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Daffodils are Also His Sermon, 1991, 153 · 207 cm (Oil on canvas)
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The *SDJ* aims to advance the practice of dentistry and care of patients among members of the Association and dentists in the region through the dissemination of information and research findings in the field of dental science and technology. The scope of the journal covers all fields related to the present-day practice of dentistry, and includes Restorative Dentistry (Operative Dentistry, Dental Materials, Prosthodontics and Endodontics), Preventive Dentistry (Periodontics, Orthodontics, Paediatric Dentistry, Public Health and Health Services), Oral Medicine, Oral Surgery and Oral Pathology. Articles pertaining to dental education and the social, political and economic aspects of dental practice are also welcomed.

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The Editor, *Singapore Dental Journal*, Singapore Dental Association, 2 College Road, Singapore 169850.
Tel: (+65) 6220-2588; Fax: (+65) 6224-7967;
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Singapore Dental Journal

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SDJ—The New Look

In 1895, Lord Kelvin, the President of the Royal Society of England declared, "Heavier than air flying machines are impossible." Several years after that prediction, the Wright brothers took off with their home-built plane into the history books. In 1899, urban legend had it that the then Commissioner of the US Patent and Trademark Office, Charles H. Duell stated that "Everything that can be invented has been invented". He felt he should retire because patent applications would soon dry up and he would be left without a job. The US Patent and Trademark Office are still very busy today.

Dentistry like the other sciences has marched on. During the Renaissance, important works on dentistry began to be published. One of which was *A Treatise on Teeth* by the French surgeon Pierre Fauchard (1678–1761) where he described oral anatomy, disease and treatment. In the following years, many others build on his work. For example, Greene Vardiman Black (1831–1915) suggested that tooth infections were caused by bacteria. He also developed the first dental drill driven by a foot engine. These important discoveries and invention shaped the foundation of modern dentistry.

Man will know toothache for as long as they have teeth. The best defence against dental diseases is prevention and knowledge. The *Singapore Dental Journal* (*SDJ*) is part of the growing knowledge ecosystem. It will provide an international, peer-reviewed journal of original scientific research and clinical knowledge in dentistry.

This year, *SDJ* turns 35. Our past editors have set a high standard and laid a strong foundation. It will be a stepping stone to greater heights. Building on the past needs not means doing things the same way. *SDJ* will change to keep pace with its readership. We hope you will enjoy the journal gift-wrapped with a new look and fresh layout.

As the journal is better known as the *SDJ*, it will be named as so. To add colours to its front cover page, each issue of *SDJ* will feature an artist's work. After all, dentistry is a union of science and art. We begin this issue with the painting of eminent Singapore artist Tan Swee Hian.

SDJ is your journal, enjoy it.

<div align="right">

Dr. Tan Peng Hui
Editor-in-Chief

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Review Article

Dental Caries—Blame It On Genes!

YB Aswini MDS

Department of Preventive and Community Dentistry, Moradabad, U.P., India.

Abstract

Dental caries continues to be a common health problem, despite the claims that 50% of schoolchildren are caries-free. There are widespread variations in the prevalence of caries worldwide. Although significant progress has been made in our understanding of the aetiology, pathogenesis and prevention of dental caries, it still remains a scientific and clinical enigma worthy of the attention of the best scientists. The humane genome project and the sudden spur in the area of genetics and molecular sciences have made it prudent for even the dental profession to gear their knowledge regarding genetics affecting the oral health and disease. Hence, the aim is to review the effect of genetics on dental caries from various studies published. Various factors that cause dental caries have their own genetic predispositions. Laboratory studies have shown that chromosomes 1, 2, 7 and 8, and the H2 region on 17 are associated with caries susceptibility, and their locations have been found. But susceptibility to caries is controlled to a significant but minor degree by heredity and is also affected by environment. [*Singapore Dent J* 2009;30(1):1–7]

Key Words: chromosomes, dental caries, genetics, saliva, susceptibility

Dental caries is a widespread infectious disease and although dental caries in children and young adults has declined recently, it continuous to be a major health problem, particularly in young children. Although several methods, such as topical or systemic use of fluorides, fissure sealants and dietary constituents have been developed to prevent dental caries, they are insufficient to eradicate human dental caries. In the past, hereditary aspects were generally relegated to a relatively minor position, although the general public do seem to feel that "bad teeth run in families". The more common a genetic trait is, "the more difficult it will be to demonstrate its genetic character". The extremely high prevalence of caries makes the genetic analysis rather inefficient, even if dental caries were very strongly determined.[1–4]

Correspondence to:
Dr YB Aswini,
Department of Preventive and Community Dentistry,
Teerthankar Mahaveer Dental College and Research Center, Teerthankar Mahaveer University, Bagarpur, Moradabad 244001, Uttar Pradesh, India.
Phone: +91 9012715700,
E-mail: aswiniyb@hotmail.com

A century of research has described the process of initiation and progression of caries. And it has been said to be caused by four factors: the susceptible host, the pathogenic microorganisms, the cariogenic substrate and time. However, recent evidence shows that these factors may be genetically mediated, and these genetic differences in the individuals make certain environmental factors potentially more cariogenic for some people.[1–5] Hence, the main aim of this review was to find out the genetic influences on dental caries. A thorough search was made on the PubMed and other reliable sources. Among the 666 related articles cited in PubMed, the relevant articles were consulted. Two hundred and fifty-six articles were reviewed and rests were excluded as they were mainly inconclusive or were repetition of the previous studies. This subject has and also is undergoing a lot of research, as new things are adding up daily and to wrap it up in few words is mammoth and impossible job.

Researchers have pursued the pathways of genetic expression in four main approaches:[6–8]
1. Experimental/breeding studies in rats.
2. Caries experience in otherwise healthy subjects:
   a. Familial studies

b. Twin studies
c. Inbreeding and hybridity
d. Racial differences
e. Comparative studies between caries-resistant (free) and caries-susceptible (active) individuals
3. Caries experience in inherited disorders
    a. Reduced susceptibility
    b. Increased susceptibility
4. Association with genetic markers

Experimental/Breeding Studies in Rats

Two best-known experiments of this kind are the Hunt–Hoppert experiment at Michigan and another study at Harvard. Hunt and Hoppert from Michigan State College started planning in 1937 and later executed an original noteworthy and enormous experiment. They placed over 100 albino rats (*Mus norvegicus*) on a cariogenic diet that they devised. The surviving animals developed dental caries in 28–209 days. They developed genetically resistant and susceptible strains of rats. The strains were studied for many generations and included about 9800 rats altogether. The average time for inducing the first carious lesion in the lower molars of the susceptible rats decreased from 57 days in the second generation to 35 days in the 25th generation. The average time to induce carious lesions in the resistant strain increased from 116 days for the second generation to 505 days for the 17th generation.[9,10] Shaw and Griffiths used the Harvard strains with high and low susceptibility of dental caries development. In the experiment they transferred the newborn of the resistant strain to the mothers of the susceptible strain to be nursed, and vice versa.[11,12] The characteristic level of dental caries development for particular strain prevailed in spite of cross-nursing.[6] The experiments of Rosen, Hunt and Hoppert are in agreement with those of Shaw and Griffiths.[11,12] In another cross-breeding experiment, the authors concluded that both parents exert equal influences on the caries activity of the offspring, and that nursing mothers had little, if any, effect.[12] Keyes reported that dental caries can be induced in caries-inactive animals by exposing them to caries-active animals.[13] Rosen, on the basis of Keyes' observation, performed another experiment to further substantiate the genetic factors in dental caries development. The susceptible strain of rats showed an increase in caries activity after depression of the penicillin-sensitive flora with penicillin and reinoculation with faeces. The resistant strain did not show a significant increase in caries activity after depression of the penicillin-sensitive flora and reinoculation with the same kind of faeces.[14]

These experiments demonstrated that dental caries could be transmitted, provided that the animals are genetically predisposed to development of dental caries. If the animals are genetically resistant, however, even massive infection with faecal material containing cariogenic flora will not cause a significant increase in dental caries development.[9] Larson and Simms demonstrated that the dental caries is appreciably more active in Osborn–Mendel (O-M) strain than in National Institute of Health (NIH) black rats (B-R) when both were exposed to identical diets.[10,15] In another experiment, the same researchers used (O-M) female rats and mated them with both (O-M) and NIH black rats (B-R). The litters contained both (O-M) white and crossbreed grey to black offspring, and both groups developed significantly different dental caries status. The (O-M) and the NIH black crossbreeds had significantly lower caries activity than pure (O-M) strain. This phenomenon occurred even though the rats were exposed to identical environmental conditions.[14] Heredity rather than the nature of the oral microflora is the dominant factor in determining the different levels of caries activity.[10,14,15]

Caries Experience in Otherwise Healthy Subjects

Familial studies
Several studies have indicated that children show remarkable similarity in caries experience to their parents when the parent's susceptibility is the same. If, however, the caries experience of the parents is unlike, the children's susceptibility tends to be more like that of the mother than the father. Maternal effects can be an important determinant of family resemblance and can be mediated through either environmental, maternal genotype or genotype due to environmental interaction. It was found that the caries prevalence in the siblings of the caries susceptible group was

more than twice that of the siblings of the caries-resistant children. And changes in environment, such as exposure to fluoride-containing water were not sufficient to mask the genetic factor.[6–8,14]

The decayed, missed and filled surfaces (DMFS) of relatives of caries-susceptible group was three times more than that of the relatives of the caries-free group. This could be because of differences in oral hygiene and dietary habits, but it was found that they did not vary much in these two aspects. Thus, familial studies also show that it is difficult to escape the conclusion that good and bad teeth run in families.[6,9,15,16]

Twin studies

It was found that despite different environment, diet and professional care, the monozygotic twins demonstrated significant genetic variances.[16] Some have demonstrated greater similarity of caries experience within monozygous than within dizygous pairs, particularly for caries of lower anterior teeth and smooth surface approximal lesions. A significant difference between monozygotic and dizygotic twins in salivary flow, pH and amylase activity was found, which could be attributed to the difference in caries prevalence level.[5] Similarity in food favouritism was also demonstrated in monozygotic twins, but a study in the reared together population may have been biased by modelling on other family members or the co-twin.[17] Total immunoglobulin and antibody levels are more highly correlated in monozygotic twins than that in dizygotic. Even though the twins of the reared apart, experiments showed similar caries experience; it is unlikely that the mouths of the twins were colonized with microorganisms of identical serotype in particular *Streptococcus mutans*. Most monozygotic and dizygotic twins were separated shortly after birth and before eruption of the primary teeth, and therefore were unlikely to harbour similar strains of microorganisms. Thus, the findings in the twins reared apart provide additional evidence for a genetic component to dentate status and caries experience and is consistent with earlier studies of dental disease in twins reared together. The Minnesota Study of Twins Reared Apart (MSTRA) has produced the most convincing evidence for heritability of caries susceptibility.[18] This study has supported the association between dental caries and genetic background and extended it.

