

CLINICAL ROUNDTABLE



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Question:

What impact will the link between periodontal disease and systemic disease have on the future of using periodontal therapy to save teeth vs extraction and implant placement?

ROBERT LEVINE, DDS

It should have minimal effect on treatment except that this link needs to be discussed with patients at the outset of treatment along with other "risk factors" that would have an influence on the long-term success of their care. When patients present to my office for consultation, we meet and interview them away from the clinical area in a non-threatening space where we have an opportunity to establish a rapport. A review of their chief complaint and their beliefs/values are determined through conversation and oral data gathering. We specifically look for potential "risk factors" that may hinder treatment success, such as diabetes control or risk (ie, a close family member with diabetes), smoking habit and their desire to quit, compliance to prevention, osteoporosis (which will limit the ability to immediate- or early-load because loading protocols should not be reduced with patients on oral medication for osteoporosis), and prior history of periodontal disease and/or therapy. In addition, a familial history of heart disease with periodontal disease is taken, as genetics plays an important role in risk. Once these factors are addressed and openly reviewed with the patient, then we move into our clinical setting for a comprehensive periodontal, occlusal, and soft tissue exam. After the diagnosis and prognosis of specific teeth are determined, we determine with the restorative dentist and the patient what an appropriate plan(s) would be to address and satisfy their chief complaints.

With what we know today regarding bacteriology of the pocket in gingivitis, beginning, moderate, and advanced periodontal disease states, an anti-infective, inflammation reduction approach is recommended. As part of Phase One therapy

(non-surgical periodontal therapy; scaling and root planing) we place subgingival antibiotics (5-mm to 6-mm pockets) and supplement with systemic oral antibiotics in the more advanced cases (> 6-mm pockets) for 1 week after Phase One, either based on clinical presentation of the patient or subgingival culturing of the pocket flora. In both early and moderate-advanced periodontitis cases, we routinely prescribe oral doxycycline (20 mg) for the benefits of collagenase stabilization and thus inflammation reduction, which aids in healing of both soft and hard tissues. This three-pronged approach is excellent in reestablishing signs of clinical periodontal health and determining necessary Phase Two (periodontal surgical) therapy to further establish periodontal health. Lastly, gingivitis used to be considered not as important on the scale as other periodontal diseases. However, the periodontal-systemic link does not differentiate between gingivitis and periodontitis. Thus we need to include gingivitis with appropriate Phase One therapy to prevent gingivitis' continued inflammatory response.

FRANCIS G. SERIO, DMD, MS, MBA

First, irrespective of the association between periodontal inflammation and systemic diseases, I think that the treatment-planing paradigm has already shifted to the earlier removal of periodontally involved teeth and their replacement with endosseous implants. In the past, before the availability of relatively predictable implant therapy, periodontists and their patients went to great lengths to control periodontal inflammation and maintain an intact or relatively intact natural dentition, sometimes supplemented by complex fixed or removable prostheses. The decision now is not

which periodontal therapy can maximize the longevity of a natural tooth but at what point should a natural tooth with moderate to severe attachment loss be removed to ensure the availability of a sufficient volume and quality of bone to successfully support one or more implants and their restoration.

Regarding the relationship between periodontal inflammation and its contribution to various systemic conditions, there is significant statistical evidence showing an association between periodontal inflammation and several systemic diseases, but the proof of a causal relationship of the former to the latter is still elusive. The proof of causality, and to what extent one disease contributes to the progression of another, is particularly difficult in diseases with complex etiologies and pathogenic mechanisms. At this point and for the foreseeable future, I would suggest treating periodontal disease for its own sake, as inflammation is harmful no matter where it is found in the body. As teeth are lost from attachment loss, then a decision must be made on a replacement. In the face of an epidemic of obesity, diabetes, smoking among approximately 25% of the population, high stress, lack of exercise, and poor nutrition, the suggestion to remove teeth and replace them with implants as an approach to controlling systemic disease is an idea whose time has not yet come, and may never come without addressing the aforementioned factors, and others, as well.

RAY C. WILLIAMS, DDS

I don't believe the emerging link between the presence of periodontal disease and the risk for certain systemic conditions will affect the profession's longstanding focus on saving teeth, nor do I see this relationship

of oral health/systemic health shifting the profession toward a greater likelihood to remove teeth and replace teeth with dental implants. The main consideration should continue to be that periodontal disease is a disease that is very preventable and treatable. In fact, new treatment strategies are being introduced regularly that bode well for additional successes in managing the periodontal diseases. Of particular excitement is the increasing evidence that we can more predictably rebuild/regenerate lost periodontal attachment structures with growth and differentiation factors. In addition, we can successfully regenerate periodontal structures with signaling molecules such as enamel-related matrix proteins. Research in the ecology of the etiologic biofilm that causes periodontal disease suggests that we are beginning to understand the build-up of the critical climax community of microorganisms and this new knowledge should open new doors for disease treatment focusing on the causative bacterial flora. In addition, we have a much better understanding of the role of the susceptible host in the tissue destruction of periodontal disease. And in particular, we have a new appreciation of the inflammatory response. With this new understanding of inflammation and its resolution, we are poised to more effectively manage the resolution of periodontal inflammation with new and emerging treatment strategies. All in all, it couldn't be a more exciting time for periodontal disease prevention and treatment. And yet, fortunately for our patients, in those instances in which periodontal disease cannot be successfully treated, and a tooth or teeth are lost, dental implants are a predictable and successful treatment strategy for replacing missing teeth.