss. Bone loss occurs rapidly. One study[13] has documented an average attachment loss of 4-5 microns per day.

General Health: LJP occurs in healthy patients. No systemic disease occurs with this condition. When rapid periodontal destruction occurs in patients with certain systemic conditions such as Papillon Lefevre syndrome or cyclic neutropenia it is called "periodontitis" not LJP. A recent study which examined the microbiota associated with Papillon Lefevre syndrome[14] demonstrated that a flora similar to adult forms of periodontitis was present. The microbiota was remarkably different from that seen in patients with LJP (see below).

Recent studies (see below) have also suggested that an underlying defect in polymorphonuclear leukocytes (PMN) may predispose affected patients to LJP. Clark et al.[15] have suggested that the PMN dysfunction is genetically transmitted and is relatively mild. Affected patients have no other predisposition (as far as is known) to other infectious agents. This observation does not alter the conceptual basis for defining LJP as occurring in healthy patients.

Table 4. LJP: Late clinical findings.

Mobility and Migration
Diasthemata
Pocket Formation
Root Sensitivity
Pain Upon Mastication
Abscess Formation
Plaque and Inflammation
Burn Out

Epidemiology

Incidence: A recent unpublished study conducted by Hew and Killoy[16] examined 22,000 U.S. Air Force recruits for evidence of LJP. Their findings indicated an overall incidence of 0.255%. When race was considered, blacks had an overall incidence of 0.410% while caucasions had an overall incidence of 0.198%.

Sex Ratio: Hew and Killoy[16] and others reported an overall female to male ratio of 1.05:1. When racial considerations were analyzed the female to male ratio among blacks was 1.16:1, and among caucasions 1.19:1. Horman and Frandsen[8] found that the overall female to male ratio was 2.5:1 in their caucasion population. When broken down according to age, younger females (12-18) had a female to male ratio of 5.3:1 while older females had a ratio of 2.4:1.

Familial Pattern: The familial pattern of the occurrence of LJP is well documented. Potential genetic predisposition is very likely based on recent immunologic studies as well as from historical data.

Etiology: The Role of Microorganisms

While many details describing the microbiota of the LJP lesions have not been clarified, a number of observations seem consistent (Table 5). The number of organisms in LJP lesions is less than in most forms of destructive periodontal disease[3] ranging from 10^5-10^6 bacteria per pocket. This may be one to three orders of magnitude lower than counts of organisms in adult periodontitis pockets of similar depth and associated bone loss. From microscopic studies of *in situ* plaque, it appears that the organisms at the apical portion of the pocket are loosely and sparsely attached to the tooth surface[13,17,18] with little evidence of calcification. Clinically, the root surfaces feel hard and smooth.

The predominant cultivable microbiota of the LJP lesion is dominated by gram negative rods.[1,19,20,21] Most of the isolates from such lesions have been saccharolytic and capnophilic to anaerobic (Table 6). At the present time, three groups of organisms appear to be more frequently encountered in lesions of LJP patients, as well as in other patients with early onset highly destructive periodontal disease.[22] These organisms are not isolated in the same magnitude or frequency in diseases affecting adult patients.

Table 5. Microbiologic features associated with LJP.

Fewer bacteria
Unusual gram negative rods
Major proportion of plaque — unattached
Pathogenic potential in animal models[20]
Immunologic response
Antibacterial treatment

One group which has received considerable attention consists of fusiform-shaped, capnophilic rods which have the ability to glide on agar surfaces. Members of this group had been called *Bacteroides ochraceus,* but are now designated by the genus name *Capnocytophaga.*[23,25,26] The creation of the new genus was based on the recognition of a typical "wet spreading" colony demonstrating the gliding ability of the organisms on the solid agar surface. The organisms are not anaerobic (as originally described by Newman et al.[20]), but require increased carbon dioxide tension for their growth. The ability to glide on surfaces has long been recognized in organisms colonizing other sites in nature, particularly the soil. Since the oral isolates most resembled the gliding aerobic genus *Cytophaga,* a new genus *Capnocytophaga* was proposed to include the carbon dioxide loving oral strains.[24] The genus *Capnocytophaga* includes three species: *Capnocytophaga ochracea, Capnocytophaga sputigena* and *Capnocytophaga gingivalis.*[23,24] In early publications, the *Capnocytophaga* strains had been designated Group II.[19,20] All three species have been detected in LJP lesions with total *Capnocytophaga* counts averaging 25% of the organisms isolated.