Inbreeding and hybridity

Inbreeding increases and hybridity decreases the incidence of recessive genetic disorders. No effect of inbreeding on DMFT could be demonstrated either among children of consanguineous marriages in Japan or the isolated inbreed populations in USA. No effect of hybridity was found among children of the inter crosses in Hawaii. It, therefore, seems unlikely that recessive genes make more than a minimal contribution to caries susceptibility.[6,18]

Racial differences

Racial differences in caries prevalence and severity appear to have a largely non-genetic basis being attributable to variation in the quantity of dietary sugar, ingested fluoride and use of fluoridated toothpaste. But, in multiracial populations of Hawaii even after correcting for ethnic differences in various sociological variables and oral hygiene practices, decayed, missed and filled (DMF) teeth and prevalence of caries-free individuals were found to vary between groups of different ethnic origin.[6,13,15,18]

Comparative studies between caries-resistant (free) and caries-susceptible (active) individuals

The aim is to define the defensive or protective phenomenon, to identify and to characterize the molecules involved, and then hopefully in time the molecular biologists would find the responsible genes. Human crown morphology is known to be under a degree of genetic control and a direct relationship was shown between crown morphology and caries.[19–22] In rats differences in occlusal fissure depth and angle were found between the caries susceptible and Hunt–Hoppert lines, which may have contributed to the difference in prevalence between the groups.[21] Genetic variation contributes about 40% to interindividual differences of tooth alignment in man. The alignment of hydroxyapatite crystals varied in individuals and is perhaps nutritionally or genetically determined. In the outer layer the alignment crystals was reported to be superior in caries-resistant than in caries-susceptible individuals.[19–21]

One of most important host factors influencing caries is *saliva*. Flow rate is reduced in monogenic disorders, such as aplasia and cystic fibrosis. Rampant caries is seen in parotid aplasia, but caries experience in cystic fibrosis is low because of increased salivary buffering.[23] Twin studies

have demonstrated greater similarity within monozygotic and dizygotic pairs and between siblings for both *flow rate* and *pH*. Inherited deficiencies of lactoferrin in human and lysozyme in rabbits have been demonstrated, and different forms of human salivary peroxidase are known. No significant difference between caries-resistant and caries-susceptible individuals was found.[24–28] The results of attempts to relate caries activity to different genetic forms of salivary proteins have also been inconsistent, although types *Db+*, *Pa+* and *Pr22*, respectively, of the double-banded, parotid-acidic and proline-rich systems have been associated with relatively high levels of caries activity.[29–31] Yu et al found an association between caries experience and proline-rich proteins (PRP) in saliva.[30] These PRPs are a complex of eight proteins coded by a region on chromosome 12p. Greater similarity in within MZ than within DZ twin pairs has been found for agglutinin activity. Differences between caries-resistant and caries-susceptible individuals have been found for the susceptibility of various salivary protein factors to degradation by *Streptococcus mutans* or *sanguis* and for the ability of proteins fractions to serve as growth substances for these organisms.[24–28,32,33] Recent studies of twins reared apart also support a genetic contribution to salivary total protein concentration as well as some specific proteins. It was also seen that, *T-lymphocytes* of caries-free individuals have significantly greater potential to proliferate on stimulation with *Streptococcus mutans* antigens than *caries-resistant* individuals.[34,35] Histatins display polymorphic expression, and *arginine peptide* most likely do as well; hence genetic factors could be significant in controlling their availability. Both parotid and submandibular saliva, plaque and pellicle of caries-resistant individuals contained fewer lipids. The process of salivary lipids binding to proteins involves specific *enzymes*, which are genetically engineered regulated, and it provides another potential genetic pathway in caries resistance.[27,28,36]

Low caries prevalence has been associated with high *serum IgG antibodies* to *Streptococcus mutans*. In rats, the susceptible line showed depressed immune response to cariogenic streptococci fecalis compared with the resistant line. In man, dietary experience is the major determinant of interindividual differences in taste preference and is not under significant genetic control.[34–36]

Caries Experience in Inherited Disorders[5–8,18,23,36,37]

Reduced susceptibility

1. **Hereditary fructose intolerance** is an autosomal-recessive disorder caused by deficiency of the enzyme fructose-1-phosphate aldolase, the blood glucose level may fall in response to fructose ingestion causing pallor, vomiting, sweating and even coma. Thus individuals develop a strong aversion to sweets and high proportions are caries free.
2. In **primary immunodeficiencies** relatively low caries experience is probably as a result of prolonged antibiotic therapy.
3. **Chronic renal failure** that occurs in a number of inherited disorders also inhibits caries due to high salivary pH.
4. In **congenital chloride diarrhoea**, an autosomal-recessive disorder, low caries experience has been reported. This may be because of high salivary pH as a result of metabolic alkalosis.
5. **Growth hormone deficiency** has been associated with resistance to caries probably because of the retarded eruption with consequently increased time for enamel maturation before exposure to oral environment.
6. **Turner's syndrome** patients also demonstrated low caries experience perhaps through interdental spacing.
7. An unusually low caries incidence particularly approximal lesions, has been observed in trisomy 21 (**Down syndrome**), possibly related to delayed eruption and interdental spacing.

Increased susceptibility

1. In **dystrophic epidermolysis bullosa**, extensive caries is found possibly as a result of poor oral hygiene associated with painful oral blistering following minor trauma.
2. In connective tissue disorders, namely **cutis–laxa**, **Rapp–Hodgkin ectodermal dysplasia** and in **focal dermal hypoplasia**, gross caries was found.
3. In **Rubinstein–Taybi syndrome**, marked caries was found perhaps as a result of poor dental care resulting from small mouth opening, malalignment of teeth and mental retardation.
4. **Klienfilter's syndrome** (47,XXY males) was also associated with increased susceptibility.

Association with Genetic Markers

Laboratory investigations suggested that caries susceptibility might be influenced by HLA type. However, a study of caries-free and caries-active military recruits was unable to confirm this in the clinical situation. Relative susceptibility to caries was found in persons with blood type A of ABO system and type N of MN system. In Papua New Guinea, caries susceptibility has been reported to be influenced by allelic variation in the gene controlling the enzyme red cell acid phosphatase, the allele P (a) being associated with relative caries resistance. The ability to taste phenylthiocarbamide (PTC) is controlled by a single autosomal gene.[38] Tasters have shown greater resistance to caries of the deciduous dentition than non-tasters.

Chromosomes 1, 2, 7 and 8,[39] and the H2 region on 17[38] are associated with caries susceptibility.

Candidate loci determining caries susceptibility are seen around the markers D1Mit21, from D2Mit237 to D2Mit101 and from D2Mit255 to D2Mit311 on chromosome 2, and the region distal to D7Mit31 of chromosome 7, and from D8Mit231 to D8Mit280 on chromosome 8. Data in Table 1 show the candidate genes around the regions of the suggestive and significant QTLs detected, according to mouse genome database 2000 and map view (build 29) of the National Centre for Biotechnology Information. There are a few genes on chromosome 1, about 1700 genes on chromosome 2, about 1300 genes on chromosome 7, and about 1100 genes on chromosome 8 in the regions in which QTLs were detected, including genes for which functions are known and unknown. Of these genes, focused on several associated with salivation and immune response.

HLA class II genes that are engaged in the wide range of immune response may play a role

Table 1. Candidate genes around suggestive and significant QTLs[42]

Detected regions	Chromosome position from centromere, cM	Gene name (symbol)
2	33.9	Calcium channel, voltage-dependent, beta 4 subunit (Cacnb4)
	38	Integrin alpha 6 Itga 6, Sjogren syndrome antigen B (Ssb)
	41	Beta-2-microglobin (B2M)
	69	Immune response-2 (Ir2)
	70	Granulocyte-macrophage antigen-3 (Gm-3)
7	71	Chloride channel current inducer (Clcni)
	50.0	Histocompatibility I (111)
	50.0	Parathyroid hormone (PTH)
	52.5	Calcitonin (Calc)
	54.0	Chloride channel, (Clcn3), potassium intermediate/ small conductance calcium-activated channel subfamily N, member I (Kenn 1)
8	32.2	Growth factor receptor bound protein 2-associated protein 1 (Gab l), calreticulin (Calr)
	37	Interleukin (IL-15), cahnegin (Clgn), T cell cytokine receptor (Tccr)
	38	Calcium channel, voltage-dependent, P/Q type, alpha lA subunit (Cacnala) matrix
	38	Metalloproteinase 2 (Mmp2)
	38.5	Matrix metalloproteinase 15 (Mmp15)
	42.9	Nuclear factor of activated T cells, cytoplasmic 3 (Nfat3), nuclear factor of activated T cells 5 (Nfat5)
	45.5	Neuronal calcium binding 2 (Necab2) – interleukin 17e (IL-17e)

in oral accumulation of mutans streptococci and lactobacilli. HLA class II alleles, HLA–DQA1*0102, DQB1*0604, DRB1*0802, DRB1*1302 and HLA-DQB10601 may be related to the salivary numbers of oral microorganisms, such as mutans streptococci and lactobacilli.[40]

Critical is the realization that genes and environment do not act independently of each other. Susceptibility to caries is controlled to a significant but minor degree by heredity. This genetic control is multifactorial in nature and such a polygenic background strongly implies considerable environmental modification.[3,4,6–8,41] With general decline in dental caries incidence and the increasingly uniform environment brought about by beneficial dietary and oral hygiene practices, host genetic variation in caries susceptibility will become increasingly important. And maybe we will be looking for genetic tests in clinics for dental caries in coming future followed by gene therapy, tissue engineering and nanotechnology. A lot of area is left untouched regarding this issue and needs further research to get a valid conclusion whether solely genetics have effect on dental caries or not.

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Scientific Article

A Quantitative Study of Dentine Removal During Apical Enlargement of Curved Canals Using Rotational Manipulation of Flex-R Files Versus Push–Pull Manipulation of K-Flex Files

[1]Ng Yuan-Ling MSc MRD RDS PhD, [2]Tseng Patrick SK MSc, [3]Lam M Jac Meng MSc,
[1]Gulabivala Kishor MSc FDS RCS PhD

[1]Unit of Endodontology, UCL Eastman Dental Institute, University College London, London, UK.
[2]Dental Centre, National University Hospital, Singapore.
[3]University of Malaysia, Malaysia.

Abstract

Dentine removal during apical enlargement of curved canals using Flex-R files in "balanced force" manipulation ($n = 10$) or K-Flex files in "push–pull" manipulation ($n = 10$) was compared. Twenty extracted mandibular molars with single-curved mesial canals with unique curvature and length were embedded in a Bramante matrix. The tooth-containing resin blocks were sectioned transversely at four predetermined levels. The re-assembled blocks enabled progressive enlargement of the canals to apical size 25 and then to size 40, using each technique in their respective samples. The digitized canal outlines were measured before and after instrumentation to sizes 25 and 40, respectively. Both techniques transported the inner canal wall at the height of curvature and the outer wall at the apical level. Transportation was more pronounced at size 40 than at size 25. At apical size 25, the Flex-R files produced a significantly ($p < 0.05$) better centred preparation than the K-Flex files (except at the mid-coronal level). In marked contrast, at the apical level at size 40, the Flex-R files achieved a significantly ($p = 0.001$) less well-centred preparation than the K-Flex files, contrary to expectation. [*Singapore Dent J* 2009;30(1):8–16]

Key Words: dentine removal, apical enlargement, rotational instrumentation, push-pull instrumentation

Introduction

The purpose of root canal preparation is to modify the size and shape of the main canal to facilitate debridement and obturation of the entire canal system.[1] The classically sought acquired shape is a regular conical taper, following the original canal curvature, minimal in diameter at the apical foramen and maximal at the pulp chamber orifice.[2] This goal can be achieved by a number of different manual or automated instruments manipulated either in rotational, in push–pull or in combination mode.[3–5] While all these modes of manipulation are in contemporary use, historically, they have each been dominantly favoured through different periods of their evolution. The transition from a predominantly rotational manipulation of stainless steel instruments (standardized technique) to the push–pull filing mode (serial flaring technique) was justified on grounds of better debridement.[6]

Correspondence to:
Dr Kishor Gulabivala,
Unit of Endodontology,
UCL Eastman Dental Institute,
University College London,
256 Gray's Inn Road, London WC1X 8LD, UK.
Phone: 44 (0) 20 7915 1033,
Fax: 44 (0) 20 7915 2371,
E-mail: K.Gulabivala@eastman.ucl.ac.uk

Unfortunately, the better debridement of serial filing was accompanied by unpredictable maintenance of canal curvatures,[5,7] particularly where the manipulation skills were deficient.[8] Attention, therefore, turned to the search for a technique that would overcome the need for skill and to reduce fatigue. There is a general impression that the introduction of manual and rotary nickel–titanium instruments would compensate for operators' skill manual and tactile deficiencies and allow effective canal enlargement while maintaining the canal curvature, with some justification.[9] However, nickel–titanium instruments lack the necessary physical properties for easy precurving at the tip for re-direction; therefore, they frequently prove to be unsuitable for negotiation past ledges, and through canal blockages caused by natural or artificial materials. In addition, there is also a high risk of instrument separation in canals with severe curvature. The need to acquire and maintain tactile skills to manipulate stainless steel instruments for such critical anatomical, iatrogenic situations, as well as to negotiate severely curved canals, remains a resident requirement for endodontists.

