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Review article

Controversy in Periodontics and implication

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ABSTRACT

Controversy is a state of prolonged public dispute or debate, usually concerning a matter of conflicting opinion or point of view. The word was coined from the Latin controversia, as a composite of controversus – "turned in an opposite direction". In periodontology some important topic have some controversies The reason of controversy on a particular topic may be related to inadequate knowledge of the etiological factors, technical difficulties, and patient related factor effecting the treatment outcomes or some unknown factors. This article aims to describe some of the current controversial topics in periodontology.

Introduction

The way of periodontal concept is indeed advanced how it had been in the past. We now have a better understanding of the etiological factors associated with periodontitis, the mechanism involved in periodontal wound healing and inter relationship between patients factors (such as smoking and diabetes) and treatment outcomes. However some controversies related to periodontology do exist. The reason of controversy on a particular topic may be related to inadequate knowledge of the etiological factors, technical difficulties, and patient related factor affecting the treatment outcomes or some unknown factors. Some of the current controversial topics in periodontology are described here.

Case definition of periodontal disease and its impact: A fundamental prerequisite for any epidemiologic study is an accurate definition of the disease under investigation. However the threshold values that determine shallow, deep or pathologic pocket varies among studies, this in turn effect diagnosis and potentially controversy. At present, it is difficult to accurately assess epidemiologic data on periodontal disease because of the wide variety of indices and measurement used. The American academy of periodontology (AAP) reported 10 different classification system in 20 years. This is one of the reason why the prevalence of gingivitis and destructive periodontal disease can range widely, depending on which reference levels are considered to be the normal versus diseased, shallow pocket versus deep pathologic pocket. It has far reaching controversial consequence in diagnosis, treatment planning and prognosis of any periodontal disease.

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Periodontal disease and its impact on systemic health

In 1900 William Hunter first developed the idea that oral infection are responsible for a wide range of systemic diseases and conditions (oral sepsis). But the focal infection theory fell into dispute in the 1940 and 1950s when widespread extraction, often of the entire dentition, failed to reduce or eliminate the systemic condition to which the supposedly infected dentition had been linked. Again in 1990s Matilla, Beck and various other studies showed that periodontal infections can negatively influence health. Since than various studies performed found association of periodontal health and systemic condition and disease (e.g. CHD,diabetes, adverse pregnancy outcome, pulmonary disease). Some other studies did not found association. So, does periodontal disease cause CHD, or adverse pregnancy outcome? The question can only be answered based on the currently available, with the full knowledge that conclusions may change as future evidence dictates. Periodontal disease may increase the risk of many systemic disorder. Biologic plausibility mechanism support the role of periodontal infection in these conditions, but periodontal infections should not be presented as the cause of such systemic disorder. For example an extensive systemic review by Scannapieco et al 2003 concluded that a moderate degree of evidence exists to support an association between periodontal disease and atherosclerosis, myocardial infection and cardiovascular disease but the causality is unclear. However intervention should always be there to prevent or to control systemic immmuno- inflammatory reaction from bacteria and their products in sulcus/pocket.

Role of genetics in periodontology

While microbial and other environmental factors initiate and modulate periodontal disease, individuals are known to respond differently to common environmental challenges, and this differential response is influenced by the individual's genetic profile. Genes clearly play a role in the predisposition to progression of periodontal Diseases (Hart TC 1996**).** Research on genetic polymorphisms has only had limited success in identifying significant and reproducible genetic factors for susceptibility to aggressive periodontitis and chronic periodontitis. Taking together the data published on gene polymorphisms in aggressive periodontitis and chronic periodontitis, there are differences among the various studies for the R allele carriage rates, even if the study populations are of the same ethnic background. Nevertheless, there is some evidence that polymorphisms in the IL1B, IL1RN, FcγRIIIb, VDR and TLR4 genes may be associated with aggressive periodontitis, and polymorphisms in the IL1B (Kornman et 1997), IL1RN, IL6, IL10, VDR, CD14, TLR4 and MMP1 genes may be associated with chronic periodontitis susceptibility as a single genetic factor in certain populations. In contrary to this, some other studies did not found association. However, most of the results have not been replicated in study cohorts with 100 or more individuals. It should be emphasized that assessment of genetic in predisposing to periodontitis represents only one of several components of individual-based risk analysis. Although a candidate gene common to all forms of periodontitis has not been identified, it is evident that, at the individual level, disease expression results from the interaction between genetic, bacterial and lifestyle (e.g. smoking) factors. This genetic information would

be valuable in therapeutic intervention on individualized approaches and preventive strategies of the development of periodontitis.

