Borrower: AZA

Lending String: *OHS,KUM,NBG,OS2,CHS

Patron: Jones, Desiree

Journal Title: General dentistry.
Volume: 46 **Issue:** 6
Month/Year: 11 1998 **Pages:** 580-7; quiz 588-9

Article Author:
Article Title: Gustke CJ; A review of localized juvenile periodontitis (LJP); II. Clinical trials and treatment guidelines.

Imprint: [Chicago, Academy of General Dentistry]

ILL Number: 80774445

Date: 8/4/2011 01:45:09 PM

Call #:
Location: main

cdd

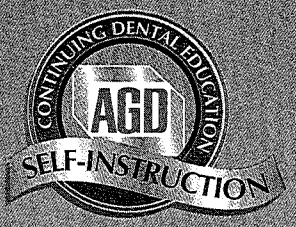
Odyssey: ~~ODYSSEY ENABLED~~
Shipping Option: Ariel/EDD
Ariel: 128.196.164.43
Fax: (520)626-2831

Maxcost: 20IFM

Shipping Address:
University of Arizona
Health Sciences Library-ILL
1501 N. Campbell, Rm 1149
PO Box 245079
Tucson, AZ 85724-5079

Borrowing Notes; We will pay via ifm or invoice. Thank you.

Carl J. Gustke, DDS, MS



Self-Instruction article for Exercise No. 65
Subject code: 495

Abstract
Localized juvenile periodontitis (LJP) causes severe alveolar bone loss and early tooth loss in adolescents and young adults. If it is not appropriately treated as an infection in association with *Actinobacillus actinomycetemcomitans*, treatment failure is likely. This review of clinical trials of treatment of LJP uses those trials to construct guidelines for LJP treatment.

A review of localized juvenile periodontitis (LJP): II. Clinical trials and treatment guidelines

Note: This is Part II of a two-part article. Part I appears on pages 491–497 of the September/October 1998 issue of *General Dentistry*.

Localized juvenile periodontitis (LJP) is an uncommon, yet severely destructive disease that occurs in adolescents and young adults. As discussed in Part I of this article, the primary etiology of LJP is infection with *Actinobacillus actinomycetemcomitans* (*A.a.*).[1] Clinical trials of treatment have focused attention on the elimination of *A.a.* as a means of arresting disease progression and maintaining the dentition. Here, diagnosis and treatment guidelines are proposed for LJP, based on a review of the scientific literature.

Diagnosis

LJP usually presents itself as severe vertical bone loss, detected on molar bitewing radiographs. For early detection of LJP, careful interpretation of radiographs is essential. Such detection also should include other factors, such as attachment loss and family history. Mann et al. evaluated radiographs for cementoenamel junction (CEJ)-alveolar crest distance greater than 1.0 mm, widening of the periodontal ligament space, absence of crestal cortical plate, and loss of trabeculae, but found no relationship with probing attachment loss in a group of 12- to 16-year-olds.[2] The normal level of the alveolar crest is about 1.0 mm apical to the CEJ. An alveolar crest more than 2.0 mm from the CEJ may represent bone loss.[3] Crestal lamina dura is not a reliable indicator of early periodontal disease activity and should not be used as a sole indicator for LJP.[4,5] Therefore, it is the height of the bone, and vertical defects in particular, that indicate the presence of LJP.

In addition to radiographs, careful probing is essential in the diagnosis of LJP. The clinician must take into consideration the degree to which the gingival tissues are inflamed, which may mean that a 5.0 mm probing depth is a pseudopocket. The probe may be stopped from reaching the base of the pocket by heavy calculus. Therefore, loss of attachment is evidence that periodontitis is present. This is detected when the probe penetrates past the CEJ of the tooth, and the root surface can be felt. Now the inflammatory process has moved beyond gingivitis, and is periodontitis. Attachment loss during adolescence often is indicative of early-onset periodontitis (EOP). Therefore, when a tentative diagnosis of LJP has been made, the clinician should include attachment level charting in his or her clinical database, in addition to probing depths.

In diagnosis of bone or attachment loss in children and adolescents, a careful distinction should be made between what is truly EOP, and what is an incidental loss of attachment (IAL). IAL is loss of attachment or bone that is directly associated with a local plaque-retentive factor, such as an overhanging restoration, deep caries, severe crowding, or a pulpal/periapical pathological entity. Both EOP and IAL may cause early bone loss, but the etiology (and therefore, treatment) of these two problems is distinctly different.