The essential problem with stainless steel instruments is the preferential removal of dentine in the plane of main canal curvature, resulting in its straightening.[5] The resulting procedural errors may be grouped under apical canal transportation, ledging, stripping and perforation.[5] The errors may be traced to:

1. the lack of control over the cutting surface of an instrument;
2. the tendency of rigid, elastic instruments to straighten;
3. the area of the instrument engaged in cutting at any given time.

Strategies have been developed to address these causes and their efficacy tested, mainly *in vitro*.[1]

The literature on push–pull and rotational manipulation of instruments is extensive, but direct comparisons are infrequent. There is a general consensus that flexible stainless steel instruments with non-cutting tips used in balanced force manipulation perform better in the curved canals than when used in the push–pull filing motion.[10–14] These studies did not compare the two techniques directly; uncontrolled variables included flaring devices and procedures, type of

files, precurving of instruments and apical preparation sizes. In an exception,[15] files with modified tips subjectively provided ideal instrumentation regardless of technique used, in epoxy blocks.

The aim of this study was to quantify and compare the pattern and extent of dentine removal in preselected transverse planes, when performing the apical canal enlargement using the K-Flex files in push–pull manipulation versus the Flex-R files in rotational manipulation.

Materials and Methods

Selection of Teeth

Extracted human mandibular first and second molars with mature root apices and the absence of unusual morphology were collected and stored in 4% formal-saline.[16] The surfaces of the teeth were cleaned by soaking in 2% sodium hypochlorite (Sainsbury's bleach, J. Sainsbury Plc, London, UK) for 10 min to dissolve the remaining soft tissue; ultrasonic scaler (Dentsply/Cavitron, Dentsply Ltd, Weybridge, Surrey, UK) was used to remove calculus. After conventional access cavity preparation, size 10 K-Flex files (Kerr, Peterborough, UK) were placed in the mesial canals beyond the apical foramina and the teeth radiographed (Kodak Ektaspeed film, Kodak UK Ltd, Hemel Hempstead, Hertfordshire, UK) using standardized distance, exposure and processing. The root canal curvatures were determined as described by Schneider.[17] A total of 20 teeth, each with a canal in the mesial root with the following characteristics, were selected:

1. patent apical foramen;
2. minimal canal length of 13 mm;
3. canal not joining the adjacent one;
4. single root curvature in the mesio-distal plane, of the order of 25–35°.

Ten teeth were randomly assigned to each of two experimental groups (Flex-R and K-Flex).

Preparation of Teeth

The selected teeth were de-coronated at the enamel–cementum junction and the coronal surfaces were ground flat to enable the teeth to stand crown down. The flat coronal surface of the tooth was cemented with the mesial and distal
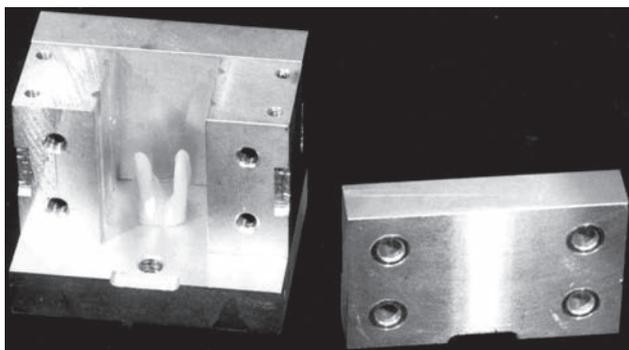
Figure 1. *Photograph of the aluminium matrix with tooth in position prior to complete assembly and embedding in resin.*

roots oriented in a standard direction to the base-plate of an aluminium matrix (Figure 1).[18]

The root canal orifices and foramina were sealed with soft wax (Dentina Ribbon Wax®, Browning Dental Supply Co. Ltd, Hull, UK) before encasing the tooth in clear auto-polymerizing methyl methacrylate (Forestacryl®, Forestacryl Dental Ltd, Milton Keynes, UK) by pouring into the matrix and pressure-curing ($2\,kp/cm^2$) at a temperature of 25–30°C (Dentarum Polyclav®, Hawley Russell, Potters Bar, UK) for 15 min. The resin blocks with the teeth were sectioned (horizontally using a 0.1-mm, 64-grit diamond continuous blade; Exact Sectioning Saw, Mederex Crossledge Farm, Wooley, Bath, UK) transversely at four predetermined levels:

1. mid-coronal (mid-way between the coronal surface and the height of root canal curvature);
2. height of curvature (mid-point of maximum curvature, measured radiographically and confirmed by visual inspection of its external root surface);
3. mid-apical (mid-way between height of curvature and 1.0 mm from the apical foramen);
4. apical (1.0 mm from the apical foramen).

Longitudinal grooves on the inner aspects of the vertical matrix plates facing the mesial and distal root surfaces enabled accurate re-assembly of the sectioned resin blocks in the matrix. Removal of the buccal and the lingual plates of the matrix permitted radiography of the tooth (Figure 1). A size 10 K-Flex file was re-introduced into the re-assembled canal to file away any minor ledges formed by re-apposition of the sections. The preoperative canal outline was taken to be that after this procedure. The working length was established at 1 mm from the apical foramen and confirmed radiographically.

Instrumentation Procedure

All canals were instrumented by one operator (P.S.K.T.). The canals were progressively enlarged to size 25 and then to size 40 at the working length using Flex-R files (Union Broach, Health-Chem Co, New York, USA) in a balanced force technique or K-Flex files (Claudius Ash & Sons Ltd, Barnet, Hertfordshire, UK) in a circumferential filing technique. The final canal shaping consisting of flaring was omitted as the purpose was to evaluate only the effect of apical enlargement. A copious amount of sodium hypochlorite (1% v/v) irrigation was used.

Flex-R group (n = 10)
Canals were sequentially prepared to a master apical file (MAF) of size 25 and then up to size 40 using the "balanced force" technique described by Roane.[19] The Flex-R files (size 15–40) were introduced without precurving, into the canal, sequentially using a 90–180° clockwise rotational motion, followed by anti-clockwise motion (360°). An adequate apical pressure was applied during each anti-clockwise rotation to affect cutting. This sequence of movements was repeated for each size until the working length was reached. Recapitulation was carried out regularly with the previous smaller file. Instrumentation with each size was terminated when the file was able to reach the working length without pressure and was just loose.

K-Flex group (n = 10)
The canals were prepared using circumferential filing motion without precurving the K-Flex files, enlarging the canal first up to apical size 25 and then up to size 40. Termination of filing was gauged by the ability of another set of precurved files to reach the working length without pressure; and it was considered to be just loose.

Evaluation of Dentine Removal

The cross-sectional outline of the canal at the four preselected levels was digitized before and

Table 1. Mean cross-sectional areas (mm^2) of dentine removed (post-instrumented area less pre-instrumented area) for each experimental group

Group		Mid-coronal		Height of curve		Mid-apical		Apical	
		#25	#40	#25	#40	#25	#40	#25	#40
K-Flex ($n=10$)	Mean	0.073	0.475	0.047	0.294	0.054	0.190	0.041	0.134
Flex-R ($n=10$)	Mean	0.056	0.112	0.041	0.184	0.042	0.136	0.021	0.198
Difference between groups (K-Flex–Flex-R)		0.017	0.363	0.006	0.110	0.012	0.053	0.020	−0.064
p value		0.02	<0.001*	0.02	0.002*	0.02	0.02	0.06	0.1

*Significant at the 1% level.

after instrumentation to sizes 25 and 40, respectively, using a Quantimet® 520 Computer-Assisted Image Analyser (Cambridge Instruments Ltd, Cambridge, UK). This consisted of a high-resolution (0.018 mm per pixel) video camera with a 55-mm Nikon™ macrolens, an image analysis processor and an IBM PC/XT computer equipped with Hi-Pad Digitizer (0.018 mm per pixel) (IBE Ltd, Basingstoke, UK). Accurate superimposition of the three images of each section was ensured by four relocating points scribed on the resin surface. The pre- and post-instrumentation images were compared and the following outcomes measured:

1. total cross-sectional area of dentine removed (post-instrumented canal area less pre-instrumented canal area);
2. maximum post-instrumentation width (Y mm) of the root canal in mesio-distal plane;
3. the maximum extent of dentine removal in the mesial (outer wall: X_1 mm) and distal (inner wall: X_2 mm) directions of the selected plane.

The centring ratio ($X_1 - (X_2/Y)$) was calculated according to Calhoun and Montgomery.[12]

Statistical Analysis of Data

The non-parametric Mann–Whitney test was used to compare the outcome measures (1) and (3) and the centring ratios of the two test groups. The differences between the extent of dentine removal in the mesial and distal directions were analysed using non-parametric Wilcoxon signed rank test. The Wilcoxon signed rank test was also used to compare the centring ratios of each preparation after enlargement with apical sizes

25 and 40, within the same test group. One per cent significant level was used.

Results

Area of dentine removed (Table 1)

After instrumentation to apical size 25, there was a small and insignificant difference in dentine removal between the K-Flex and the Flex-R groups. Instrumentation to size 40 resulted in significantly more dentine removal in the K-Flex compared with the Flex-R group at the mid-coronal ($p < 0.001$) and height of curvature ($p = 0.002$) levels. However, there was no significant difference in dentine removal at the mid-apical and apical level between the two groups.

Maximum extent of dentine removal from the outer and inner canal curvature (Table 2)

Instrumentation to apical size 25 resulted in significantly more dentine removal from the inner curvature of canals when using the push–pull filing technique than when using the rotational technique, at the mid-coronal ($p = 0.002$) and height of curvature ($p < 0.001$) levels. On the outer curvature, the K-Flex files removed significantly more dentine than the Flex-R files at the mid-coronal and apical levels, whereas at height of curvature and mid-apical levels, the difference in the extent of dentine removal between the two groups was small and insignificant at the 1% level.

Continuation of canal enlargement to apical size 40, resulted in significantly ($p < 0.001$) more dentine removal at the mid-coronal level from the outer canal curvature in the K-Flex compared to the Flex-R group. In contrast, there was greater

Table 2. Mean values ($n=10$) for maximum extent of dentine removal on outer (X_1 mm) and inner (X_2 mm) walls by section level, file type and size

Group		Outer wall (X_1 mm)		Inner wall (X_2 mm)	
		#25	#40	#25	#40
Mid-coronal					
K-Flex	Mean	0.093	0.408	0.090	0.158
Flex-R	Mean	0.024	0.030	0.025	0.057
	Difference	0.069	0.376	0.065	0.010
	p value	0.002*	<0.001*	0.002*	0.01
Height of curvature					
K-Flex	Mean	0.002	0.007	0.092	0.299
Flex-R	Mean	0.020	0.024	0.029	0.263
	Difference	−0.018	−0.017	0.063	0.036
	p value	0.4	0.8	<0.001*	0.3
Mid-apical					
K-Flex	Mean	0.067	0.139	0.047	0.103
Flex-R	Mean	0.034	0.166	0.022	0.076
	Difference	0.033	−0.028	0.025	0.027
	p value	0.03	0.7	0.4	0.3
Apical					
K-Flex	Mean	0.086	0.281	0.014	0.016
Flex-R	Mean	0.020	0.477	0.013	0.004
	Difference	0.066	−0.196	0.002	0.013
	p value	<0.001*	0.001*	0.5	0.07

*Significant at the 1% level.

dentine removal from the outer curve in the Flex-R group at the apical level ($p=0.001$).

Direction of transportation (Table 3)
Apical enlargement to size 25 and further instrumentation to size 40 in the K-Flex group resulted in significant canal transportation towards the outer canal curvature at the apical level ($p=0.005$). In contrast, the transportation towards the inner curvature was significant ($p=0.005$) at the height of curvature.

Instrumentation to size 25 in the Flex-R group resulted in no significant transportation towards the inner or the outer canal curvature at all levels. Upon further instrumentation to size 40, transportation towards the inner curvature at the height of curvature ($p=0.007$) and transportation towards the outer curvature at the apical level ($p=0.005$) were significant at the 1% level.