Role of virus in periodontal disease

Although the critical role of bacteria in the development of periodontitis is universally recognized, bacteria alone seemun unable to explain the site-specificity and other characteristic features of the disease (Slots 2005). It is not understood why, in hosts with comparable levels of risk factors, some periodontal infections result in loss of periodontal attachment and alveolar bone while other infections are limited to inflammation of the gingiva with little or no discernible clinical consequences. Also, many periodontitis patients do not show a remarkable level of classical risk factors. A plausible etiopathogenetic explanation for destructive periodontal disease includes interactions among herpes viruses, specific bacteria and immune reactions. Various studies found association of virus in periodontitis sites (Contreras et al. 2000, Michalowicz et al. 2000). Nevertheless, despite a large body of compelling research data, definitive proof is still asking that periodontal herpes viruses play a causal role in periodontitis development and do not occur merely as an epiphenomenon to the periodontal disease process. To sum up, the pathogenic significance of periodontal herpes viruses constitutes an open and promising frontier of research. The concept of herpes viral infections of the diseased periodontium may guide in a new level of understanding of the importance of preventing and controlling periodontal diseases for medical purposes (Slots et al 2015).

Adequate width of attached gingiva

Adequate zone of gingiva was considered critical for maintenance of marginal tissue health and for the prevention of continuous loss of connective tissue attachment (Oschenbein 1960). Narrow zone of attached gingiva with shallow vestibule accumulate food particles during mastication, impede proper oral hygiene. However later various other studies found that thin zone of attached gingiva have the same resistance as wide zone of attached gingiva. However in some cased augmentation for increasing the width of attach gingiva is justifiable for the enhancement of plaque removal around the gingival margin, to improve esthetics and to reduce inflammation around restored tooth.

Role of stress in periodontium

Psychologic factor particularly stress have been implicated as a risk indicator of periodontal disease. Stress and psychosomatic disorders most likely impact the periodontal health through changes in the individual behavior and through complex interaction among the nervous, endocrine and immune system. Several clinical studies and systemic review documented a positive relationship between psychosocial stress and chronic forms of periodontal disease (Genco 99, Peruzzo 07). In contrast Monterio de Silva et al in 2007 failed to find a relationship between psychologic factors and periodontal disease. So, in conclusion despite the well-known association between stress and NUG, confirming the connection between psychologic condition and other form of periodontal disease (chronic periodontitis) has been elusive. These relation are difficult to elucidate because as with many common disease, the etiology and pathogenesis of periodontal disease is multi-factorial and the role of individual risk factor are difficult to define. So this topic remained controversial.

Osteoporosis and susceptibility to periodontitis

Although studies in animal models indicate that osteoporosis does not indicate periodontitis, evidence indicates that the reduced bone mass seen in osteoporosis may aggregate periodontal disease progression. However reports in human are conflicting. More prospective, longitudinal studies evaluating the effect of estrogen and osteopenia/ osteoporosis on periodontitis are needed to improve our understanding (Klokkevold and Mealey).

Nutritional influences in periodontal disease

The following observation have been made in protein deprived animals: degeneration of connective tissue in gingiva and periodontal ligament, osteoporosis of alveolar bone, impaired deposition of cementum, delayed wound healing, and atrophy of tongue epithelium, osteoporosis result from reduced deposition of osteoid. This secondary changes can reduce the normal resistance to plaque induced periodontal disease. Vitamin C deficiency alone does not cause periodontal destruction. Local factors are required for increased probing depth and attachment loss to occur. However, acute vitamin C deficiency accentuated the destructive effect of gingival inflammation on the underlying periodontal ligament and alveolar bone. Deficiency of other vitamins (A,B, D,E,K) although some experimental animal studies found association to gingivitis and periodontitis, human studies showing association are lacking. To sum up there are no nutritional deficiencies that by themselves can cause gingivitis and periodontitis. However nutritional deficiencies can affect the condition of the periodontium and thereby may accentuate the deleterious effect of plaque induced inflammation in susceptible individuals.