A second group of capnophilic, gram negative rods frequently isolated from LJP lesions includes the species *Actinobacillus actinomycetemcomitans.*[27,28] Strains of this species were included in Groups III and IV in one of the original descriptions of the microbiota of LJP lesions.[19,20] The colonies are small, convex and smooth, while the cells are short, round-ended and occasionally slightly curved. Strains of this species produce a protein exotoxin capable of destroying polymorphonuclear leukocytes and gingival fibroblasts.[29,30,31] The exact role that this organism, its exotoxin and the host response to the exotoxin play in the pathogenesis is under intensive investigation and is one of the most exciting avenues of research concerning this disease entity.

Table 6. Changes in terminology for LJP associated bacteria.

Original Classification[20,21]	*Current* Terminology*
*Group I	Anaerobic Vibrio-STB
*Group II	*Capnocytophaga*
*Group III	Group III STB & *Actinobacillus*
*Group IV	*Actinobacillus* (Y4)
	Bacteroides corrodens
*Group V	*Eubacterim*

* Other bacteria within groups I - V are not listed under heading "Current Terminology" since they have not been characterized further.

A third group of organisms frequently encountered in LJP lesions includes organisms previously placed in "periodontosis Group V."[19,20] Strains of this group often demonstrate a type of spreading on the agar surface. Many isolates from Group V appear to fit descriptions of the species *Eubacterium saburrheum*.[27] This species stains Gram positively in very young cultures and Gram negatively in older preparations, cells are long blunt-ended rods which often produce filaments.

The three groups of organisms briefly discussed above are by no means solely confined to LJP lesions or lesions of other forms of early onset destructive diseases. However, their frequency of isolation and their proportions when isolated are clearly much higher in these lesions than in lesions of adult destructive diseases or healthy sites of adult patients or age-matched controls.

In 1978, Allen and Brady[18] found rod-shaped microorganisms which were continuously present along the entire cemental surface of extracted LJP involved teeth. The cultural and anatomical association of microorganisms associated with LJP lesions provides the basis for research which may clarify the exact role of these bacteria. These observations were prerequisite to a more accurate assessment of the response (or failure to respond) by the host to potentially pathogenic bacteria and/or their products.

Immunologic Studies

Antibody: IgG antibody response to many of the subgingival bacteria associated with LJP lesions has been documented.[32,33,34,35] In particular, antibody to *A. actinomycetemcomitans* and its leukotoxin has received the greatest attention.[36,37,38,39,40] This response has been interpreted by some as a blocking effect of the leukotoxin.[36] The antibody response to *A. actinomycetemcomitans* is also strain specific[34,41] indicating the possibility of virulent and avirulent strains.

Gingival fluid antibodies appear to parallel antibody activity in serum[35] but, antibody coating of subgingival bacteria from LJP lesions may not occur to high degree.[42] These observations may provide insight into the host-bacterial interactions which take place in the lesion. Also, the pathogenicity of the resident flora may be enhanced since the bacterial numbers would not be limited by antibody interactions occurring within the lesion.

Polymorphonuclear Leukocytes (PMN): The current research investigating PMN functions in LJP may provide a more clear understanding of the pathogenic mechanisms involved with the disease process.[15,43,44,45] Table 7 lists the major research findings associated with PMNs.

Cianciola et al.[43] compared the functions (chemotaxis and phagocytosis) of PMNs in juvenile periodontitis (JP) patients to their first-degree relatives and to normal subjects. They found a marked reduction in the ability of patients' neutrophils to respond to chemotatic agents. Peripheral monocytes from the same group of patients with JP responded normally in similar chemotaxis experiments, suggesting that the defective chemotactic response was limited to neutrophils and not shared by other phagocytes. The cells of the patients with JP showed markedly reduced (50%) bacterial phagocytic activity compared to age- and sex-matched normal controls.

Table 7. Polymorphonuclear leukocytes (PMN) associated with LJP.

LJP Association	*Observations*
Presence in lesion	Histologic sections confirms their presence
Imparied function	In serum and gingival fluid
Sensitivity to bacterial exotoxin	From *A. actinomycetemcomitans* and other bacteria?

