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Commentary

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Oral Health, Dental Education and Research: A Commentary

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ABSTRACT

Despite major advances in oral health in some communities there is rampant dental and oral disease worldwide. Indeed dental caries and periodontal disease still top the league table as the commonest infections in humans. Though more research is needed there is evidence to indicate that oral disease is linked to systemic disease. Despite these revelations and the high prevalence of oral disease, the dental education systems worldwide are facing fundamental challenges due to social, economic and technological reasons. These challenges will not be adequately addressed with anything less than a concerted response from the government, community, alumni, and industry. Only then can the faculties or schools of Dentistry flourish and provide a stimulating and quality environment for graduates to develop their full potential and to hone their competitive advantage for a challenging career in a fast-changing world. In this article the author describes the current dental landscape globally, and then addresses the multifarious challenges in education, research, and resource support that constantly challenges the delivery of dental pedagogy worldwide.

ORAL HEALTH AND THE DISEASE BURDEN: THE CURRENT LANDSCAPE

Dentistry has made great strides over the last century since the days of the Barber Surgeons. One of the major changes that led, for example, to the reduction in caries is the discovery of the properties of fluoride, and the observation that people who lived in communities with naturally fluoridated drinking water had far less dental caries than people in comparable communities without fluoride in their water supply. Community water fluoridation is arguably one of the great achievements of public health in the twentieth century, and the most far reaching of all in dentistry.

Although we have come very far from the times when old age was equated with edentulism there are significant disparities in global oral health. Indeed, there is a silent epidemic of dental and oral diseases in many underserved populations worldwide. This disease burden restricts activities in school, work, and home, and often significantly diminishes the quality of life. Those who suffer the worst oral health are found among the poor of all ages, with poor children and poor older citizens particularly vulnerable. Members of racial and ethnic minority groups also experience a disproportionate level of oral health problems as seen in US Native Indians and Australians aboriginal populations - even in so-called developed countries. The situation in the developing world is far from satisfactory as the consumption of sugary diets is increasing disproportionately without dental care and proper oral hygiene. Other smaller communities that are so disadvantaged include the medically compromised or who have disabilities.

The reasons for disparities in oral health are complex because of socioeconomic factors, as well as apathy in ignorance in poorer communities. In many situations such disparities are exacerbated by the lack of community programs such as fluoridated water supplies.

It has been said that the mouth is mirror of general health and well-being. General

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health risk factors common too many diseases, including diabetes, tobacco use and poor dietary practices, also affect oral and craniofacial health. The almost direct association between tobacco use and oral diseases has been well established for some decades now. Recent research findings have also pointed to possible associations between periodontal disease and oral inflammation and diabetes, atherosclerotic vascular disease, low-birth-weight and premature births, and lung disease in the hospitalised, kidney disease and pancreatic cancer. It should, however be stressed that these are associations only, and more work needs to be done to elucidate further the oral-systemic disease axis.

DENTAL EDUCATION

Dental education is the fountain from which all dentistry flows. It is the framework on which scientific findings and advancements in oral care are discovered and implemented. A strong dental education system produces a strong dental profession, resulting in the highest level of oral health care for the public.

The strong association between dental education and improved public oral health is well known. But in order for dental education to continue to play its vital role, it is important to ensure a strong supply of qualified, diverse dental students and faculty who have access to state-of-the-art teaching and research facilities.

Owing to the one-on-one teaching and very close supervision necessary, dental education is among the most costly professional training programmes worldwide. While costs continue to grow, government support has gradually declined over the years. The other aligned challenge is the worldwide shortage of dental educators. A scholarly and adequate, full-time dental faculty is essential to appropriately train future generations of students. This is also necessary in order to sustain the current level of research, so that the faculty maintains its scientific standing and credibility in the university community both at local and international levels.

There are several reasons for the widespread shortage of dental educators. University salaries and benefits are not keeping pace with those in industry and private practice, and the gap is ever-widening. Hence, young graduates are attracted to the lucrative arena of general practice rather than face the challenges of academia. These challenges include competing for research grants, an ever-increasing administrative burden, and having to complete more and more training pathways prior to gaining specialist status. All of these activities restrict the time that can be devoted to scholarship. In the West, the shortfall in faculty personnel is further exacerbated by graduates leaving dental school with a heavy burden of student debt that they want to discharge by joining a financially rewarding private practice. Furthermore, attracting potentially high-earning dental specialists to a career in dental education continues to be an issue. Well-trained, scholarly dental specialists will be increasingly crucial to help dental schools maintain first-class teaching facilities.

Furthermore, keeping pace with today's escalating technological advancements is a major challenge for most dental schools worldwide. The new technologies produce new contraptions such as lasers and modern imaging machines (e.g. cone-beam radiology) that provide precise images of hard and soft tissues. Yet, these are beyond access to resource starved dental schools. However, it behooves us to ensure that our dental students graduate with knowledge of and skills in using technology and equipment that are not already obsolete in the field.

DENTAL RESEARCH

In the 21st century, dental schools must have the capacity to conduct collaborative research, and equal participants in medical research programmes. Dental academics, in alliance with industry and research grant councils (eg the National Institutes of Health in the United States), have developed new and innovative products and equipment, such as fluorides and implants. Research has also led to advances in orthodontics and oro-facial reconstruction that improve a patient's self-esteem and daily functioning. Dental schools provide the greatest potential to turn clinical findings into practical applications through their connections with students, practising dentists, industry, community clinics, and public-policy advocates. In addition, dental schools play a crucial role in provision of life long learning as Continuing Professional Education courses to graduates and alumni in an increasingly fast pace world with explosive technological developments.

If inadequate resources are provided for dental research, then advances looming on the horizon might never come to fruition. Imagine the impact on the oral and general health of the public if dental researchers developed a vaccine to prevent periodontal (gum) diseases or discovered gene therapies to prevent oral cancers. Furthermore, if dental schools were to survive within a university environment it is imperative that they pursue quality research that compete with various other research programmes in other allied faculties. The research that is so pursued should be aligned to Nuffield principles of scholarship of discovery, scholarship of integration, scholarship of application, scholarship of translation and scholarship of teaching. The latter translational research and the interface between laboratory research and public understanding of research are key areas that need to be addressed if we need to sustain the momentum in dental research. Otherwise dental schools in the universities will face an existential threat of being relegated to mere technical schools.

CONCLUSION

It is clear that the challenges facing dental education

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worldwide are very similar. These challenges will not be adequately addressed with anything less than a concerted response from the government, community, alumni, and industry. Only then can the Faculties or Schools of Dentistry continue to provide a stimulating and quality environment for graduates to develop their capabilities and to hone their competitive advantage for a challenging career in a fast-changing world, and contribute to the frontiers of dental discovery.

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Case Report

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Oral Self-Injury: Report of a Case with Review of Literature

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ABSTRACT

Self-inflicted oral injuries are commonly seen in certain syndromes and systemic disorders. Most frequently affected regions of the body are the oral and perioral tissues, hands and neck. Although no medical treatment is available, timely dental intervention is essential to prevent further complications. The aim of this paper is to report a unique case of oral self-injury in a 10-month-old infant which was successfully treated with conservative therapy and also to briefly review the literature.

KEYWORDS: Oral self-injury; Tongue biting; Finger biting.