Comparison of centring ratios (Table 4)
The canals were significantly ($p<0.01$) less well centred when they were prepared to size 40 apically compared with size 25 for both techniques at all levels, except the mid-apical level for the K-Flex ($p=0.07$) and the Flex-R ($p=0.02$) groups, and the mid-coronal level for the Flex-R group ($p=0.1$).

When the canals were instrumented to apical size 25, the preparations in the Flex-R group remained centred significantly better than those in the K-Flex group, at all levels except the mid-coronal level ($p=0.9$). When the canals were enlarged to apical size 40, the canal preparations in the K-Flex group were significantly less well centred at the mid-coronal level ($p=0.001$) and conversely significantly ($p=0.001$) better centred at the apical level compared with the Flex-R group.

Table 3. Mean differences ($n=10$) in dentine removal between outer (X_1 mm) and inner (X_2 mm) walls by section level, file type and size. (Negative difference means greater removal from inner curvature and conversely, positive difference means greater removal from the outer curve)

Section level	K-Flex group		Flex-R group	
	X_1-X_2 mm	p value	X_1-X_2 mm	p value
Apical size 25				
Mid-coronal	0.003	0.7	−0.001	1.000
Height of curvature	−0.089	0.005*	−0.008	0.6
Mid-apical	0.020	0.2	0.012	0.1
Apical	0.071	0.005*	0.007	0.2
Apical size 40				
Mid-coronal	0.250	0.01	−0.026	0.3
Height of curvature	−0.292	0.005*	−0.239	0.007*
Mid-apical	0.036	0.4	0.091	0.05
Apical	0.264	0.005*	0.473	0.005*

*Significant at the 1% level.

Discussion

The experimental model was developed in our laboratory[20] and was similar in principal to the Bramante model with the same benefits of comparing the cross-sectional pre- and post-instrumentation shapes of the canal. Disadvantages include the minor ledges resulting from re-apposition of sectioned blocks and the fact that serial transverse sectioning of the block results in sections of the curved canal that are at an angle to the long axis. Inclusion of the mid-apical section, not used in other studies,[12,21] revealed previously undisclosed trends (discussed later). The root canals were selected by strict criteria to minimize the variation presented by canal anatomy. The direct digital imaging technique used for capturing the canal outlines incurred minimal error of the order of less than 1.8×10^{-2} mm, the size of a pixel or picture element.

The apical size of preparations were selected according to previous recommendations,[4,22–24] and the final coronal flaring was excluded as the main procedural errors were considered to arise during the earlier apical enlargement. Precurved instruments were not used for preparation so as to reduce the variables under study.

The operator performing the instrumentation was originally trained to use the push–pull filing technique, while the balanced force technique has been a recent practice. Acquisition of skills to manipulate instruments is not a mere matter of the following prescribed protocol, but of acquiring tactile sense by a process yet undefined.[8] A practitioner versed in certain tactile skills may find it more difficult to learn new and different tactile skills, a fact subjectively observed in the rates at which the undergraduates and the postgraduates learn a new technique.

Inclusion of the mid-apical section revealed two distinct patterns of dentine removal within the K-Flex group. In four specimens, there was substantially more dentine removal on the outer curve at the mid-coronal level compared with the rest ($n=6$). Concomitantly, there was greater transportation of the canal towards the inner curve at the mid-apical level in the former compared with the latter subgroup. The pattern of dentine removal was similar between these two subgroups at the height of curvature and apical levels. Visually this observation presents the picture of the instruments "pivoting" at the level of the height of curvature, causing excessive dentine removal in opposite directions, coronal and apical to this point.

Direct comparison of the present results with the previous studies is impossible because of variations in study design, size of apical preparation and adoption of flaring procedures. In this study, the Flex-R files used in balanced force

Table 4. Median of centring ratios (the smaller the centring ratio, the better centred the preparation)

Group/apical size	Mid-coronal		Comparison between #25 and #40	Height of curvature		Comparison between #25 and #40	Mid-apical		Comparison between #25 and #40	Apical		Comparison between #25 and #40
	#25	#40	p value	#25	#40	p value	#25	#40	p value	#25	#40	p value
K-Flex (n = 10)	0.026	0.233	0.005*	0.136	0.337	0.005*	0.101	0.168	0.07	0.125	0.372	0.005*
Flex-R (n = 10)	0.028	0.080	0.1	0.043	0.301	0.005*	0.023	0.274	0.02	0.018	0.575	0.005*
Comparison between K-Flex and Flex-R												
p value	0.9	0.001*		<0.001*	0.2		0.004*	0.2		0.002*	0.001*	

*Significant at the 1% level.

motion removed significantly less dentine than the K-Flex files used in push–pull filing motion at all levels except the apical section, regardless of apical preparation size, in contrast to another study.[13]

It was interesting that the mid-coronal sections showed a slight transportation of canals towards the inner canal curvature in the Flex-R group, but towards the outer curvature in the K-Flex group. The transportation was significantly greater when the canals were prepared to the apical size 40 in the K-Flex group. In contrast, Calhoun and Montgomery reported that both filing and rotational techniques transported the canal towards the inner wall at the coronal level, despite the fact that the K-Flex files were used in a circumferential filing motion.[12] These differences are clearly dictated by the manner of file manipulation by the operator.

Further apically, the pattern of transportation was similar in the two groups. Both techniques tended to straighten the canal regardless of apical preparation size, consistent with the findings of others.[12,13,25] In this study, upon instrumentation to apical size of 40, the Flex-R group showed significantly more transportation than the K-Flex group at the apical level. In contrast, other studies[12,13,15,25] reported no significant difference in apical transportation between the balanced force and filing techniques when the canals were prepared to apical sizes 35–45. The results of this study dispute the assertions that the balanced force technique unequivocally allows larger apical preparations without the risk of transportation.[4] The present findings are, however, consistent with the observations of Southard et al and Royal and Donnelly.[26,27] The literature offers two possible explanations for the findings:

1) excessive apical pressure during the initial clockwise rotation may preferentially cut the dentine on the outer wall at the tip of the file[28] or

2) insufficient apical forces may not allow the larger and more rigid files to conform to the natural curvature of the canal during counterclockwise rotation.[29]

The common theme to both explanations is the control of apically directed torque, a function of file manipulation that should ideally respond to an educated tactile sense. The prescription of clockwise and anti-clockwise "controlled" file

movements is therefore insufficient by itself to allow adequate control over dentine removal; the component of tactile sense during apical feed is also crucial and is disregarded by the protocol. The findings mirror the conclusion on file manipulation during the push–pull technique.[8] Rotational manipulation of instruments requires tactile skill that cannot be supplanted by prescriptive protocol. Contrary to popular belief, preparation to an apical size 40 using the balanced force technique was not as effective as the filing technique for maintaining apical curvature, in the hands of an operator preadapted to filing. The findings bear out the observation that effectiveness of learning a new technique is individual-dependent. Preconditioning by past experience may bias the rate and extent of acquisition of new tactile skills either positively or negatively. It is a factor to be considered during the coaching of these neuromotor skills to dentists.

Conclusions

Within the limitations of this study, it may be concluded that:

1. Both push–pull filing and balanced force instrumentation techniques may straighten curved canals by transportation of the inner curvature at the height of curvature and the outer curvature at the apical level. In this study, the trends were more pronounced when the canals were prepared to apical size 40 compared to size 25.

2. When the canals were prepared to apical size 25, the Flex-R files used in a balanced force motion produced significantly better centred preparations than the K-Flex files used in a push–pull filing motion.

3. In contrast, upon further instrumentation to apical size 40, the ability of the balanced force technique to achieve well-centred preparations was significantly worse at the apical level compared with the push–pull filing technique.

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Adverse Tobacco Habits and Their Relation with Prevalence of Oro-mucosal Lesions among Green Marble Mine Labourers, Udaipur District, India

Prabu Duraiswamy MDS, Rushabh J Dagli BDS, Santhosh Kumar BDS, Chandrakant Dhanni BDS, Suhas Kulkarni MDS

Department of Preventive and Community Dentistry, Darshan Dental College and Hospital, Udaipur, Rajasthan, India.

Abstract

Objective: To determine the prevalence of oro-mucosal lesions among Keshariyaji green marble mine labourers and to find its relation with adverse tobacco habits.

Materials and Methods: The study area was divided into four geographical zones, and the participants were selected by stratified cluster sampling technique. A total of 513 subjects were included in the final study, and they were divided among the four age cohorts: 18–25, 26–34, 35–44 and ≥45 years. They were interviewed for tobacco habits, and clinical examination of oral mucosa was done by one of the three examiners with the aid of an artificial light source. The agreement for diagnoses of oro-mucosal lesions was determined (field teams versus expert) using kappa statistics, and it was 0.91, 2 days prior to the examination. Statistical analysis was done using ANOVA, chi-square and multiple logistic regression analysis.

Results: An overall higher prevalence of oro-mucosal lesion was found among mineworkers (36.7%), and much higher value was found among those who were having tobacco habits (40.6%). Non-users have shown less prevalence of leucoplakia 28%, compared with that of users (regular users: 34.7%; occasional users: 40%; and ex-users: 50%). Oral submucous fibrosis was found among regular users only (4.3%). The prevalence was also increased by smoking (form) of tobacco and consumption of alcohol. Among all age cohorts higher prevalence of leucoplakia was found among the age group of 35–44 years (40%).

Conclusion: Oro-mucosal lesions among mineworkers were aggravated due to deleterious habits of tobacco consumption with increasing age and bidi smoking habits. At present, tobacco cessation programmes with patient education are needed. [*Singapore Dent J* 2009;30(1):17–25]

Key Words: marble mineworkers, oral-mucosal lesions, tobacco, Udaipur

Introduction

A large number of labourers are working in the stone crushing industry in India.[1] In whole of

Correspondence to:
Dr Prabu Duraiswamy,
Department of Preventive and Community Dentistry,
Darshan Dental College and Hospital,
Udaipur 313 001, Rajasthan, India.
Phone: +91 9928714502,
Fax: +91 294 2452273,
E-mail: drdp76@yahoo.com

Udaipur district of Rajasthan, Keshariyaji is the only place where green marble mines are found. Exposure to respirable crystalline silica and a number of other particulate matter metrics in occupational settings are unavoidable, as part of health hazards of these workers; in addition, the physically tedious work drives the labourers to consume alcohol and tobacco, which devour a significant portion of their meagre income. These mineworkers are highly associated with tobacco habits that cause deterioration of their oral health; therefore, this population was selected, and no previous oral health study was conducted among them. Due to heavy physical

workload, there were no female workers in this occupation.

The whole community is plagued by malnutrition, ill health and physical impediments from accidents. A mineworker, on an average, finishes his life at the age of 49.3 years, which is 10 years earlier than those outside the mines (Mines Department, the Government of Rajasthan). Moreover, the dust swirling around in quarries comprises mineral powder that causes a number of lung diseases, such as silicosis, tuberculosis (TB), silico-tuberculosis and asthma.