Use of systemic antibiotics in periodontal disease

Antibiotic can be used as an adjunct to mechanical treatment in some condition (example NUG, periodontal abscess, aggressive periodontitis and recurrent cases). Use of antibiotic in chronic periodontics remained controversial. Systematic review implies that systemic antibiotic, in conjunction with SRP can offer an additional benefit over scaling and root planing alone in terms of probing depth reduction, clinical attachment gain and reduced risk of additional clinical attachment loss (Herrera et al 2002, Haffajee and Socransky 2003). However, owing to heterogeneity of the study design employed to access the effect of systemically administrated antimicrobial agents, it is difficult to reach a conclusion whether clinical benefits are there or not. If yes, then it is not clearly known which is is the best drug and doses and what is the most effective timing of drug administration in relation to mechanical therapy. Antibiotic strength 500 times greater than the usual therapeutic dose may be needed to be effective against bacteria arranged in biofilm (Greenstein 2005). So mechanical treatment is required for thorough removal of plaque before any antibiotic intervention. However overuse of the drugs has contributed to the emergence of resistant microorganisms that has become a major public health problem. Another potential problems associated with antibiotic use is the collateral damage to the human microbiome that may have negative long term consequences. So, all these factors should be taken into consideration.

Use Laser in periodontal therapy

Lasers have the advantages of bactericidal effect, detoxification effect, removal of the epithelium lining and granulation tissue which are desirable properties

for the treatment of periodontal pockets. Furthermore, potential biostimulation effects of scattering and penetrating lasers on the cells surrounding the target tissue during irradiation might be helpful for the reduction of inflammation and healing of periodontal tissues. Considering the various advantages of laser irradiation, its use in combination with conventional mechanical treatment or alone has the potential to improve the condition of the periodontal pockets more than mechanical therapy alone. Due to an excellent soft tissue ablation capacity, CO_2 lasers have been successfully used as an adjunctive tool to deepithelialize the mucoperiosteal flap during traditional flap surgery (Centty et al. 1997). Diode and Nd: YAG lasers were mainly used for laser-assisted subgingival curettage and disinfection of the periodontal pocket with various degrees of success (Moritz et al. 1998, Liu et al. 1999). However, controversy exist of usefulness of these lasers, as because several studies reported on thermal side effects, such as melting, cracking or carbonization when CO_2 and Nd: YAG lasers used directly on root surfaces. In contrast, the ability of the Er:YAG laser to effectively ablate dental calculus without producing major thermal side effects to adjacent tissue has been demonstrated in numerous studies (Aoki et al. 1994, Schwarz et al. 2003). Controlled clinical trials (Schwarz et al. 2003) and case report studies have indicated that non-surgical periodontal treatment with an Er:YAG laser may lead to significant clinical improvements as evidenced by probing depth reduction and gain of clinical attachment. So in conclusion based on the research so far, the Er:YAG laser holds promise as a useful tool to debride safely and effectively both the root surface and gingival tissue of the periodontal pockets, and peri-implant tissue.

Photodynamic therapy

PDT combines the use of a photoactivatable non-toxic chemical agent (photosensitizer) with low-level light energy (Chan & Lai 2003). The role of photodynamic therapy as a local treatment of oral infection, either in combination with traditional methods of oral care, or alone, arises as a simple, nontoxic, non-invasive and inexpensive modality with minimal risk of microbial resistance. Studies found SRP combined with PDT led to significant improvements of the investigated parameters over the use of SRP alone (Andersen et al 2007, Braun et al 2008). Some other studies did not fond additional benefit of PDT compared to SRP alone (Ruhling et al 2009, Al-Zahrani et al 2009). A systematic review published in 2011 concluded that the use of photodynamic therapy adjunctive to SPR provide short term benefit, but microbiological outcomes were contradictory. No evidence of effectiveness for the use of antimicrobial photodynamic therapy as an alternative to SRP was found Sgolastra et al 2013). So in conclusion, despite the abundance of promising data on the advantages of its use, there is still controversy regarding the real benefits of antimicrobial photodynamic therapy in periodontal treatment.