INTRODUCTION

Self-injury or self-mutilation can be defined as a behavioural disturbance that consists of deliberate disturbance of or damage to body tissues but is not associated with a conscious intent to commit suicide.[1] A lesion is considered to be a self-injury if it satisfies the following features: must be socially unacceptable, repetitive and causes mild or moderate tissue damage.[2] Cuts, burns, scratches, blunt injury, bites, and interference with wound healing are the most common forms of self-injury.[3] Self-injury can affect individuals of any age, sex, or ethnic group but its frequency has found to increase amongst adolescents and young adults.[4]

REVIEW OF LITERATURE

Cannavale, et al. concluded that oral self-injury was uncommon in clinical practice and presented itself as the first sign of psychiatric disorder.[5] Ragazzini, et al. introduced a new technique, a resin mouthguard to obtain immediate healing of the oral lesions.[6] Limeres, et al. proposed that treatment for oral self-injury must be individualized as there is lack of treatment protocol.[7] Arhakis designed a maxillary intraoral appliance to prevent oral and perioral self-injury.[8] Santos, et al. concluded that association of different laser therapies was effective in establishing painless mastication.[9] Pretty, et al. alerted that the odontologists should always consider self-injury as an explanation for intra-oral injuries of unidentified origin.[10] Silva, et al. emphasized on early detection and intervention to enhance the patient's quality of life.[11] Jackson used a tongue depressing stent to prevent self-injury in a comatose patient.[12] Gutman, et al. reported a case of congenital analgesia in which the injury to tongue was due to erupted lower incisor teeth.[13]

CASE REPORT

A ten-month-old boy was referred to the Department of Pediatric and Preventive Dentistry by a Pediatrician for evaluation and treatment of large ulceration present on the ventral surface of the tongue, noticed by his parents 1 month back. Physical examination revealed the presence of 2×2 cm large ulcerated lesion on the ventral surface of the tongue, covered by pseudomembranous slough and surrounded by erythema (Figure 1). Also the tongue was bifurcated

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at its tip. Lesion was present since last one month and the parents observed the infant moving his tongue over his erupted teeth on multiple occasions.

Patient was not using any medication for tongue lesion.

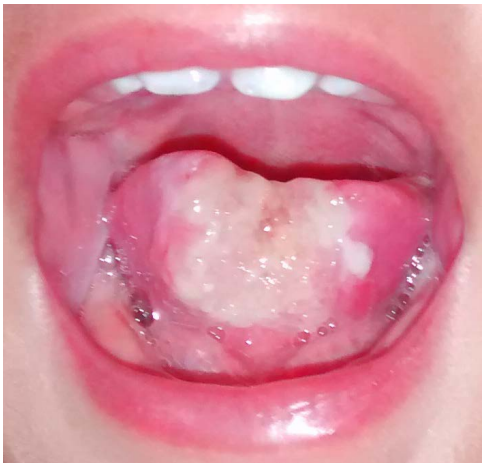

Figure 1: Clinical image showing ulcerated lesion on the ventral surface of the tongue.

On examination it was observed that finger of the right hand had a raw wound and necrosis was evident (Figure 2).

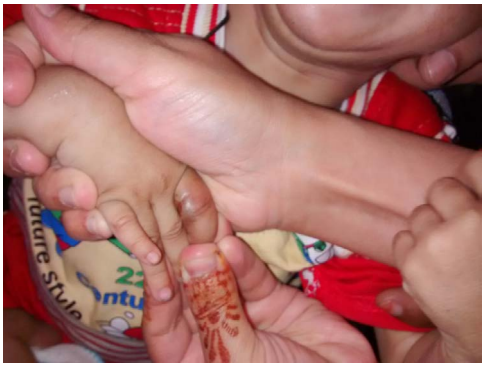

Figure 2: Raw wound evident on the finger of the right hand chewed by the patient.

Family history was negative for developmental disorders and congenital syndromes. The parents had history of consanguineous marriage. The patient had history of pneumonia one week after birth. Presently the patient was undergoing treatment for infection in blood and urine. When complete blood count was done it was found that hemoglobin level was low i.e. 9.7 g/dL and leukocytosis was observed as the WBC count was 20.1 thou/microlitre. The level of C-reactive protein in the serum was found to be high i.e. 13.6. Incisional biopsy was employed to rule out oral mucosal disease.

In the present case parafunctional activity or habits were ruled out and conservative therapy was planned for tongue lesion. Mandibular primary incisors were grinded to avoid further injury to the tongue. The patient was recalled after 1 month and examined. It was noticed that the tongue had healed (Figure 3).

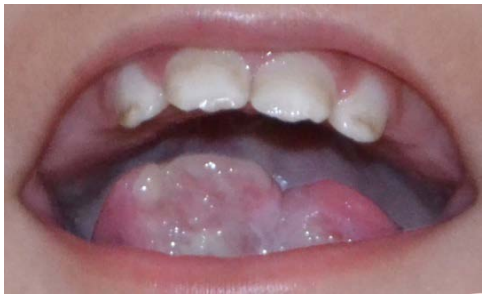

Figure 3: Clinical image showing healing of the tongue after 1 month.

DISCUSSION

The role of dentist is imperative in the management of self-mutilating behaviour, as teeth are the main cause of self-injury. Infants with such habits pose a challenge for the clinician. Self-injury is commonly seen in child patients in genetic syndromes such as Lesch-Nyhan syndrome, Cornelia de Lange and Gilles de la Tourette syndrome.[14-16] It has also been observed in mental retardation,[15,17] congenital malformations[15,18] and infectious disease such as encephalitis.[15,19] Finger-biting can also be a manifestation of neuropathies.[20]

Oral mucosal disease was ruled out through employment of incisional biopsy.

Various therapeutic approaches have been proposed in the literature. Preventive intra-oral devices have been designed, but have the disadvantage of extensive fabrication time, associated fungal infections and low acceptance rate in case of infants. Selective grinding of tooth cusps can be done. Acrylic splints or cast silver splints can be given to prevent gross laceration of the tongue or fingers. Extraction of teeth is advisable in severe cases if the lesion is not resolving.[20]

A thorough clinical evaluation is required to detect such habits as they may be associated with some systemic disease or be the first sign of psychiatric disorder. Prevention should be the standard of care for such patients. Careful monitoring is of paramount importance and is recommended to prevent possible complications.

CONCLUSION

- Early detection and intervention of self-injurious habits is essential part of preventive dentistry. The importance of accurate history taking and examination of suspected cases cannot be underestimated and helps to rule out other habits and parafunctional activity.
- In general it is prudent to rule out oral mucosal disease through employment of incisional biopsy in longer standing non-healing lesions.
- Dentists should be aware of the clinical signs and treatment

options for child patients with self-mutilation habit.

- Optimal management of patients with self-injurious habits depends on a lot of factors i.e. age, severity and cooperative potential and requires careful monitoring.

SOURCES OF SUPPORT: Nil.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONSENT

The patient has provided written permission for publication of the case details.

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Review

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Managing Denture Biofilm Related Diseases

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ABSTRACT

The oral cavity harbors more than 700 microbial species and is one of the most complex ecosystems ever described. While the majority of these microbes are considered commensal, some of them are responsible for oral infectious diseases such as dental caries, periodontitis, halitosis and stomatitis. The advancement of modern science has greatly furthered our understanding of oral microbes and their roles in host health and disease. It has also led to the development of new tools for early detection, effective treatment, and prevention of oral microbial infections. This perspective provides a general understanding of oral microbiology, and its clinical relationship to oral infectious diseases, with a specific focus on denture-related microbial infections. The perspective also discusses the potential for developing innovative interventions for managing denture-related disease based on recent advances in our understanding of oral microbiology and denture-associated biofilms.