As with other forms of periodontitis, the baseline database includes an assessment of oral hygiene, and recording of sites that bleed in response to probing. These factors are necessary to assess the response of the patient to oral hygiene instructions, and to assess reduction of inflammation in the gingival tissues. Without such a comprehensive database, it is difficult to know how to treat different sites appropriately, to determine the magnitude of clinical improvement after therapy, and to assess long-term stability in the maintenance phase.

An additional means that may be used in the diagnosis of LJP is microbial testing. Microbial testing can be useful for determining whether the pathogen *A.a.* is present, and whether it has been eliminated after therapy. Microbial culture and antibiotic sensitivity testing can assist in appropriate antibiotic selection. However, culture and sensitivity studies can be prohibitively expensive for many patients, so antibiotics often may be used empirically, based on the association of *A.a.* with LJP. A less expensive alternative to culture and sensitivity studies is the DNA probe, which can determine the presence of *A.a.*, but not the antibiotic sensitivity for the individual patient's specific strain. Further, microbial testing can be used in the maintenance phase, to monitor for reinfection with *A.a.*

Finally, in diagnosing LJP in its early stages, it may be helpful to obtain a family history of periodontal disease and tooth loss experience. LJP has a genetic basis for susceptibility, and tends to occur in families.[6] The patient's history may reveal that his or her parents or older siblings have lost several teeth at an early age as a result of LJP. If this is the case, incipient bone loss or periodontal pockets in an adolescent patient are more likely to be interpreted as the onset of LJP. A guide for diagnostic data collection for LJP is summarized in Table 1.

Table 1. LJP diagnostic data collection.

A. Full-mouth probing chart with attachment levels and bleeding on probing
B. Oral hygiene assessment
C. Radiographs: panoramic, bitewings, PA of each affected tooth
D. Sibling screening/family history
E. Microbial testing (this is an optional adjunctive step)

Treatment

Treatment strategies have been developed for LJP; the elimination of *A.a.* is the goal of treatment. This should stop the progression of the disease, and produce an environment in which healing can occur. Continued suppression of *A.a.* is the goal for post-treatment stability. A secondary treatment goal may be restoration of the lost attachment apparatus.

The first clinical trials of the treatment of LJP as an *A.a.* infection were published in 1983–85. Slots and Rosling treated six LJP patients over the course of 22 weeks, with the following sequence: (1) 6 hours or more of scaling and root planing during the first 12 weeks; (2) 10 minutes of subgingival iodine application at week 16; and (3) 2 weeks of systemic tetracycline (250 mg, 4 times per day) administered from weeks 20 to 22.[7] Although scaling and root planing decreased inflammation and pocket depths somewhat, the attachment levels continued to deteriorate. Most importantly, mean *A.a.* concentrations remained high, and all pockets tested were still infected. The *A.a.* levels remained unchanged after iodine irrigation. Two weeks after tetracycline therapy, only 4 of 20 pockets (all in one patient) still had *A.a.* at detectable levels, and the mean levels were decreased by more than 90 percent in the pockets that still harbored *A.a.* After tetracycline therapy, attachment level gains were seen, but the pockets that still were infected continued to lose attachment. This demonstrated that scaling and root planing alone and with iodine irrigation were ineffective in controlling *A.a.*, the etiologic organism in LJP, and could not arrest its progression. It also demonstrated that systemic tetracycline was very efficacious for reducing or eliminating *A.a.* from periodontal pockets, and was effective clinically. However, at 9 to 15 months, half of the patients were *A.a.*-positive again, and the levels of this organism appeared to be increasing.

Lindhe and Liljenberg reported the results of treatment of LJP with a five-year follow-up period.[8] Sixteen patients were treated with modified Widman flap surgery (including removal of granulation tissue and root planing, but no osseous surgery), and a 2-week regimen of tetracycline, 250 mg, 4 times per day. For the first 6 months, the patients returned each month for polishing. For the remainder of the five-year period, they returned every 3 months for maintenance. Plaque scores were 67 percent at baseline and decreased to 19 percent at 6 months and 14 percent at 5 years. Mean probing depths of diseased molars decreased from 8.6 mm to 3.4 mm at 6 months, and remained stable for 5 years. For

Table 2. LJP treatment sequence.