Full mouth disinfection

The full mouth disinfection approach aimed at preventing re-infection from untreated pocket (Quirynen et al 1995). The full mouth disinfection protocol includes full mouth scaling and root planing (SRP) in 24 hours, in addition twice daily chlorhexidine mouth rinsing, tongue scraping, chlorhexidine tonsil spraying and subgingival irrigation with chlorhexidine 3 times within 10

minutes and repeated after 8 days. The full mouth SRP protocol includes full mouth SRP without antiseptics. At the European Workshop on Periodontology in 2008 the question of the effects of full-mouth debridement, with and without antiseptics, in patients with chronic periodontitis was addressed. Based on evaluation of the systematic reviews by Lang et al. (2008) and Eberhard et al. (2008), the consensus of the workshop was that full-mouth debridement and full-mouth disinfection do not provide clinically relevant advantages over conventional staged quadrant-wise scaling and root planing in the treatment of patients with moderate to advanced periodontitis. Furthermore, the clinical recommendations given were that (a) all three modalities may be recommended for debridement, and (b) clinicians should choose the modality of debridement according to the needs and preferences of the patient, their personal skills and experience, the logistic setting of the practice and the cost-effectiveness of the therapy rendered. It should be noted that the performance of optimal oral hygiene practices is an inseparable principle to be observed with any protocol of mechanical debridement.

Periodontal regeneration

Guided tissue regeneration (GTR) represents the most well documented regenerative procedure for obtaining periodontal regeneration. Guided tissue regeneration has demonstrated significant clinical improvements beyond that achieved with debridement alone in treating periodontal defects. An added benefit may be obtained by the use of bone grafting materials in combinition with guided tissue regeneration. But the added benefit of bone graft sometime may not a true regeneration but a simple repair by formation of long junctional epithelium. Bone graft material also can disappears over time or become encapsulated by fibrous tissue or become surrounded by bone leading to failure. In wide defect, particularly wide two wall and one wall defect and grade two, grade three furcation defect usefulness of GTR is controversial and success is not predictable. So the question arise here is that what will the future bring in terms of the ability to reverse the destructive processes that result from periodontitis in predictable way? These may include novel barrier-membrane technologies, the use of growth factors and other cell stimulating proteins, and gene-delivery approaches, tissue bioengineering, use of stem cell to regenerate the periodontium (Ramseier et al 2012, Hynes et al 2012).

Conclusion:

In the last 50 years there have been many technological advances in the methods used for the clinical examination of periodontal tissues. We now have a better understanding of the etiological factors associated with periodontitis, the mechanism involved in periodontal wound healing and inter relationship between patients factors (such as smoking and diabetes) and treatment outcomes. In addition to great volume of research and investigated work, education and teaching in periodontology has been unpinned by the critical evaluation of both old and new concepts.

So in near future we can expect that current controversial topic will get general agreement. However it is also possible that new controversial topic may come.

References

1. Hynes K, Menicanin D, Gronthos S, Bartold PA. Clinical utility of stem cells for periodontal regeneration Periodontology 2000 2012;59: 203–227.
2. Karring T, Cortellini P. Regenerative therapy: furcation defects. Periodontology 2000 1999; 19: 115-137.
3. Laine MJ, Crielaard W, Loos BG. Genetic Susceptibility to Periodontitis. Periodontology 2000 2012; 58: 37–68
4. Lindhe J, Lang N. Clinical Periodontology and implant dentistry. 5th edition.
5. Newman MG, Carranza FA, Takei H, Klokkevold PR. Carranza's clinical Periodontology. 10th ed. Elsevier health sciences; 2006.
6. Newman MG, Carranza FA, Takei H, Klokkevold PR. Carranza's clinical Periodontology. 11th ed. Elsevier health sciences; 2011.
7. Sculean A, Schwarz F, Berakdar M, Windisch P, Arweiler NB, Romanos GE. Healing of intrabony defects following surgical treatment with or without an Er:YAG laser: A pilot study. J Clin Periodontol 2004; 31: 604–608.
8. Slots J. Herpesviruses in periodontal Diseases. Periodontology 2000 2005; 38: 33–62.
9. Soukos NS, Goodson MJ. Photodynamic therapy in the control of oral biofilms. Periodontology 2000 2011; 55: 143–166.
10. Tomasi C, Wennstrom JL. Full-mouth treatment vs. theconventional staged approach for periodontal infection control. Periodontology 2000 2009; 51: 45–62.
11. Yoshie H, Kobayashi T, Tai H, Galicia J C. The Role of Genetic Polymorphisms in Periodontitis. Periodontology 2000 2007:43:102–132.
12. Ramseier C A, Rasperini G, Batia S, Giannobile W V. Advanced reconstructive technologies for periodontal tissue repair Periodontology 2000 2012; 59: 185–202.