KEYWORDS: Stomatitis; Oral microbiota; *Candida albicans*.

ABBREVIATIONS: SAP: Aspartyl proteinases; PL: Phospholipase; PDT: Photodynamic therapy; STAMPs: Specifically Targeted Antimicrobial Peptides.

INTRODUCTION

The association between microbes and oral diseases had long been suspected. Dr. W. D. Miller is generally recognized as the father of modern dental microbial pathogenesis. His 1890 seminal book titled *Microorganisms of the Human Mouth*[1] makes the first connection between bacteria in dental plaque and tooth decay, and remains a foundation of current understanding of dental disease. For a long time, oral microbes had been indiscriminately regarded as pathogens. In fact, their removal from the oral cavity has become the main objective of dentists. Not until recently, did we realize that like microbes associated with other parts of the human body, most of the oral microorganisms are commensal and might have protective role in preventing the colonization of pathogens.[2,3] More importantly, increasing evidence suggests that oral infectious diseases such as dental caries and periodontitis are often the result of the disturbed host homeostasis, and an imbalanced oral microbial ecology often leads to overgrowth of otherwise low abundant opportunistic pathogens.[4,5]

Recent advances in molecular biological techniques are broadening our understanding of bacterial diversity and the societal community interactions which occur between species in the oral cavity.[6] This has led to tremendous advances in our understanding of oral microbiology and its involvement in health and disease, including tooth decay, gum diseases, as well as the diseases associated with artificial dental apparatus introduced through modern dentistry.[6,7]

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ADVANCING UNDERSTANDING OF ORAL MICROBIOLOGY THROUGH MOLECULAR BIOLOGICAL APPROACHES

Our bodies are home to a multitude of microbial organisms that form distinct microflora inhabiting the gut, skin, vagina and oral cavity. These microbial communities have been of great interest to scientists in recent years due to their impact on host health and disease. Increasing lines of evidence indicate that these commensal microbiotas have important metabolic, trophic, and protective functions and greatly affect the host's physiology and pathology.[8,9] For example, the importance of the gut flora in digesting unutilized substrates, training the immune system and protecting against epithelial cell injury is well appreciated,[10-12] and we are beginning to understand its potential role in systemic diseases, such as inflammatory bowel disease[13,14] and obesity.[15]

Molecular biological tools have been critically important for identifying the diversity of these host-associated microbiotas, including the oral microbial community.[16] Prior to the availability of such tools, determining the diversity of complex microbiological communities, such as those of the oral microbiota had been essentially dependent on the ability to culture and identify individual organisms. However, we then realized that only a small fraction of the organisms comprising these microbial communities has been isolated.[6] In fact, accumulating lines of evidence suggested that there are extensive physical and metabolic interactions between different microbial species within the same community, which are essential for the growth and persistence of certain microbes[17] and make them recalcitrant to cultivation. The power of molecular biological approaches, such as culture-independent 16S rRNA gene sequencing-based methods allows us to identify yet-uncultivable species and provides a more comprehensive and detailed inventory of human oral microbiota.[6]

The studies using culture-independent approaches have revealed the sheer magnitude of the diverse microbes, including yet-uncultivable species residing within the oral cavity.[18,19] The human mouth is estimated to harbor more than 700 different bacterial species, comprising one of the most complex microbial flora.[18] The diversity of microorganisms that inhabit the oral cavity includes bacteria, archaea, protozoa and fungi.[20,21] An interesting perspective regarding diversity of the oral flora is the presence of Archaea as a constituent.[22] Phylogenetically, Archaea is among the oldest known type of prokaryotes; it has previously been isolated from ocean bottom, and yet also appears to be a colonizer of the human oral cavity with yet-to-be-determined role in oral microbial ecology.[23] The diversity of the microbial flora reflects tremendous genetic information and immense bio-physiological potential that may have huge impact on host health and disease. If we consider that an average bacterial species has 2,000-6,000 genes, then an oral bacterial population of some 700 individual species represents a pool of over 1 million genes, 10 times more than human host genes. This provides the oral microbial environment with a huge quantity of information related to unique metabolic pathways, the generation and secretion of various factors that can control and modify their ecological niche, and factors that may impact function of the human host.

THE STRUCTURE OF DENTAL AND DENTURE PLAQUE

Bacteria in the oral cavity often reside within biofilms, such as those that form dental and gingival plaque.[24] For edentulous and partially edentulous individuals who wear dentures, a denture-associated biofilm, or denture plaque, forms on the denture surface and could potentially serve as a reservoir of pathogenic microbes for infections.[7]

Dental and denture plaque are not simple matrices. They consist of a diverse collection of microbial species, and furthermore have a highly organized structure in which different species can occupy specific sites or niches within the biofilms.[25] During dental biofilm formation, bacteria that are early colonizers, such as Streptococci (i.e., *S. gordonii*, *S. oralis*, etc.) with specific adhesins can effectively bind to proteins deposited as a pellicle coat on the tooth surface. This is followed by the subsequent recruitment of intermediate and late colonizing species through cell-cell coadhesion *via* specific adhesin-receptor interactions.[25] These specific bacterial physical associations eventually generate a highly structured microbial community, which we recognize as dental plaque or biofilms.[5,6,26] Furthermore, bacterial species within dental biofilm are often engaged in extensive signaling and metabolic interactions to ensure their survival within the microbial community.[6]

Dental biofilms of healthy subjects harbor a commensal oral microbial community with properties that limit the invasive potential of opportunistic pathogens.[3,27] And likemost ecological communities, once established, dental biofilm generally has a stable and controlled population of different organisms and displays resilience to environmental disturbance.[28,29] However, as will be discussed in the following section, the microbial composition of denture biofilm flora and their pathogenic potentials could differ significantly from that of healthy dental biofilm, thus contributing to the pathogenesis of denture-biofilm related diseases, such as stomatitis.

CONNECTIONS BETWEEN MICROBES AND ORAL DISEASES ASSOCIATED WITH DENTURE WEARING

Denture stomatitis is a common disorder in subjects wearing dentures, which are prostheses that provide important functional and esthetic improvements for edentulous and partially edentulous patients.[30,31] The disorder is characterized as inflammation and erythema of the oral mucosal areas covered by the denture. The current view regarding the etiology of denture stomatitis is that it is a multifactorial infectious disease. It involves a number of associative factors, including denture-induced trauma, continual denture wearing and denture plaque harboring pathogenic microbes, such as Candida.[30] Among those

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factors, the microbial biofilm formed on the denture surface plays a significant role in contributing to the disease pathogenic process. Whereas the normal commensal oral microbial community could prevent infection by interfering with the invasive potential of opportunistic pathogens, this is altered in the denture biofilm. Indeed, the microbial composition of the biofilm which forms on denture surfaces differs significantly from that observed in the oral cavity of healthy individuals.[32,33] This could be due to the fact that denture wearing can alter normal oral physiology by affecting normal salivary flow that plays an important role in shaping the microbial community.[34] Meanwhile, the older segment of the population often has a comparable higher proportion of denture wearer.[35] These individuals are more likely to have systemic health conditions and could have already had imbalanced oral microbial ecosystem due to disturbance in host homeostasis. Furthermore, a denture provides a unique abiotic surface for microbial colonization, which often leads to the development of a denture biofilm with microbial composition and structure different from normal oral biofilm.[7,32]