Most of the previous studies about cigarette, cigar or pipe smoking and their significance for oral diseases were carried out in the Western part of the world, and marked changes in oral disease pattern were observed during the past decades.[2,3] More recently, meta-analysis of oral health report of these studies has been carried out, and the results have indicated that over the past 40 years there is contrasting disease trend, depending on the country population status and the socioeconomic conditions.[4] During the past 10–15 years, in several Western industrialized countries, there is prevalence and severity of dental disease.[5] Africa over the past 40 years revealed contrasting disease trend depending on the country population groups and socioeconomic conditions.[6]

Tobacco smoking causes pronounced structural reorganization of the oral and gingival mucosa due to its atrophic changes. Significant differences in the local humoral immunity (immunoglobulin G (IgG) dysimmunoglobulinaemia) and cytokine spectrum in non-smoking and tobacco-smoking patients were detected. Subcompensated level of immune resistance in tobacco smokers prompts them referring to a group at high risk of chronic pathological processes of the oral cavity.[7] Tobacco used in both smoke and smokeless forms induces oro-mucosal changes in which intra-oral mucosal pigmentation is one of the clinical manifestations.[8]

The mouth is the only body site that permits viewing with the naked eye the ravages of tobacco in both smoke and smokeless (chewing) forms. For a given patient, it is often possible to observe the mouth during a clinical examination of normal tissue, premalignant lesions (e.g., leucoplakia) and malignant tumours.[9,10]

Various previous studies suggest that both cigarette smoking and alcohol drinking co-exist in a significant proportion of male adolescent, which will have a major implication on oral lesions.[11] Majority of the people living in the rural areas have limited access to essential oral health care because of the environmental conditions and economic barricade.[12,13] Exposure to chemical, physical and biological agents in the workplace can result in adverse effects on workers ranging from simple discomfort and irritation to debilitating occupational diseases, such as lung fibrosis, neuropathy, deafness, organ damage and cancers of various sites.[14]

With respect to the prevalence of dental disorders and oro-mucosal lesions very few studies have been carried out among Indian population, especially with the rural subjects[15] and with specific occupation. Moreover, there are only few studies available with respect to oro-mucosal lesion in the Asian population, among which one reported the prevalence of oro-mucosal lesion at Thai and Malaysian dental schools[16] and the other studied only precancerous lesions related to drinking, smoking and chewing habits.[17]

Study Population and Method

Rajasthan is located in the north-western part of India, in which Udaipur district located at the southernmost part.

The study conducted by the Department of Preventive and Community Dentistry, Darshan Dental College, known as the "First Rajasthan Mines Oral Health Study", was intended as an inventory of the present state of oral health, oral-health-related behaviour and use of dental treatment services of the mineworker population of Rajasthan, India. Recording of oro-mucosal lesions and co-relating it with stress was one part of this study, and this study solely focuses on these results.

Udaipur district consists of many green marble mines from which marble is exported all over the world. Keshariyaji is the place where all the green marble mines are located, and all the mines are situated within a radius of 25 km. It is divided into four geographical zones: Masoroi ki Obri (southeast), Rushabhdev (north-east), Khandiovri (southwest) and Kagdar Bhatiya (north-west). Except the possibility of treatment in major hospitals at Udaipur, there is no public support for the population regarding dental health at Keshariyaji.

The minimum sample size required for this survey was based on the lowest prevalence of a significant target lesion that was observed in the previous surveys; World Health Organization (WHO) sampling procedure (stratified cluster sampling procedure) was used to collect the representative population for a cross-sectional study, as there were no previous studies available for oro-mucosal lesion among these population.[18,19] With the help of marble mine owners association a special leave was granted for the workers. The total population of mineworkers provided by the mine owners' association from all the four geographical zones was 4832 mineworkers from 29 mines. From each zone, two mines were randomly selected, in which all the present workers (521 workers) were examined. Eight workers (1.53%) dropped out for quality-neutral reasons and remaining 513 males were selected and included in this study.

The oral cavities of these 513 selected workers were examined using additional artificial light. To visualize the oral mucosa two mouth mirrors were used, and palpation was done when there is a need to detect the consistency of lesion. The "WHO Oral Health Performa, 1997" was used to record the demographic information. No biopsies were done.

All clinical data were recorded by three trained project teams each consisting of a dentist, a dental nurse and a recording assistant. Recording of data of the oro-mucosal lesions was based on the publications of WHO,[20,21] and comparable authorized publications.[22–24] Definitions of diseases of oral mucosa were adapted from publications by Axe'll (1976) and Zain (1995).[25,26] In addition, the International Classification of Diseases for Dentistry (ICD-DA)[27] was also used for the same. Participants' tobacco habits, including the form of tobacco, duration and frequency of usage, as well as alcohol habits, were recorded by asking simple questions to habitual patient in their local language which was easy for the mineworkers to understand and to communicate with the dentist.

All three teams had undergone a full day theoretical and practical training by the senior dental calibrator prior to the commencement of the project. To verify whether the data obtained were of sufficient quality to compare, reliability studies were performed by the participating project calibrator during the field work. The agreement for diagnoses of oro-mucosal lesions was determined (field teams versus expert) using kappa statistics at 0.91, 2 days prior to the examination.

Bivariate frequency distribution was used to analyse the data. ANOVA test and eta-square test (performed with ANOVA for precise interpretation of variance) were performed for statistical evaluation of mean values, and the models were developed to determine whether variables were significantly associated with the outcomes of oro-mucosal lesions. For the models, the explanatory variables included were the following:

i) Age
ii) Tobacco use
   a. Form of tobacco used
   b. Duration of tobacco use
   c. Frequency of tobacco use
iii) Alcohol use

Statistically, $p < 0.05$ was accepted as significant and $p < 0.01$ was set to be highly significant. Pearson's chi-square test was used for comparison for location of lesion (leucoplakia). The statistical analysis was performed using the SPSS (10.0) software package.

Ethics

The study was conducted in the month of February 2007, for which prior permission was obtained from the concerned authorities to prevent any inconvenience to marble mine owners and workers. Ethical clearance was obtained prior to the survey from "Ethical Committee of Research" of Darshan Dental College and Health Authorities. The informed consent of local authority and each patient were taken prior to recording oro-mucosal lesions.

Results

The results show a higher prevalence of leucoplakia (40%) in the age group of 35–44 years and lower (20%) in the age group of ≥45 years (Table 1). OSF was reported only in the age group of ≥45 years with 20% of cases, and papilloma occurs only in the age group of 18–24 years (5.2%). The data in Table 2 show the topographical prevalence of leucoplakia in tongue and buccal mucosa in various age groups; the results show that buccal mucosa was the more common site

Table 1. Prevalence of individual oro-mucosal lesions in various age group*

Oro-mucosal lesions	18–24 (%) ($n=171$)	25–34 (%) ($n=162$)	35–44 (%) ($n=135$)	$\geq$45 (%) ($n=45$)	Total ($n=513$)
No lesions	99 (57.9)	117 (72.2)	81 (60)	27 (60)	324 (63.1)
Leucoplakia	63 (36.8)	45 (27.7)	54 (40)	9 (20)	171 (33.3)
Papilloma	9 (5.2)				9 (1.7)
OSF				9 (20)	9 (1.7)

*ANOVA; OSF = oral submucous fibrosis; $f = 12.42$; $p = 0.001$ (HS); $\eta^2 = 0.006$.

Table 2. Topographical prevalence of leucoplakia in various age groups

Age group (yr)	Buccal mucosa	Tongue
15–24	45 (26.3%)	18 (10.52%)
25–34	36 (22.2%)	9 (5.55%)
35–44	27 (20%)	36 (26.6%)
$\geq$45	9 (20%)	0

$\chi^2 = 24.02$; df = 3; $p = 0.001$ (HS).

than tongue for leucoplakia in all the age groups except 35–44 years ($p<0.01$).

Data in Table 3 show that among the 513 participants, 288 were tobacco users (56.2%), whereas 225 were non-users (43.8%). An overall higher prevalence of oro-mucosal lesion was found among mineworkers (36.7%) and more in those who were having tobacco habits (40.6%). Non-users have shown less prevalence of leucoplakia 28%, compared with users group (regular 34.7%, occasional 40% and ex-users 50%). OSF was present in 4.3% of regular users.

Data in Table 4 show that among the tobacco users ($n=288$), most of them were chewers ($n=207$; 71.8%), and leucoplakia was found in 34.8% of them, whereas the smokers ($n=63$; 21.8%) showed a higher prevalence of leucoplakia (57.1%). Participants using tobacco in more than one form were only 3.1%. OSF was found only among smokers, and all were in the age group of $\geq$45 years ($p<0.01$).

Data in Table 5 show a higher prevalence (66.7%) of leucoplakia among tobacco users in lesser duration (<5 years; 66.7%) than that of longer (6–10 and 11–20 years; 28.5% and 30%, respectively) ($p<0.01$). Data in Table 6 show a

higher number of leucoplakia lesion (66.7%) that was associated with the consumption of tobacco 1–2 times a day rather than more times (>10 times a day; 33.3%).

The reason behind this relationship of duration (Table 5) and frequency (Table 6) found in this study can be due to their mode of consumption of tobacco; that is, most of the participants who were taking tobacco with lesser duration and 1–2 times a day were tobacco smokers rather than chewers. This shows that the form of tobacco is a more important factor for aggravating the oro-mucosal lesions rather than the duration and frequency.

Among the 513 participants, 135 were alcohol users (26.3%), whereas 378 were non-users (73.7%). Non-users have shown less prevalence of leucoplakia (33.3%) compared with users (regular users 33.3%, occasional users 40%). OSF was present among 20% regular users of alcohol, which shows that a synergetic effect of alcohol with smoking form of tobacco would be related to OSF (Table 7).

Multiple logistic regression analysis model of the prevalence of oro-mucosal lesions was increased with age and form of tobacco (Table 8).

Discussion

Epidemiological studies can be subdivided into incidence and prevalence studies. The occurrence of disease with a short duration is better elucidated in the incidence studies, whereas that of chronic nature, as in the case of majority of oral lesions, is illustrated more clearly in the prevalence study.

The relatively small population investigated and the highly selective occupational character

Table 3. Oral lesion and tobacco users*

Oral lesion	Non-users	Regular users	Occasional users	Ex-users	Total
No lesions	153 (68%)	126 (60.8%)	27 (60%)	18 (50%)	324 (63.1%)
Leucoplakia	63 (28%)	72 (34.7%)	18 (40%)	18 (50%)	171 (33.3%)
Papilloma	9 (4%)				9 (1.7%)
OSF		9 (4.3%)			9 (1.7%)
Total	225	207	45	36	513

*ANOVA; OSF = oral submucous fibrosis; $f = 1.069$; $p = 0.36$ (NS); $\eta^2 = 0.006$.

Table 4. Oral lesion and form of tobacco*

Oral lesion	Non-users	Smoking	Chewing	Snuff	More than one form	Total
No lesions	153 (68%)	18 (28.5%)	135 (65.2%)	9	9	324 (63.1%)
Leucoplakia	63 (28%)	36 (57.1%)	72 (34.8%)			171 (33.3%)
Papilloma	9 (4%)					9 (1.7%)
OSF		9 (14.3%)				9 (1.7%)
Total	225	63	207	9	9	513

*ANOVA; OSF = oral submucous fibrosis; $f = 18.39$; $p = 0.001$ (HS); $\eta^2 = 0.12$.

Table 5. Oral lesion and duration of tobacco use*

Oral lesion	Non-users	<5 yr	6–10 yr	11–20 yr	21–30 yr	Total
No lesions	153 (68)	27 (33.3)	81 (64.2)	63 (70)		324 (63.1)
Leucoplakia	63 (28)	36 (66.7)	36 (28.5)	27 (30)	9	171 (33.3)
Papilloma	9 (4)					9 (1.7)
OSF			9 (7.1)			9 (1.7)
Total	225	63	126	90	9	513

*ANOVA; OSF = oral submucous fibrosis; $f = 3.64$; $p = 0.006$ (HS); $\eta^2 = 0.03$.

Table 6. Oral lesion and frequency of tobacco use*

Oral lesion	Non-users	1–2 times a day	3–5 times a day	6–10 times a day	>10 times a day	Total
No lesions	153 (68)	27 (33.3)	72 (66.7)	54 (60)	18 (66.7)	324 (63.1)
Leucoplakia	63 (28)	36 (66.7)	36 (33.3)	27 (30)	9 (33.3)	171 (33.3)
Papilloma	9 (4)					9 (1.7)
OSF				9 (10)		9 (1.7)
Total	225	63	108	90	27	513

*ANOVA; OSF = oral submucous fibrosis; $f = 4.05$; $p = 0.003$ (HS); $\eta^2 = 0.03$.

of this population in comparison with other epidemiological studies give precarious results, and thus the results should be interpreted with great caution.