A. Oral hygiene instructions/patient education
B. Gross calculus debridement (if needed)
C. Surgical treatment of pockets >5.0 mm, root planing and curettage of shallower lesions. Use chlorhexidine 1 to 2 weeks after surgery, then add interproximal brushing.
D. Antibiotic therapy concurrent with surgery and/or root planing (empirical regimen or based on microbial testing)
E. Evaluation of results (4 to 6 weeks after therapy)
[Repeat microbial testing—*A.a.* should be eliminated]*
Success = good oral hygiene, reduced probing depths, no bleeding on probing, gain of clinical attachment [elimination of *A.a.*]*
F. Retreatment of deep, bleeding pockets (if needed)
G. Maintenance every 3 months
1. Probe
2. Radiographs, as needed
3. Reinforcement of oral hygiene instructions
4. Prophylaxis/root planing
[5. Microbial testing]*

*Brackets indicate optional adjunctive step

diseased incisors, probing depths decreased from more than 7.0 mm to less than 4.0 mm. Molars gained 5.0 mm, and incisors gained 2.0 mm of clinical attachment at 6 months; this attachment was sustained for five years. However, 25 percent of the patients had recurrent lesions within the first 12 months after treatment, indicating a high potential for recurrence. These patients were treated again in the same manner as before, and there was no further recurrence for five years. Additionally, partial to nearly complete resolution of the bony defects was observed (though not quantified). This study demonstrated that a combined surgical/tetracycline treatment protocol, in a patient sample that complied with a three-month maintenance interval, was highly effective in the treatment of LJP. However, in the first 12 months, recurrence could be a problem.

Christersson et al. demonstrated the importance of surgical treatment of LJP patients to remove tissues that were infected with *A.a.*[9] Comparing root planing to modified Widman flap surgery, *A.a.* was not reduced in the lesions that were root planed only, but in 80 percent of the surgical sites, *A.a.* was undetectable 1 week later. Sixteen weeks later, 50 percent of the sites still did not have detectable levels of *A.a.* Probing depths improved in the surgery group, but not in the root planing group. In this study, tetracycline was not used, and the results were not as favorable as in the Slots and Rosling study.[7] It was suggested that suppression of *A.a.* would be more predictable if surgery and antibiotics were used together.

The need for surgical treatment and antibiotics was questioned by Wennstrom et al.[10] Sixteen LJP patients had modified Widman flap surgery on one side of the oral cavity, and scaling and root planing on the other side. No antibiotics were prescribed. The teeth were polished (for plaque removal) every month for six months, and then maintenance was performed every three months for two years. From two to five years, the patients were released from the maintenance schedule to see their usual dentist "as needed." At six months, probing depths in the surgical sites decreased from baseline 7.4 mm to 3.7 mm. Probing depths at nonsurgical sites decreased from 7.0 mm to 3.8 mm. These improvements remained stable for two years, but deteriorated by five years, after maintenance was discontinued. In both groups, attachment gain of approximately 2.0 mm was observed at six months and at the two-year evaluation, but at five years, the attachment gain decreased to about 1.0 mm. Mean bone fill of 2.0 mm was observed at two years. Numerous sites experienced recurrent attachment loss during the three-year period without maintenance. Eighty-four percent of the sites that lost attachment were in the nonsurgical group. Unfortunately, the only time at which *A.a.* was monitored was at five years, so the short-term post-treatment effect on *A.a.* is unknown. This study challenges the conventional wisdom (and many other studies) that suggests that surgery and/or antibiotics are necessary for the treatment of LJP. Optimal, frequent, professional plaque control was noted to determine the success of the treatment. The clearest finding of this study is that a three-month recall interval for maintenance is needed to sustain the probing depth reduction and attachment gain from LJP treatment over the long term. However, surgically treated sites were less likely to lose attachment during the period with inadequate maintenance visits.

Palmer et al. used tetracycline in a double-blind, controlled clinical trial for treatment of juvenile periodontitis.[11] In the initial phase of the study, 19 controls received root planing and oral hygiene instructions. In addition to root planing, the 19 test subjects also received tetracycline 250 mg, 4 times per day for 14 days. Three months after treatment, the patients who received tetracycline had superior probing depth reduction, less bleeding on probing, and better clinical attachment gain, compared to the controls. Additionally, only 58 percent of the initially affected teeth in the tetracycline group required