Compared to the normal oral and dental biofilms, denture biofilmsare associated with a much higher occurrence of *Candida* yeasts, particularly *Candida albicans*.[36] *C. albicans* is a commensal fungal species commonly colonizing human mucosal surfaces. It co-exists with diverse oral microbial species and has long been adapted to the human host.[37] In healthy individuals, *C. albicans* is usually a minor component of their oral microfloras. However, under conditions of immune dysfunction or local predisposing factors such as poor oral hygiene or ill-fitted dentures, colonizing *C. albicans* can become an opportunistic pathogen. In these patients, *C. albicans* becomes more predominant and invasive, causing recurrent mucosal infections such as denture stomatitis.[38] The presence of *C. albicans* on denture and oral mucosal surfaces of denture wearers is positively associated with denture stomatitis.[39] The virulence factors of *C. albicans* have been well documented.[40] Among them, multiple host recognition biomolecules, such as Als1p and Hwp1p,[41,42] as well as the secreted enzymes, including Aspartyl proteinases (SAP)[43] and Phospholipase (PL)[44] have been shown to play important role in determining *C. albicans'* pathogenicity. Meanwhile, its polymorphic growth patterns[40] as well as phenotypic switching[45] have also been implicated in contributing to its virulence. While *C. albicans* infection cannot be claimed as the single causal pathogen for inducing denture stomatitis, it has a strong associative presence when the disorder occurs, and its eradication from denture and mucosal surfaces is associated with reversal of the condition.[46,47] Hence, it is generally accepted that *C. albicans* is a main opportunistic pathogen which is involved in the development and pathogenesis of denture stomatitis. Meanwhile, certain bacterial species, such as *Prevotellasp*., *Veillonellasp*. and *Staphylococcus sp*. have been found to be enriched in denture biofilms,[48,49] although their potential role in denture stomatitis pathogenesis remains to be determined. More importantly, increasing lines of evidence indicate the extensive Candida-bacterial interactions, which could impact their pathogenicity.[37] For example, co-infection of *C. albicans* and *Staphylococcus aureus* has been shown to lead to increased mortality in animal model.[50] A better understanding of the physiology of Candida and bacteria co-existence and the inter-kingdom Candida-bacterial interactions would shed light on the impact of polymicrobial infection on the etiology of denture-related stomatitis.

TREATMENT OF DENTURE-RELATED INFECTIOUS DISEASE

Plaque formed on the denture surface often serves as a reservoir of opportunistic pathogens, including *C. albicans* for infections. In addition to maintaining good general oral hygiene, the most recommended approach to managing and preventing microbial-related disease associated with denture use is for patients to maintain a high level of denture hygiene by appropriate cleaning.[51,52] Common approaches to denture cleaning utilized by patients include brushing with abrasive cleansers, such as toothpastes, and washing or soaking dentures using commercial chemical cleansers with antimicrobial compounds designed for this purpose. The latter is preferred, as brushing with abrasive cleansers has been shown to be less effective for removal of the biofilm, and furthermore, can roughen the denture surfaces and result in more rapid bacterial adherence and biofilm growth.[7,53] However, as observed with other biofilms, a problematic issue associated with denture plaque is that it reduces the effectiveness of antimicrobial, including antifungal treatment.[54] The mechanisms by which biofilm environments enhance antimicrobial resistance are not fully understood. However, putative mechanisms likely include decreased ability of the antimicrobial agents to penetrate and diffuse within the biofilm matrix, protective functions conferred to the putatively susceptible bacteria due to slower growth rates and even changes in phenotype, and perhaps protective factors secreted by other microbes within the biofilm community which can degrade the applied antimicrobial agents.[55] In addition, the materials used in the manufacture of dentures can also affect adherence and colonization by microbes, including *C. albicans*, as well as impact the efficacy of antimicrobial treatment on thebiofilm.[56]

Soaking dentures in an appropriate commercial cleanser has been shown to be effective in removing attached microbes without increasing surface roughness.[57] Overnight denture removal is also important for controlling denture plaque, as it isolates the denture from salivary secretion that provides nutrients for microbial growth of denture biofilm. In addition to maintaining denture hygiene, various antimicrobials, including imidazole (clotrimazole, ketaconazole), triazole (fluconazole, itraconazole) and polyene (nystatin, amphotericin B) antifungals for treating Candida, and antibiotics for treating bacterial pathogens were also recommended for controlling denture-related mucosal infections.[58,59]

More recently, a new antifungal therapeutic approach Photodynamic (PDT) therapy has been used to treat denture stomatitis.[60] PDT uses a photosensitizing agent and light of appro-

priate wavelength. The interaction between the photosensitizer and light in the presence of oxygen produces reactive species that induce cell damage and death.[61] In a recent clinical trial, PDT was shown to be as effective as topical nystatin in the treatment of denture stomatitis.[62] Since PDT can effectively kill Candida species, including strains resistant to conventional antifungal agents,[63] it has been regarded as a promising method for the treatment of dental stomatitis. Recurrence of stomatitis is frequently observed within short period of time after stopping antifungal treatment.[64] This is likely due to reinfection by residual pathogens that remain within plaque on dentures and are resistant to treatment. Meanwhile, many patients failed to respond to the usual treatment, largely due to the development of drug resistance of candida species. For patients with systemic diseases, such as type 2 diabetes mellitus or being immunocompromised,[65] they often show less responsiveness to the treatment as well. When treating these patients, combined efforts including antifungal treatment and improving patients overall health status are critical in determining the outcome.[66]

The knowledge we are gaining from molecular biological studies of dental and denture biofilms is contributing to the development of novel therapeutic tools.[6,67] One approach is to build on our ability to identify specific pathogenic organisms that inhabit the biofilm, and develop therapeutics that specifically target these organisms. An example of this approach undertaken by our research group is the development of STAMPs (Specifically Targeted Antimicrobial Peptides).[68,69] A typical STAMP consists of two functional moieties conjoined in a linear peptide sequence: a nonspecific antimicrobial peptide serves as the killing moiety, whereas a species-specific binding peptide provides specific binding to a selected pathogen and facilitates the targeted delivery of the attached antimicrobial peptide. The feasibility of this approach has been demonstrated by the development of C16G2, a STAMP specifically targeting *S.mutans*, the bacterium known to cause dental caries. C16G2 has been shown to remove *S. mutans* within *in vitro* multi-species biofilms with high efficacy and specificity,[68,70] and is under further animal and human evaluations.[71] The successful demonstration of this targeted approach could serve as proof-of-concept for applying this technology to the treatment of denture-related Candida infections.

CONCLUSIONS

The past decade has witnessed significant advances in our understanding of oral microbiota. We now better understand the structural and functional complexity of dental and denture plaque, and a strong connection between oral microbial ecology and host health and disease has been established. It is well known that the control of microbial pathogens, such *C. albicans* on dentures and in the oral cavity is critical for the oral health of denture wearers. Continued efforts using modern scientific methods will help us develop more diagnostic tools and therapeutic interventions for the identification, treatment and prevention of denture infections. New and improved approaches will be able to treat and control denture infections with less physical damage to denture surfaces by providing improved mechanisms for killing and removing microorganisms in the denture biofilm. We can envisage products that will have targeted killing of selective pathogens without affecting other commensal species within the same denture biofilm. Finally, we can also expect to see new products that will be able to enhance natural oral immunity, and provide cavity protection or control gingival disease in dentate individuals, and other inflammatory disorders in denture wearers.