The intention of this study was to provide systematic information on the use of tobacco and oro-mucosal lesion of marble mineworkers, in Keshariyaji, and the results would aid in the

Table 7. Oral lesion and alcohol users*

Oral lesion	Non-drinkers	Regular drinkers	Occasional drinkers	Ex-drinkers	Total
No lesions	243 (64.3)	54 (66.7)	18 (40)	9	324 (63.1)
Leucoplakia	126 (33.3)	27 (33.3)	18 (40)		171 (33.3)
Papilloma	9 (2.4)				9 (1.7)
OSF			9 (20)		9 (1.7)
Total	378	81	45	9	513

*ANOVA; OSF = oral submucous fibrosis; $f = 3.57$; $p = 0.03$ (HS); $\eta^2 = 0.01$.

Table 8. Multiple logistic regression analysis of dependent variable (oro-mucosal lesion) showing likelihood ratio tests

Effect	2 log likelihood of reduced model	Chi-square	df	Significance
Age	230.348	74.514	90	0.000*
Tobacco use	165.537	9.703	6	0.138
Tobacco form	202.497	46.663	6	0.000*
Tobacco duration	157.273	1.439	6	0.963
Tobacco frequency	168.010	12.176	6	0.058
Alcohol use	166.214	10.381	6	0.110

*$p < 0.01$ (HS).

planning and evaluation of oral health promotion programmes. Moreover, comparable prevalence data were not recorded previously. Hence, the study would also gather data for comparing the prevalence of lesions among other populations in India and in other countries.

Rationale for this comparison was that the former Indian population, especially workers, differed considerably—occupationally, politically, socially and particular in health care utilization—compared with the population of other countries. It was therefore expected that differences in occupation, lifestyle and health care access will have an effect on oral health including the prevalence of oro-mucosal lesions.

For this study, stratified cluster sampling procedure was used to achieve representative sample. It incorporates the sufficient examination sites of all four geographical locations at Keshariyaji. The study was designed as an epidemiological survey of the cross-sectional study type based on representative samples of the population taken from the age cohorts, 18–25, 26–34, 35–44 and ≥45 years. There were no mineworkers below 18 years of age. Lesser number of representative populations of older age ≥45 years was found in this study, as the work is more associated with physical strenuous nature.

Moreover, tobacco habits were recorded using the questionnaire in local language of mineworkers, which was easy for them to communicate, and thereby more reliable data for tobacco habits were collected. Alcohol habits was also recorded for all mineworkers as a potential confounding factor with tobacco in this study. There might be an under-presentation of tobacco and alcohol habits by mineworkers. This should be kept in mind while interpreting results.

Lesions were more predominantly observed on buccal mucosa, whereas lesions were merely observed on tongue. The reason for buccal mucosa being more prone for mucosal lesions may be due to the adverse tobacco habits in the smoking form of bidi (an unfiltered handmade form of tobacco, made by rolling a dried rectangular piece of temburin leaf with 0.15–0.25 g of tobacco). Most of the subjects reported tend to be chronic chewers of tobacco.

Bhonsle et al[15] recorded that the habit of chewing tobacco along with lime in the canine–premolar region of the mandibular groove is widespread (28%) in the rural population of Maharashtra, India where leucoplakia was prevalent (0.67%) and tobacco quid lesion was found (2.9%).[15] The prevalence of oro-mucosal lesions of 9.2% was found from the epidemiological survey in Malaysia by Zain et al,[26] which is comparatively much less than that in the present study. Various previous studies suggest that both cigarette smoking and alcohol drinking together in a significant proportion of male adolescent will have major implications on oral lesions.[11] However, Corbet et al[28] found no difference in the prevalence or the number of oro-mucosal lesions between those defined as users of tobacco and alcohol and those defined as non-users.[28] A result similar to the present study was found by Baric[29] that two of three leucoplakia lesions (66.7%) were encountered among smokers in the elderly subjects.[29]

The prevalence of white patch in the previous study of Danish glassblowers was reported 40% by Schiodt et al.[30] Chronic smoking is associated with a lower systemic status of several subgroups of vitamin B, reduced oral folate and changes in folate form distribution in the mouth.[31] Buccal cell concentrations of the carotenoids, retinoids and tocopherols were generally lower in heavy smokers compared with light smokers,[32] which increases the susceptibility of oral mucosal disease prevalence and might be the reason for having higher incidence of oral submucous fibrosis in smokers group. Risk associated with tobacco use can also be modified by other exposures such as diet, alcohol consumption and genetics.[33–35] The result of this study was to be used in the planning and evaluation of oral health promotion activity, which being a part of general health care action plan at district level.

Conclusion

In a revitalization of the existing oral health service that is mainly treatment based, it should be oriented more towards preventive health care. The authors conclude that in such population as in this study tobacco control, research and interventions will need to be conducted in concert with nutrition research and interventions to improve the overall health status of the population. Tools for combating the epidemic include public policies intended to discourage tobacco use through taxation. In the restrictions on promotion, media campaigns are designed to prevent smoking initiation and encourage cessation, individual counselling techniques and medications designed to promote and maintain smoking cessation, modification of tobacco products to reduce harmfulness and substitution of less harmful for more harmful products (e.g., pharmaceutical nicotine for smoked tobacco).

Acknowledgements

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Annexure 1

Comprehensive Tobacco and Alcohol Questionnaire

Tobacco

| T | F | D | FR |

T—Tobacco use
    0: non-user
    1: regular user
    2: occasional user
    3: ex-user
F—Form
    1: smoking
    2: chewing
    3: snuff
    4: more than one form used
D—Duration
    1: less than 5 years
    2: 6 to 10 years
    3: 11 to 20 years
    4: 21 to 30 years
    5: more than 30 years
FR—Frequency
    1: 1 to 2 times a day
    2: 3 to 5 times a day
    3: 6 to 10 times a day
    4: more than 10 times a day

Alcohol

| A |

A—Alcohol use
    0: non-user
    1: regular user
    2: occasional user
    3: ex-user

A Case of Mucormycosis with Alveolar Bone Sequestration and Review of Literature

[1]Benay Tokman, [1]Burcu Sengüven, [2]Süleyman Bozkaya, [2]İnci Karaca

[1]*Department of Oral Pathology, Faculty of Dentistry, Gazi University, Ankara, Turkey.*
[2]*Department of Oral and Maxillofacial Surgery, Faculty of Dentistry Gazi University, Ankara, Turkey.*

Abstract

Mucormycosis is an opportunistic, potentially lethal fungal infection that occurs primarily in patients who are immunocompromised. This is a disease of sinuses, brain or lungs caused by fungi of class Phycomycetes. An oral ulceration, a necrotic lesion and even perforation of the hard palate can indicate this disease. Urgent biopsy of involved necrotic-appearing tissue is necessary. Because the initial cultures of diseased tissue may be negative, for the definite diagnosis a tissue specimen must be analysed. The authors report a case study of mucormycosis of maxilla presenting in a patient with diabetes mellitus and rheumatoid arthritis and review the literature on mucormycosis. [*Singapore Dent J* 2009;30(1):26–30]

Key Words: alveolar bone sequestration, mucormycosis, opportunistic infection, oral fungal infections

Introduction

Mucormycosis, also known as zygomycosis and phycomycosis, is an acute fungal disease with a high mortality rate, which predominantly occurs in patients with impaired host defenses or diabetes mellitus.[1,2] This opportunistic infection caused by fungi of class Phycomycetes was first described by Paltouf in 1885.[1,3] *Rhizopus* species are the most common causative organisms; with *Rhizomucor*, *Cunninghamella*, *Saksenaea* and *Apophysomyces* species all being less frequent. It is common in patients with diabetes mellitus, especially in those with diabetic ketoacidosis.

Seventy-five percent of patients with rhino cerebral form of mucormycosis have ketoacidosis.[4,5] Chronic steroid use, metabolic acidosis, organ transplantation, leukaemia/lymphoma, treatment with deferoxamine, and AIDS are the other conditions most commonly associated with mucormycosis.[5,6] Mucormycosis in immunocompetent hosts is rare, and it is often related to trauma.[7] This infection is caused by common fungi frequently found in soil and in decaying vegetation. These fungi do not usually cause disease in healthy people with intact immune systems, but patients with a number of conditions can be predisposed to the development of invasive fungal disease. These conditions include diabetes mellitus, renal failure, malignancies, intravenous drug abuse, malnutrition states, as well as immunosuppression and corticosteroid therapy.[8]

Mucormycosis can have multiple clinical presentations including rhino cerebral mucormycosis, cutaneous, pulmonary, gastrointestinal and disseminated forms.[4,8] The most commonly reported form of the disease is rhino cerebral

Correspondence to:
Dr Benay Tokman,
Department of Oral Pathology, Faculty of Dentistry,
Gazi University, Ankara, Turkey.
Phone: +90 312 2034385,
Fax: +90 312 2239226,
E-mail: benay@gazi.edu.tr

Figure 1. *Exposed alveolar bone on the left molar region.*

Figure 2. *Destruction of the medial wall and base of the left maxillary sinus, and mucosal thickening and opacification.*

mucormycosis, which is characterized by progressive fungal invasion of the hard palate, paranasal sinuses, orbit and brain.[2]

Case Report

A 56-year-old white male was referred to the Department of Oral and Maxillofacial Surgery of the Gazi University, Faculty of Dentistry, in January 2005, complaining about numbness in the left zygomatic region, nasal discharge and stuffiness, and pus drainage and pain in the maxillary left molar region for the past 1½ months.

There was no evidence of cervical lymphadenopathy during extra oral examination. Vital signs were within normal limits. During intraoral examination, a 2.5-cm ulcer was observed on the left molar region that demonstrated an erythematous border and a central 2.0-cm area of exposed bone (Figure 1). In addition, except for the left central incisor, all other teeth in the quadrant had been extracted. The maxillary right quadrant exhibited evidence of moderate to severe periodontitis.

The medical history of the patient was significant for insulin-dependent diabetes and rheumatoid arthritis approximately for the past 10 years.

Panoramic radiography revealed a bone sequestration in the alveolar process of the left maxillary posterior region, which was approximately 2.0 cm in diameter. In the maxillary right quadrant, bone destruction that was con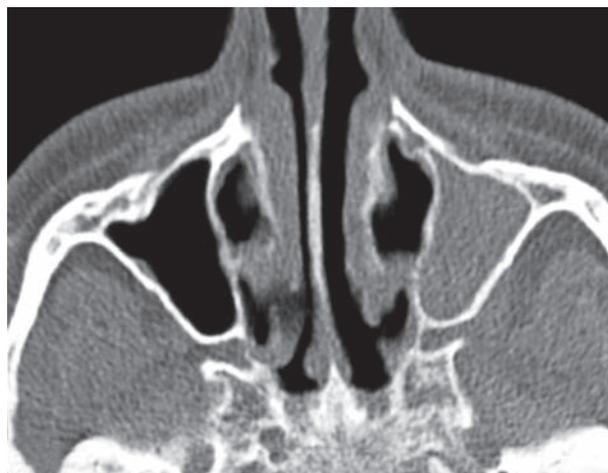sistent with chronic periodontitis, was observed. Plain sinus films revealed vague opacification of the left maxillary sinus, but there was no evidence of bone destruction. However, computed tomography showed destruction of the medial wall and base of the left maxillary sinus, which might have been caused by inflammatory soft tissues that were localized in the left maxillary posterior region with mucosal thickening and opacification of the left maxillary sinus (Figure 2).

At the time of admission, the patient's initial haematological analysis was within the normal limits, except for a high glucose level (246 mg/dL). After the consultation with endocrinology service, the sequestrated maxillary alveolar bone was removed under local anaesthesia and submitted for histopathological examination to the Oral Pathology Department of Gazi University, Faculty of Dentistry.