The PMNs of siblings showed a marked reduction in chemotactic response in all of the siblings with JP and in several younger siblings with no signs of periodontal disease. Older siblings with normal neutrophil chemotaxis did not show clinical manifestations of JP. It has been concluded that reduced neutrophil chemotaxis seen in subjects with JP does not result from the disease; it may be inherited and predispose to the early severe bone loss seen in JP. However, these observations did not show to what extent compromised neutrophil function is a factor in determining the severity of extreme forms of periodontal disease.

In 1977, Clark et al.[15] evaluated the serum chemotactic activity and the presence of serum chemotactic inhibitors in JP patients. A reduced level of complement-derived chemotactic activity was demonstrated in serum from four of nine patients. Serum from five of the JP patients contained a heat-stable, non-dialyzable factor that markedly inhibited the chemotaxis of normal neutrophils. One patient who had defective chemotaxis was evaluated again after being successfully treated for JP. No improvement in chemotaxis was observed.

Cell-Mediated Immunity: Using a non-specific immunostimulation drug, levamisol, Budtz-Jorgensen et al.[46,47] evaluated the competency of the drug to stimulate immunologic responsiveness in LJP patients. They concluded that LJP is associated with an *in vitro* cell-mediated deficiency. Previous studies by Lehner et al.[48] have suggested a similar deficiency.

Treatment

Since the suggestion that subgingival plaque may be etiologic and contribute to the destructive process in LJP, much of the treatment employed in these patients has been directed towards the control of the associated bacteria.[19,49] The results of treatment can be excellent especially when initiated early in the clinical course of the disease and/or when the destructive process begins late in the teen years (Table 8). Table 4 summarizes the current treatment modalities.

The clinical course of LJP is rapid and fairly predictable. However, there is a phenomenon referred to as "burn out" which is seen in many cases. Burn out refers to a sudden and unexplainable decrease in the rate of bone destruction, and is almost always seen in the mid- and late twenties. In these cases, the onset of alveolar resorption apparently occurred in the mid- to late teens rather than during the circumpubertal period. Although many theories have been postulated regarding this phenomenon, none are based on scientific fact.

The use of systemic tetracycline in patients with LJP appears to enchance treatment success in some patients. When employed by the author, the combination of surgical intervention, stablization, and systemic antibiotic appears to have success when the patients were selected on the basis of antimicrobial susceptibility testing of the organisms found in the lesions (Figure 1). The predictability of treatment success can be enchanced (Table 9) as in other forms of destructive periodontal disease in man. There is no better treatment than prevention through early diagnosis.

Table 8. Diagnostic features associated with treatment of LJP.

Diagnostic Feature	*Effect on Treatment*
Age of onset	Late onset favors successful treatment
Depth of lesion	Incipient lesions favors treatment
Furcation involvement	Decreases success
Oral hygiene	Improves prognosis
Occlusion	Excessive forces decreases success

Table 9. Treatment modalities currently used in LJP patients.

Nonsurgical
Plaque control
Root planing
Antibacterial agents
Orthodontic treatment
Occlusal adjustment
Surgical
Mucoperiosteal flap
Osseous grafting
Autologous tooth transplantation
Hemisection, root amputation
Extraction

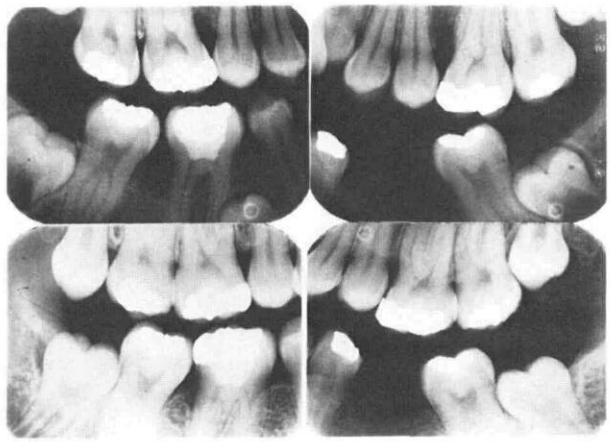

Figure 1. Roentgenographs of 18-year-old girl treated for LJP with conventional therapy and systemic tetracycline. Patients bacteria were tested for susceptibility to tetracycline prior to treatment. Top: Bitewing roentgenographs at age 14 demonstrating moderate bone loss around remaining first molars. Bottom: Bitewing roentgenographs four years later demonstrating cessation of visable bone loss and improvement in roentgenographic picture. Clinically, pocket depths were 3 mm or less in all areas. M. G. Newman, E. Montierth and R. Williams.

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