CONFLICTS OF INTEREST

The authors declare that Wenyuan Shi is an employee of C3 Jian, Inc. which has licensed technologies from UC Regents that could be indirectly related to this research project.

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Review

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Peri-Implantitis: A Review of the Disease and Report of a Case Treated with Allograft to Achieve Bone Regeneration

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ABSTRACT

Dental implants offer excellent tooth replacement options however; peri-implantitis can limit their clinical success by causing failure. Peri-implantitis is an inflammatory process around dental implants resulting in bone loss in association with bleeding and suppuration. Dental plaque is at the center of its etiology, and in addition, systemic diseases, smoking, and parafunctional habits are also implicated. The pathogenic species associated with peri-implantitis include, *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis,* and *Tannerella forsythia*. The goal in the management of peri-implantitis is the complete resolution of peri-implant infection with function. Therapies using various biomaterials to deliver antibiotics have been used in the treatment of peri-implantitis e.g. fibers, gels, and beads. The use of guided tissue regeneration barrier membranes loaded with antimicrobials has shown success in re-osseointegrating the infected implants in animal models. Several uncertainties still remain regarding the management of peri-implantitis. The purpose of this article is to present a background of peri-implantitis along with a case of peri-implantitis successfully treated for bone regeneration.

KEYWORDS: Peri-implantitis; Defect fill; Surgery; Peri-implant mucositis; Bone grafting; Implant failure.

ABBREVIATIONS: GTR: Guided Tissue Regeneration; ePTFE: expanded Polytetrafluoroethylene.

INTRODUCTION

Tooth loss occurs for a variety of reasons and results in resorption of the alveolar ridge if left unreplaced.[1] Achievement of successful periodontal regeneration provides a great challenge to the dental surgeons.[2] Dental implants are a successful in replacing teeth, with implant survival rates reported to be greater than 89% at 10-15 years follow up.[3-5] Around two million implants are placed each year and the number of implants placed per year is expected to increase both due to the success of implant therapies and the aging world population.[6] While success rates of dental implants are initially quite high, 6-12% of dental implants fail and are lost or must be removed.[4,7,8] Failures are due to biomechanical and/or biological imbalances.[6,9-13] High functional loads at the implant–bone interface due to bruxing/clenching or mechanical damage to implant superstructure leads to biomechanical failures.[6,9,12,14] Biological failures are associated with microbial plaque accumulation, bacterial infections, bone loss and sensory disturbances.[6,10,12,15,16] Early biological failures are associated with contaminated surgical implant

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placement or un-favorable healing response.[4,5,10,15] However, late implant failures are typically peri-implantitis, infections induced by plaque.[9,17,18] The consequences of this early or delayed, biomechanical or biological, peri-implant disturbances can lead to loss of implant.[19] Interventional therapies using non-surgical or surgical techniques with or without various biomaterials[1,20-22] and antibiotics have been employed in the management of peri-implant disease to prevent implant failure.[23,24] This article presents literature surrounding peri-implant disease and a clinical case involving a failing implant and its stabilization with allograft bone material.

PERI-IMPLANT DISEASE

Peri-implant disease is an inflammatory process that affects the surrounding tissues of a functional osseointegrated implant.[25] Peri-implant disease is the general term used to describe host tissue inflammatory reactions and is of two types. The first type is peri-implant mucositis which is defined as a reversible inflammatory reaction in soft tissues surrounding the dental implant.[6,26] The second type is the peri-implantitis, defined as an inflammatory process affecting the tissues (soft and hard) surrounding an osseointegrated implant resulting in loss of supporting bone and associated with bleeding and suppuration.[6] Peri-implantitis is usually the result of the disturbance of the equilibrium between the micro-flora and the defense system.[27] The soft and hard tissues surrounding an osseointegrated (bone-to-implant contact) implant shows some similarities with the periodontium in the natural dentition. It is important to realize that the diagnosis of peri-implant disease is not synonymous with implant failure. An infection of the implant does not automatically mean that the implant will fail.[6,10,12,28]

EPIDEMIOLOGY AND PATHOGENESIS OF PERI-IMPLANTITIS

Data from clinical investigations with up to 5 years follow-up show that the incidence of implants exhibiting peri-implantitis is low (0.3-14%) and that the highest incidence of implant loss is within the first 12 months after implantation.[4,5,7,12,29,30] However, this incidence rate could be an underestimation due to poor clinical diagnosis due to avoidance of probing around dental implants during routine clinical exams and the short duration of clinical studies which are less than 5 years. It may take more than 5 years for peri-implant disease to reach clinical expression.[6,10-12,31] Therefore it can be assumed that the incidence of peri-implant disease and implant loss may increase if longer evaluation periods were considered. It has been reported that ~11% failure rate is seen in patients who are smokers versus ~5% for non-smokers, which is attributed to impaired immune function and compromised healing in smokers.[32-35] It has been proposed that smoking cessation before implant placement can result in rates similar to those of non-smokers.[35] Patients with conditions such as osteopenia, osteoporosis, diabetes, hyper-inflammatory phenotype and bisphosphonate use, have been associated with increased risks of implant failure due to poor bone healing and infection.[12] Material surface, properties and design of dental implants also play a role in development of peri-implantitis. It has been observed that exposed rough surfaces tend to accumulate and retain more bacterial plaque than smooth surfaces and are more frequently surrounded by inflamed tissues.[10,36] Calcium phosphate coatings are used to promote osseointegration by increasing the surface area however they increase the risk for peri-implant infection in comparison to non-coated implants.[3,37,38] Peri-implantitis is complex and multivariate with respect to implant design and surface along with patient factors, such as systemic health, smoking, oral hygiene and periodontal disease.[39]

Although bacterial species are known to have complex symbiotic arrangements to optimize survival, changes in composition of plaque microflora such as an increase in acid-producing bacteria or gram-negative anaerobes can lead to diseases such as peri-implantitis.[40-42] Poor oral hygiene results in the development of the bacterial plaque around implants. Maturation of plaque (peri-implant pathogens evolve) contributes to inflammatory infiltrate formation in mucosal tissues, also known as perimucositis.[43] After implantation (in patient with periodontal disease), bacteria move from periodontal pockets of remaining teeth and other oral tissues to colonize the implant surfaces.[9,44,45] Colonization of the implant starts at surface irregularities (supra-structure/ implant collar) and spreads down the implant towards the base.[10] A deficient implant-abutment interface also provides a favorable site for bacterial accumulation, which contributes to the peri-implant inflammatory reactions.[38,46] Although the periopathogenic species in periodontitis and peri-implantitis are almost similar, there are differences observed with respect to relative numbers and species present.[40,47-50] Surviving and successful implants are observed to be populated with gram-positive coccoid cells, rods, gram-negative anaerobes, and a low ratio of anaerobe/aerobes.[10,51] Failing implants show greater proportions of periodontal pathogens, including gram-negative anaerobe rods, fusiform bacteria, motile rods, and spirochetes.[52]