On the macroscopical examination, greyish white-brown coloured necrotic bone, measuring $2.5 \times 1.7 \times 1.5$ cm in size, was observed. Short-term decalcification procedure was performed with 10% formic acid. Paraffin-embedded specimen stained with haematoxylin–eosin showed extensively necrotic bone trabeculae within numerous large hyphae and together with micro-abscesses. Masses of microorganisms were also found within the bone marrow spaces. The microorganism has broad, large, non-septated hyphae with branching at 90°. Although the hyphae could be easily identified with haematoxylin–eosin stain, they were highlighted with Gomori methenamine silver nitrate and Periodic acid–Schiff stain (Figures 3 and 4).
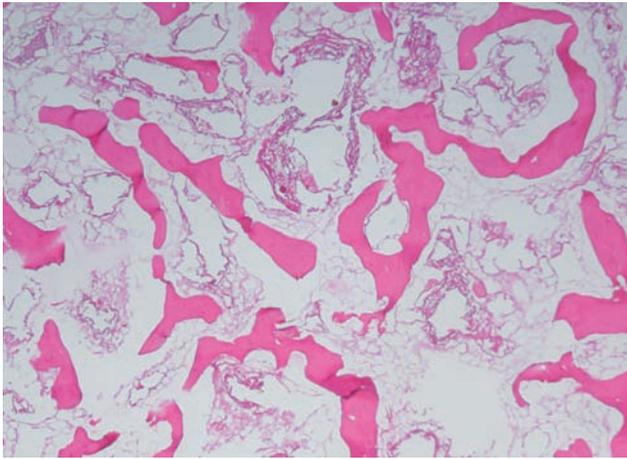
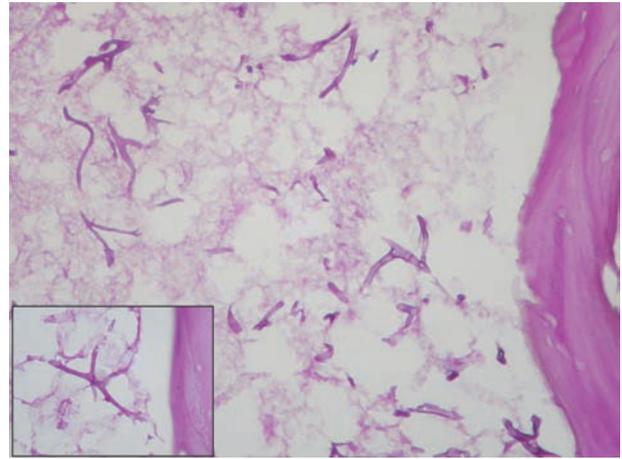**Figure 3.** *Necrotic bone trabecula and bone marrow spaces (×40, haematoxylin–eosin).*

Figure 4. *The microorganisms with broad, large, non-septated hyphae with branching at 90° (×200, and inset ×400, Periodic acid–Schiff stain).*

After the diagnosis of mucormycosis, the patient was immediately referred to the Oto-Rhino-Laryngology Clinic of the Gazi University Faculty of Medicine for the screening of other systems, especially central nerve system and lungs, and also for the commencement of the antifungal therapy. However, insufficient cooperation of the patient led to incomplete management and follow-up.

Discussion

Mucormycosis is an acute opportunistic infection caused by a fungus that belongs to class Phycomycetes.[2] The infection usually results from inhalation of spores through nose or mouth. In immunocompetent individuals these spores will be phagocytosed by macrophages; therefore, they do not develop the disease. Mucormycosis is described almost exclusively in patients with compromised immune systems or metabolic abnormalities. In the USA, 50–75% of patients with mucormycosis have poorly controlled diabetes mellitus and ketoacidosis. Rhino cerebral mucormycosis is especially aggressive and fatal in diabetic patients with ketoacidosis, possibly additional alterations on host immune mechanisms and increased availability of micronutrients. The high-iron, glucose-rich, acidic environment facilitates fungal growth.[5,6,9] The spores attach to the nasal or oral mucosa where massive spore formation occurs. Once the spores begin to grow,

fungal hyphae invade blood vessels, producing tissue infarction. Spread occurs when it invades the nasal cavity and maxillary sinuses. Areas of ischaemic infarction and necrosis are seen in the infected tissue. The fungi invade the blood vessel lumina and cause thrombosis through inflammatory occlusion.[2,4]

Despite advances in diagnosis and treatment, mucormycosis still has high mortality rates. Death rates range from 25% to 80% depending on the site involved as well as the underlying immune problems and also how early and aggressively it is managed. Rhino cerebral disease causes significant morbidity in patients who survive because the treatment requires extensive facial surgery. Loss of neurological functions because of nerve involvement, thrombosis and blindness, if the optic nerve is involved, are the most important complications of disease.[1,2,4,8,10]

Sex of the patient is not likely to impact the occurrence of mucormycosis because the underlying conditions are the major predisposing factors. Reviews of cases show an equal sex distribution. Mucormycosis is found in patients of a wide age range. No racial factors play a predisposing role in mucormycosis.[2,4]

Early diagnosis and treatment are the key factors in controlling mucormycosis. A delay of even 12 hour in diagnosis may be fatal. The diagnosis requires a high degree of clinical suspicion. Mucormycosis is distinguished by its fulminate course with evidence of tissue necrosis. Fever is usually present. Common symptoms include the

orbital and facial pain, sinusitis, headache, fever, visual changes, nasal discharge or stuffiness. Black discolouration (necrotic tissue) on the nasal turbinate, septum and palate is almost diagnostic.[1,2,4,8] Definitive diagnosis of mucormycosis requires analysis of a biopsy specimen of the tissue. The predominant histological findings are ischaemia or haemorrhagic necrosis. Histologically, mucormycosis is characterized by extensive tissue necrosis and the presence of numerous large fungal hyphae that are non-septated and have a ribbon-like appearance, with budding and branching that tends to approximate 90° angles.[2,6,11] Histopathological examination is essential for early diagnosis, as initial cultures of diseased tissue may be negative.[1,2,4]

Effective management centres on early diagnosis and treatment to reduce morbidity and ensure the best chance of survival. For management of mucormycosis, aggressive surgical debridement of all necrotic tissue is necessary, sometimes requiring multiple debridements. Patients also should receive adjuvant antifungal therapy (Amphotericin B, liposomal (Amphocin and Fungizone)) for long term. Hyperbaric oxygen has been suggested as a potential treatment, but its exact role remains unclear.[9,11]

As a necrotic lesion, a palatal ulcer or an extraction can be a part of rhino cerebral mucormycosis, many conditions, such as other fungal infections or carcinomas, should be added to differential diagnosis.

Aspergillosis is another opportunistic mycosis, which has characteristically black ulcerated areas of necrosis in the oral lesions. But it has smaller, septated and more acutely branching hyphae.[11]

If mucormycosis involves the maxillary sinus, it may present as a mass in the maxilla that resembles antral carcinoma. A malignant salivary gland tumour arising from the minor glands of palate should also be included in the differential diagnosis. Squamous cell carcinoma may be thought to be a likely condition because it represents about 90% of cancer affecting the oral mucosa. But all these lesions have dramatically different histological findings, such as islands of anaplastic tumour cells, cellular atypia and atypical mitosis, compared with mucormycosis.[3,11]

Wegener's granulomatosis is an uncommon necrotizing vasculitis characterized by the triad of acute necrotizing granulomas of the upper respiratory tract (oral cavity, sinuses and throat), widespread vasculitis and necrotizing glomerulonephritis. Morphologically the upper respiratory tract lesions range from inflammatory sinusitis to ulcerative lesions of the nose, palate or pharynx, rimmed by necrotizing granulomas and accompanying vasculitis. The granulomas reveal a geographical pattern of necrosis rimmed by lymphocytes, plasma cells, macrophages and variable numbers of giant cells.[3]

Extranodal NK/T-cell lymphoma, nasal type (lethal midline granuloma), is an unusual malignant condition which characteristically involves the midline oro-nasal region. Tuberculosis, syphilis and osteoradionecrosis should also be included in the differential diagnosis of rhino cerebral mucormycosis with oral manifestations.[2,4,11]

Mucormycosis is a rare infection; hence, it is difficult to calculate its incidence accurately. But fungal infections caused by rare fungi have increased in recent years. The incidence of this disease also appears to be increasing. This may be due to the increase in the number of immunocompromised patients. Under these circumstances, mucormycosis has gained importance, as it is not uncommon any longer.[5,10]

Large maxillary osteonecrosis is rare in patients with rhino cerebral mucormycosis. Necrosis of the palate may be the result of thrombosis of the sphenopalatine or internal maxillary artery. The maxillary sequestration gives evidence to the necrotizing potential of mucormycosis.[12] Without appropriate treatment, the disease spreads into the orbit and brain and results in death. Because the initial signs and symptoms of disease often involve the oral, facial and cranial structures, it is critical that dentists and oral pathologists should be aware of mucormycosis infections, especially in the diabetic or immunocompromised patients.

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Case Report

A Regenerative Technique Utilizing Autogenous Bone and a Connective Tissue Barrier in the Treatment of Intrabony Periodontal Defects

Edwin Chia Kian Heng BDS (Singapore), MSD Oral Biology (Boston, USA), Cert Periodontology (Boston, USA)

Head Dental Branch, HQ Medical Corps, Singapore.
Visiting Adjunct Lecturer, Postgraduate Periodontology, Harvard School of Dental Medicine, Boston, MA, USA.

Abstract

Intrabony defects develop as a result of periodontal disease. They are characterized by vertical or angular bone loss, and such defects are common in patients experiencing moderate to severe periodontitis. Periodontal surgery to resolve intrabony defects can be either resective or regenerative. Osseous resection is usually not preferred to be performed in moderate to severe defects because this may compromise the existing support of both involved and adjacent teeth. Hence, regenerative techniques to restore the lost periodontal apparatus would improve the long-term prognosis of such teeth. The aim of this study is to showcase a technique of treating deep intrabony defects through regenerative techniques utilizing solely the autogenous materials (intraoral bone autografts and connective tissue) from the patient. Two patient case scenarios are also illustrated to give the reader a more profound understanding of this technique. [*Singapore Dent J* 2009;30(1):31–8]

Key Words: intrabony periodontal defects, bone grafts, connective tissue, periodontal repair, periodontal regeneration

Introduction

The American Academy of Periodontology has defined an intrabony defect as "a periodontal defect within the bone surrounded by one, two or three bony walls or a combination thereof". An intrabony defect, developed as a result of periodontal disease, constitutes vertical or angular bone loss, and the pocket base is apical to the osseous crest. The presence of such a lesion relates to associated possible loss of tooth support and site specificity of further periodontal destruction.[1,2]

Correspondence to:
Dr Edwin Chia Kian Heng,
Head Dental Branch, HQ Medical Corps,
701 Transit Road, #04-01, Singapore 778910.
E-mail: periodontist@hotmail.com

Periodontal surgery to resolve intrabony lesions can be resective or regenerative. Osseous resective surgery involves the combined use of osteoplasty and ostectomy to re-establish the marginal bone morphology around the affected tooth, so that it resembles "normal bone with a positive architecture", albeit at a more apical position.[3] Periodontal regeneration is the reproduction or reconstitution of a lost or injured part of the tooth-supporting tissues, including cementum, gingiva, periodontal ligament and alveolar bone.[4] However, it should be highlighted that periodontal repair rather than true regeneration, may be obtained in many regenerative techniques utilizing the guided tissue regeneration (GTR) concept. The key distinction is the presence of a long junctional epithelium in periodontal repair that can be seen histologically. However, the controversy is that there is really no definite conclusion in the current periodontal literature to state

that periodontal repair is inferior to regeneration in the clinical setting or vice versa.

The aim of this paper is to showcase a technique of treating deep intrabony defects through regenerative techniques utilizing only autogenous materials (intraoral bone grafts and connective tissue) from the patient. Two patient case reports presenting localized deep intrabony defects are also illustrated.

Case Report

Patient A (Figures 1–14)

Patient A (aged 42 years old) was referred for periodontal treatment. Her general medical history and social history were not significant. She complained of gingival bleeding on brushing and "bad taste" from the upper left posterior region. She started noticing these signs for the past 8 months. Intra-oral examination revealed that there was

periodontal pocketing of 5–8 mm at the upper left molars, with the deepest probing of 8 mm at the mesial of tooth 26 (Figures 1 and 2). Radiographical examination confirmed the presence of

Figure 3. *Pre-op radiograph (probing of 8 mm at #26 mesial).*

Figure 1. *Pre-op (buccal view).*

Figure 4. *Incisions (buccal view).*

Figure 2. *Pre-op (palatal view).*

Figure 5. *Incisions (palatal view).*

an angular intrabony defect at mesial of tooth 26 and mild to moderate generalized bone loss at this sextant (Figure 3). All teeth in this upper left quadrant were not mobile.