DIAGNOSIS OF PERI-IMPLANTITIS

Accurate and timely diagnosis of peri-implantitis is a clinical challenge and relies on accurate assessment of the status of peri-implant tissues.[23,53-55] Aspects commonly assessed are pain, probing depth, mobility, bleeding index and radiographic evaluation of bone loss.[23,54-57] Signs of peri-implant disease include bleeding or suppuration after probing, swelling, peri-implant pockets greater than 4 mm, bone loss or saucer-shaped radiolucency around the implant, implant mobility and pain.[58] The diagnosis of peri-implant mucositis is generally associated with the presence of exudate release, swelling and/or bleeding on probing but without loss of bone. Peri-implantitis exhibits crestal bone loss,[6,11,47,54,57] and moderate to advanced peri-implantitis is diagnosed by radiographs showing saucerization of bone loss around implant, loss of gingival attachment, probing depths greater than 4 mm, mobility of implant, bleeding and sup-

puration.[59,60] A scale developed by James and Misch for classifies peri-implant disease on the basis of clinical symptoms and radiographic appearance ranging from optimum conditions to absolute failure.[23] The major diagnostic parameters considered in the classification include absence of pain, probing depth, rigid fixation, bleeding index, and radiographic evaluation of bone loss. The classification into groupings is based on the following findings:

Group 1: In this group the implant causes no pain or tenderness upon palpitation or function, stable probing depth, no horizontal or vertical mobility under loads of 500 g, less than 1 mm bone loss in the preceding 3 years, no exudates, and no radiolucency.

Group 2: This group is indicative of mucositis, or inflammation without any bone loss. There may be presence or history of exudate, swelling and/or bleeding on probing but without crestal bone loss.

Group 3: This includes moderate peri-implantitis, in which patients exhibit some degree of bone loss, and chronic inflammatory reaction around the implant, but the implant remains stable in the bone.

Group 4: This group represents clinical failure, where implants cause pain upon palpitation or function, greater than 1 mm horizontal mobility (also presence of vertical mobility a possibility), uncontrolled exudate, and radiolucency upon radiographic examination.

Group 5: Here in this group there is absolute failure, which occurs when the implant is surgically removed or exfoliated by the body.[23]

MANAGEMENT OF PERI-IMPLANTITIS

Various therapeutic strategies are employed in the management of peri-implant diseases (peri-implant-mucositis & peri-implantitis) in order to prevent failure of implant treatment.[19,61-63] Implant removal is indicated only when peri-implantitis has led to loss of osseointegration with more than 60% loss of bone to implant contact along with mobility of implant.[19,23] The following literature discusses some of the available treatment strategies for the management of peri-implant disease.

Non-Surgical Mechanical Debridement

The first line of treatment of peri-implantitis is scaling and root planning, sometimes referred to as non-surgical debridement.[63] A metal or plastic instrument is used to physically scrape the subgingival surface of the implant around the affected areas. This is accompanied with oral hygiene instructions to make the patient follow a strict oral hygiene regimen. The purpose of scaling and root planning is to reduce the inflammation by mechanically disrupting the biofilm on the implant surface.[64] In some cases, scaling is also combined with chlorhexidine irrigation and/or topical application.[39] Concerns have been raised regarding scratching and roughening the implant-abutment assembly with scalers which may contribute to potential increased plaque accumulation.[65,66] Therefore, it is desired that scaling and surface decontamination processes should leave the surface smooth or to avoid further plaque accumulation.

Local Antimicrobial Delivery in Periodontitis and Peri-Implantitis

It has become standard practice in treatment of periodontitis to locally administer antibiotics to patients with moderate to severe disease progression.[67] To maintain a sustained level of antibiotic at the site of infection, controlled release devices such as chips, gels, polymeric fibers, or microcapsules have been investigated and developed.[68-72] Various antiseptics and antibiotics have been incorporated into these devices, including doxycycline, tetracycline, minocycline, chlorhexidine, and metronidazole.[72-77] These devices are intended to keep the concentration of antimicrobial agent elevated in the gingival crevicular fluid for an extended period before degrading or being removed. One approach is to use local delivery devices developed for treating periodontitis, and implement them in the management of peri-implantitis. However, Mombelli, et al. in their clinical study showed that it is difficult to advance a local delivery device to the bottom of a deep peri-implant pocket.[78] This indicates that simply using periodontal therapies to treat peri-implantitis may not be an adequate and ideal solution.

Surgical Debridement and Bone Grafting

When scaling, implant surface debridement and local anti-microbial therapies fail to cease the progression of peri-implantitis, surgical debridement may be necessary. This mainly involves the resection of affected tissues (granulation tissue), debridement, implant surface decontamination, followed by bone grafting, with or without the use of barrier membranes.[39] Barrier membranes are used to promote the osseointegration of the titanium surface, and provide a barrier for epithelial migration into the defect space.[21] These membranes can be natural or synthetic and can be fabricated using resorbable or non-resorbable materials.[21] Studies comparing the use of resorbable (polylactic acid) and non-resorbable membranes, such as expanded Polytetrafluoroethylene (ePTFE) have shown similar clinical efficacy for the two approaches.[79] In addition, Guided Tissue Regeneration (GTR) membranes, intended for periodontal bone regeneration and antimicrobial activity have been investigated. These GTR membranes are usually degradable and have been modified with tetracycline,[80] doxycycline,[81] chlorhexidine[82] and metronidazole.[83] Furthermore, Bone grafting materials are utilized for bone regeneration in combination with GTR membranes. There are four classes of bone-grafting materials based upon the mode of action:

i. Autografts: Autogenous bone (usually harvested from mandibular ramus and chin) is an organic material and forms bone

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by osteogenesis, osteoinduction, and osteoconduction.

ii. Allografts: Graft tissues such as demineralized freeze-dried bone are osteo-inductive and osteoconductive and may be cortical and/or trabecular in nature. Allograft is derived from humans and is harvested from an individual other than the one receiving the graft.

iii. Xenografts: Graft tissues harvested from animals, for instance bovine and porcine (Bio Oss). These usually contain mineral portion (hydroxyapatite) of the bone.

iv. Alloplasts: Synthetic grafts such as hydroxyapatite, tricalcium phosphate, dicalcium phosphates, bioactive glasses etc. may be synthetic or natural, vary in size, and are mainly osteoconductive. These can be further divided based upon the porosity of the product.[14,20,84]

Clinical studies have shown improvement in defect fill and probing depth when patients were treated with bone graft regardless of the use of resorbable membranes.[85] These materials have different properties and therefore their indications may vary. The use of the three classes of materials in diverse combinations depends upon the size and topography of the bony defect. Small defects or defects with four walls of host bone can be repaired with alloplasts alone or allografts in combination with alloplasts.[86,87] The mechanisms that provide a rationale for bone grafting are as follows:

Osteoconduction is a function of a bone graft that provides a tridimensional scaffold for ingrowth of host capillaries and osteoprogenitor cells.[88] The bone graft material serves as a scaffold for new bone growth and the osteoblasts from the margin of defect utilize the bone graft material as a framework upon which new bone formation occurs. Bone regeneration in early phases at grafted sites is dominated by active bone resorption and formation throughout the graft material. The latter phase is characterized by osteoconduction and a process known as creeping substitution.[89]

Osteoinduction involves stimulation of osteoprogenitor cells to differentiate into osteoblasts and then begins formation of new bone. The osteoblast precursors differentiate into mature osteoblasts under the influence of osteoinductors and synthesize new bone during the first weeks. Growth factors involved in bone formation act on fibroblast and osteoblast proliferation, extracellular matrix deposition, mesenchymal cell differentiation and vascular proliferation.[90] A bone graft material that is osteoinductive, not only serves as a scaffold for currently existing osteoblasts but will also trigger formation of new osteoblasts, promoting faster integration of the graft.

Osteogenesis occurs when vital osteoblasts originating from bone graft material contributes to the growth of new bone along with bone formation.[91] A requirement for bone regeneration is the presence or recruitment of osteoblast precursors and growth factors at sites of augmentation.

Osteopromotion involves the enhancement of osteoinduction process without possessing of the osteoinductive properties.[92] For example, enamel matrix derivative enhances the osteoinductive effect of demineralized freeze-dried bone allograft.[93]

CASE REPORT

A 49 year old male presented in the outpatient department complaining of swelling, bleeding on brushing and pain on chewing around the implant site in the anterior maxilla from 6 months (Figure 1).

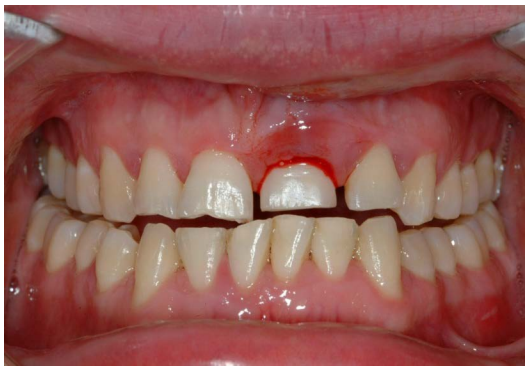

Figure 1: Pre-operative anterior view showing bleeding from the gingival margin of the affected implant.

Implant fixture was placed 4 years ago and was restored 3 months after surgical placement. Patient's oral hygiene was average with some plaque deposits on posterior teeth. The anterior tooth relation was in edge to edge incisor position. Clinical examination revealed gingival marginal bleeding, visible bone deficiency and mobile prosthetic component with displacement. The patient had extremely sensitive gingival tissues that bled instantly on probing and had a history of smoking. Patient was lacking in posterior stability due to moderate tooth wear on anterior teeth and over-eruption of mandibular anterior teeth. Excursive and protrusive guidance was on central and lateral incisors. Moderate to severe teeth attrition on anterior teeth was suggestive of heavy non-axial occlusal loading on the anterior teeth (Figure 2).

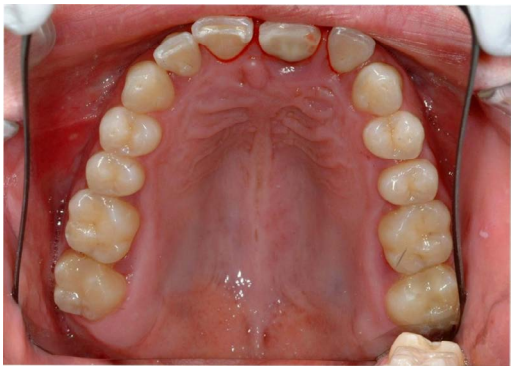

Figure 2: Pre-operative occlusal view.

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Implant was restored using a cement retained preformed standard abutment and cement retained crown. A periapical radiograph revealed significant bone defect around the marginal areas of the implant (Figure 3).

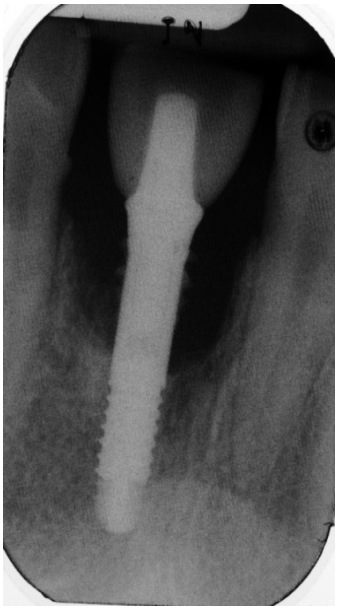

Figure 3: Pre-operative clinical radiograph showing bone resorption around the dental implant.

All of the above mentioned signs and symptoms, both clinical and radiographic were the classic indications of implant failure and peri-implantitis.

Treatment

After gathering all the relevant information, it was planned to undergo surgical debridement of the infected implant with peri-implantitis due to severe pocketing. Another goal of the surgical approach was to allow access for debridement and bone grafting. The abutment was removed and soft tissue flap was raised to expose the implant surface. Debridement (with removal of granulation tissue) of the implant surface and the defect was carried out and MinerOss Cortical & Cancellous (Biohorizons, USA) was placed in the defect followed by the placement of AlloDerm GBR (Biohorizons, USA). Soft tissue closure was carried out over a cover screw and post-operative instructions were given to the patient. Healing of the surgical wound was uneventful. At 8 weeks follow up, clinically soft tissue contours around the implant and the healing screw were well established. At 12 weeks follow up, radiographic appearance showed increased presence of bone around the implant fixture, at this stage impression was taken and the implant was loaded a week later with a standard implant abutment and cement retained crown. At 6 months follow up, peri-apical radiograph was taken to assess the implant and the surrounding tissues (Figure 4).

The implant fixture along with the prosthetic crown is seen in the desired position however, the bony defect had again started to occur. Clinically, signs of inflammation were visible again over the soft tissue surrounding the implanted tooth (Figure 5).

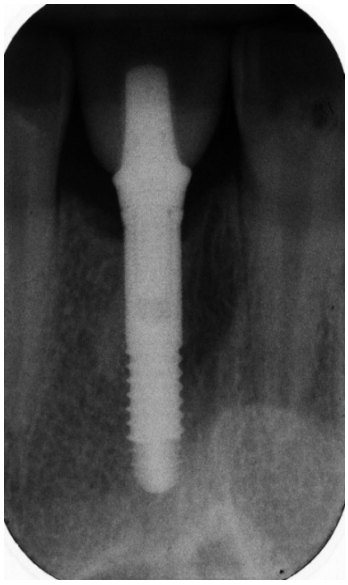

Figure 4: Post-operative clinical radiograph, taken 6 months after allograft placement showing higher bone levels in comparison with what was observed before treatment.

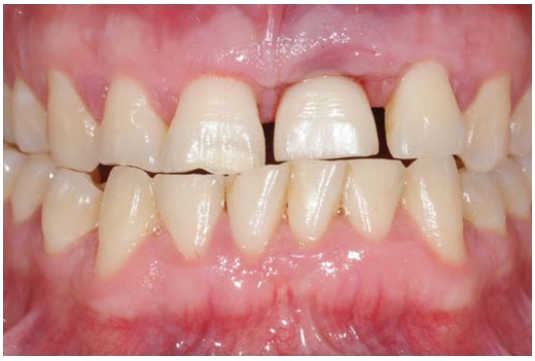

Figure 5: Post-operative anterior view, 6 months after treatment showing healthier soft tissue margins.

DISCUSSION

Endosseous dental implants have become quite significant in prosthodontics and restorative dentistry since the early 1970's.[94,95] Despite many advances in the techniques, materials and implant design, there is always the potential for clinical failure and hence it has been a significant concern both for the patient and the dentist. Dental implant failures occur occasionally, and clinicians may hesitate to perform a second implantation because of the uncertain prognosis.[96] This also underlines the necessity for a predictable treatment strategy for the maintenance and therapy of peri-implantitis. There is an acute lack of scientific guidelines for the management of peri-implantitis. As no agreed standard of care protocol for the treatment of peri-implantitis exists, it is reasonable to present a case report using principles previously reported to be efficacious in the treatment of periodontitis.[58]

Factors implicated in the etiology of the peri-implant infection include status of the tissue around implant, degree of roughness, external morphology and excessive mechanical load. Indicators of an implant failure include horizontal and vertical mobility, progressive bone loss, pain during percussion or function and infection.[97-99] While observing the clinical and the radiographic features of the patient, anterior attrition was evident with the history of heavy brushing, bruxism and smoking. According to the literature, the most common patient habits that adversely affect the dental implants are bruxism and smoking. Para functional habits (such as chewing ice and nibbling on hard objects) may cause premature implant failure.[32,100,101] In addition, the patient in this case report was also a smoker and several epidemiologic studies have shown the negative influence of smoking on periodontal status and an increased risk of developing periodontitis.[102-104] The relationship between smoking and implant failures has been evaluated in several retrospective and prospective clinical studies and there are reports that significantly greater percentage of implant failures occur in smokers than in non-smokers.[33,34,105] Therefore, it can be concluded that para functional activity in combination with smoking were major contributing factors in the development of peri-implantitis in the case presented in this report.

From the case reports available in literature, it can be concluded that treatment of peri-implantitis lesions with the combination of grafts and barrier membranes may lead to bone infill. However, the results of a comparative study by Khoury & Buchmann on treatment of peri-implantitis indicate that placement of barrier membranes in addition to bone grafting does not provide any adjunctive effects.[106] Unfortunately, not all peri-implantitis lesions are favorable to regeneration. For implants with thin facial and lingual walls, peri-implantitis typically does not produce a crater-form defect with four walls. In some of these cases, the defect will present as a complete loss of the surrounding bony walls leaving regeneration as an unpredictable treatment choice. Charalampakis, et al. evaluated the longevity and incidence of relapse of multiple different treatments on peri-implantitis lesions.[107] Over half of the cases evaluated relapsed and were not controlled. Patient habits and early disease development were associated with higher rates of relapse and surgical therapy with lower rates of relapse.[107] Long-term success of an implant depends on regular maintenance program. During maintenance phase, peri-implant tissue should be evaluated for inflammation. Patient education and relieving the possible risk factors need to be addressed to ensure longevity of the implant fixtures. All the treatment modalities in combination can result in a long-term good prognosis however; the treated cases must be watched closely as relapse is common.

CONCLUSIONS

In summary, development and management of peri-implantitis continues to be a challenge for dental practitioners and surgeons providing implant treatment. The microbial populations differ widely from patient to patient, and have the ability to change and develop over time which makes the treatment of peri-implantitis a difficult task. The use of bone allografts along with GTR in filling peri-implant defects is a pragmatic treatment option. However, there is lack of credible evidence suggesting that a specific treatment protocol or biomaterial is superior to others in treating peri-implant defects.

RECOMMENDATIONS

It is clear that several uncertainties still exist regarding management of peri-implantitis. Most of the studies reporting either open debridement with pocket reduction therapy or implant detoxification with the use of antibiotics for the treatment of peri-implantitis are case reports that are short-term and include a few cases only. However, long-term monitoring of consecutively treated cases in form of randomized controlled trials are further required. This will help in establishing predictable and stable improvements.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

CONSENT

Authors obtained written and informed consent from the patient for submission of this manuscript for this publication.

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Opinion

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Accelerating Orthodontic Treatment: A Continuous Challenge

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Every clinician will always aim for providing effective treatment, this can be accomplished by delivering the planned treatment goals over the shortest time possible, with minimal biological side effects and high levels of patient satisfaction. Unlike most of the dental specialities, orthodontic treatment tends to take relatively longer duration; this can range from as short as a several months to as long as several years (representative average 24 months).[1] This can be explained by the conservative nature of orthodontic treatment that mainly depends on the dento-alveolar response to gentle orthodontic forces.

It is not surprising that patient satisfaction with fixed orthodontic treatment outcome is generally high.[2] However, according to an interesting survey, the majority of orthodontic patients expressed that their duration of treatment was considered too long and that they wish if the duration of orthodontic treatment could be less than 12 months i.e. reduced to half.[3] Nowadays, there seem to be voices in the dental field claiming this is achievable. One must be wondering if this is true?

Let us first agree that this unresolved challenge is not new. Since Edward Angle introduced the edgewise fixed orthodontic in the early 1900's and hundreds of studies were conducted to identify factors influencing the duration of orthodontic treatment. Interestingly, most studies agreed that these factors can be categorised into: patient-related and treatment-related factors.[4]

As it was assumed that the patient-related factors are difficult to change, it was not a surprise to know that the main focus during the last few decades was to develop the mechanics around the fixed orthodontic appliance system. This included advanced technologies in the archwires (e.g. Nickel titanium), bracket design (e.g. self-ligation) and force application (e.g. elastomerics and NiTi coils). Although, these advances might have been useful in different aspects, none were found to significantly shorten the duration of orthodontic treatment.[3] This had confirmed to the clinician that there is no such thing called "fast brace systems".

It then became obvious that new approaches had to be considered. Approaches that are mainly focused on influencing patient response to orthodontic forces leading to accelerated tooth movement. This includes surgical and non-surgical interventions.

Adjunctive surgical interventions geared towards reducing orthodontic treatment times have been collectively described as 'surgically assisted orthodontics'. These methods are based on the principle of initiating inflammation after the bone is irritated surgically. This usually leads to increased rate of bone remodelling and in turn accelerates tooth movement. Although there is some evidence from several studies that these interventions can accelerate tooth movement, the results should be considered with caition.[5] This is mainly because the effect of the intervention was evaluated for few months (e.g. space closure stage) and not the whole duration of treatment; which may question the clinical significance of the intervention.[6] It is also worth mentioning that the effect of the surgical intervention was found to decay after few months.[7] This may suggest that repeated surgical intervention may be required through the

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treatment; which in turn may affect the popularity of the intervention among patients who already prefer non-invasive orthodontics.[3] Recently, less invasive surgical methods were proposed e.g. piezosurgery and microosteoperforation.

Non-surgical mechanical or physical stimulation of the dento-alveolar process has also been used in conjunction with conventional braces to increase the speed of alveolar bone remodelling. These non-surgical adjunctive interventions include Photobiostimulation (e.g. lasers), cyclic vibration and direct electric current. The popularity of these interventions has grown in the orthodontic field due to their non-invasive nature and the influence from marketing companies. Interestingly, some of these companies promote devices by claiming that they can reduce the orthodontic treatment time to half. However, currently there is no sound evidence to support these claims.[1] There are currently several ongoing research projects in this field and hopefully the future will tell us more about these interventions.

Unfortunately, accelerating orthodontic treatment is still an ongoing challenge. The evidence available regarding the adjunctive interventions is not supportive, but can be promising. Obviously, there is a rapid increase in the research around the non-invasive adjunctive interventions. It is important to remember that effective orthodontic treatment is not solely about the duration. I trust that patients expectations will continue to drive proper research for the continuous development of our speciality.

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