After two sessions of root planing, there was no improvement in probing depth. A regenerative approach to treat the defect surgically was deliberated. A pocket reduction procedure was also planned to reduce the probing depths at this quadrant.

Figure 9. *Connective tissue from distal wedge.*

Figure 6. *Incisions (occlusal view).*

Figure 7. *Elevation of flap (buccal view).*
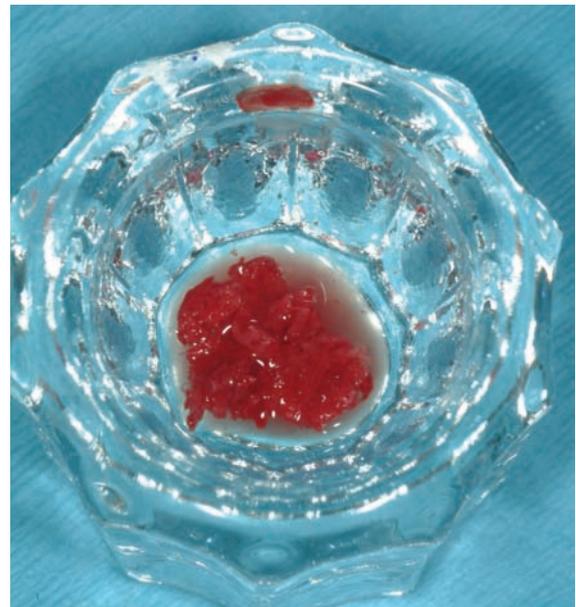
Figure 10. *Autogenous bone from palate.*

Figure 8. *Elevation of flap (occlusal mirror view).*

Figure 11. *Packing of autogenous bone into intrabony defect.*

Figure 12. *Suturing of connective tissue over bone defect and flap closure (palatal mirror view).*

Figure 15. *Pre-op (buccal view).*

Figure 13. *Post-op 1 year (occlusal mirror view).*

Figure 14. *Post-op 1 year (probing of 2 mm at #26 mesial).*

Submarginal incisions of 2 mm and a distal wedge were made to effect pocket reduction (Figures 4–6). The flaps were raised and all granulation tissue was thoroughly debrided from

intrabony defect (Figures 7 and 8). A sleeve of connective tissue was obtained from the excised distal wedge (Figure 9) and autogenous bone was collected via rounding off irregular bone margins with a back-action chisel (Figure 10). The collected bone was packed into the mesial defect at tooth 26 with a condenser (Figure 11). The wedge of connective tissue was then sutured over the defect with a 4-0 silk sling suture around tooth 26, simulating a "natural barrier membrane". This was followed by closure of the periodontal surgical site, with 4-0 silk vertical mattress sutures (Figure 12). Postoperative instructions were given, and patient was prescribed 5 days of paracetamol (500 mg, 3 times daily) for postoperative pain relief and a bottle of chlorhexidine mouthwash to be used three times daily.

Suture removal was done a week later. There was postoperative gingival recession of 2 mm on both buccal and palatal aspects, but this was attributed to the pocket reduction submarginal incisions we had done during the surgery.

Uneventful healing was observed for 1 week, 1 month, 3 months and 1 year postoperatively (Figure 13). The 1-year postoperative radiograph was taken and the probing depth recorded at tooth 26 mesial was 2 mm (Figure 14).

Patient B (Figures 15–24)
Patient B (aged 50 years old) was referred to the periodontal department for follow-up treatment on a localized intrabony defect at mesial of tooth 16. At the initial visit, tooth 47 was already extracted by her referring dentist due to poor prognosis (Figure 15), and two rounds of gross scaling and root planing had already been done. Patient

Figure 16. *Pre-op radiograph (9 mm probing at mesial of #16).*

Figure 17. *Incision and flap elevation (buccal view).*

Figure 18. *The intrabony defect (palatal view).*

Figure 19. *Packing of autogenous bone into bony defect.*

Figure 20. *Placement of connective tissue over autogenous bone.*

had no relevant medical and social history. She complained of frequent food impaction at the upper right quadrant. On clinical inspection, there was a deep probing of 9 mm on the mesial of tooth 16 which matched with the radiograph provided by her referring dentist (Figure 16).

An intra-sulcular flap with vertical releasing incisions from teeth 15 (mesial) to 17 (mesial) was raised (Figure 17). The intrabony defect at tooth 16 (mesial) was thoroughly debrided of granulation tissue (Figure 18). Autogenous bone was obtained from the palatal vault with a back-action chisel and then packed into the defect with a condenser (Figure 19). A wedge of connective tissue was obtained through thinning the palatal flap on the inside, and this tissue was then placed above the packed autogenous bone (Figure 20). The author ensured that this connective tissue barrier could be stabilized by the overlying flap on both sides before final closure of the periodontal

Figure 21. *Flap sutured (buccal view).*

Figure 22. *Flap sutured (palatal view).*

Figure 23. *Post-op 1 year (palatal view).*

surgical site with 4-0 silk simple interrupted sutures (Figures 21 and 22). Postoperative instructions were given, and the patient was prescribed 5 days of paracetamol (500 mg, three times a day) for pain control and a bottle of chlorhexidine mouthwash to be used three times a day. Suture removal was done a week later. No gingival recession was observed.
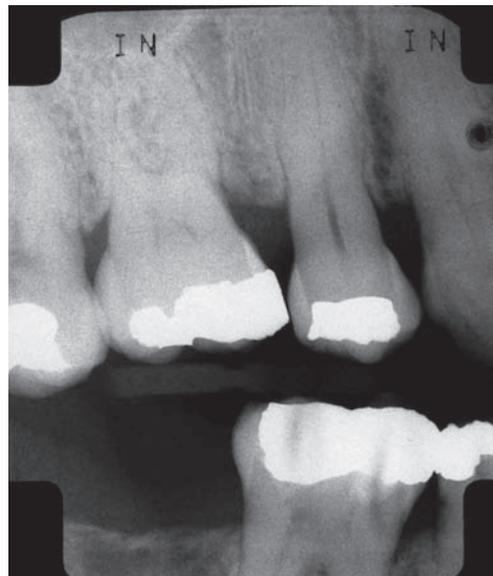

Figure 24. *Post-op 1 year (probing of 2 mm at mesial of #16).*

Further uneventful healing was observed for 1 week, 1 month, 3 months, 6 months and 1 year postoperatively (Figure 23). The 1-year postoperative radiograph was taken and the corresponding probing depth recorded at tooth 16 (mesial) was 2 mm (Figure 24).

Discussion

The general objective of periodontal therapy is not aimed at just inflammatory control of gingival tissues, but also at pocket reduction and correction of associated bony defects. In moderate to severe intrabony defects, the regeneration of the lost bone and periodontal attachment improves tooth support and long-term prognosis.

A review of controlled studies suggests that more clinical bone fill is usually obtained in grafted (3 mm bone fill) rather than nongrafted sites (<1 mm of bone fill) in the treatment of moderate to severe intrabony defects.[5]

Utilizing autogenous bone grafts is still considered to be the "gold standard" in bone grafting dental procedures. Other existing sources of bone replacement grafts include allografts (e.g. freeze-dried bone allografts and demineralized freeze-dried bone allografts), xenografts (e.g. bovine-derived hydroxyapatite) and alloplasts (e.g. tricalcium phosphate and synthetic bioactive glasses).

The ideal bone graft should be able to trigger osteogenesis that is only achieved with autogenous grafts due to the presence of progenitor stem cells within the grafts.[6] The other types of bone grafts do not have such cells. After osteogenesis, the next best scenario would be osteoinduction that involves the stimulation of surrounding progenitor cells around the existing defect to produce osseous tissue. To date, only demineralized freeze-dried bone allografts (DFDBA) have been shown to exhibit osteoinductive capabilities due to the presence of bone morphogenetic proteins (BMPs). The remaining bone grafts are osteoconductive, and they simply provide "building scaffolds" to allow osseous ingrowth and matrix deposition.

Clinical successes have been documented in the use of autogenous bone grafts harvested from intraoral sites in the treatment of intrabony defects.[7–9] Common donor intraoral sources are the maxillary tuberosities, exostoses, existing edentulous alveolar areas and healing extraction sockets.

In a separate scenario, the sole use of autogenous periosteal grafts in the treatment of intrabony defects have also been shown with predictable results.[10,11] Clinical attachment gain obtained could be due to

1. increased resistance to probe penetration caused by a reduction in inflammation;
2. reformation of gingival fibres;
3. formation of long junctional epithelium;
4. connective tissue attachment similar to that, which has been observed following mucogingival procedures.[12]

Periodontal flap surgery usually results in postoperative recession. It is noteworthy that in the previous studies when postoperative recession was compared using GTR membranes and subpedicle connective tissue grafts in mucogingival correction, there was lesser recession with the connective tissue grafts.[13]

A similar new periodontal attachment procedure based on retardation of epithelial migration was shown with successful results in the early 1970s.[14] This procedure utilized the use of autogenous bone and free gingival grafts from the palate. For the two cases illustrated in this report, the use of the connective tissue graft as a natural barrier would give a better colour blend in comparison to the free graft.

Hence, knowing that osteogenesis can occur through the use of autogenous bone grafts in intrabony defects, plus the added potential of connective tissue grafts as "natural membrane barriers" in gaining some attachment and giving better gingival aesthetics, the author has combined the use of both autogenous materials in enhancing this regenerative therapy of treating intrabony defects. The author believes that this regenerative technique would bring the best possible healing outcome for treating intrabony defects.

Conclusion

In conclusion, periodontal regenerative techniques show great promise in the treatment of intrabony defects. It would definitely be any clinician's professional satisfaction to arrest the ongoing periodontal disease and to restore lost and injured tissues for affected patients. The combined use of autogenous bone as a filling material in the intrabony defect and an overlying connective tissue graft as a "natural membrane barrier" would give the best possible healing potential in such patients.

Acknowledgements

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Management of a Three-rooted Mandibular Second Premolar Diagnosed with Periodontal–Endodontic Disease: A Case Report

Dalia Abdullah BDS (Malaya) MClinDent Endodontology (London) FDSRCSEdin

Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia.

Abstract

This case report describes the clinical management of a three-rooted lower mandibular second premolar presented with a periodontal–endodontic (perio-endo) lesion. The lower mandibular second premolars have been shown to exhibit an aberrant canal anatomy, and the three-rooted mandibular premolar is a rare variant of its usual morphology. The importance to recognize any additional roots and canals that may present in this tooth could not be overemphasized to avoid treatment failure. The clinical examination and radiographical evaluation of tooth 45 presented with a perio-endo lesion was described in this study. Perio-endo lesions are difficult to classify and diagnose, especially at the late stage, as they lack characteristic manifestations of strictly endodontic or periodontal lesions. The progression of the periodontal lesion in this case could be promoted to a larger extent by the morphology of the root complex structures. [*Singapore Dent J* 2009;30(1):39–46]

Key Words: mandibular premolar, nickel–titanium instrument, perio-endo lesion, three-rooted

Introduction

Success of root canal treatment depends on thorough cleaning, shaping and complete obturation of the pulp spaces, to be followed by placement of coronal restoration to prevent future recontamination.[1] In everyday clinical practice, practitioners would be treating root canals, most of the time, with predictable morphology as described in the textbook. In a situation at which the tooth exhibits variation of its internal root canal configuration, possessing the knowledge, skill and proper equipment, will facilitate the clinician in locating and treating the entire root canal system.

Mandibular premolars are probably one of the most difficult teeth to treat endodontically because of its variation in the internal canal morphology. There are not many studies available in the literature on the root canal morphology of mandibular second premolars. The existing studies using tooth-clearing technique revealed that most mandibular second premolars were single-rooted, with no findings of the tooth with three roots.[2–7] In these studies, although single canal is predominant, variations occur mainly in the number of canals, number of foramen and the internal canal configurations. Three-rooted mandibular second premolar is an extremely rare variant from its usual anatomy. In one particular study, Zillich and Dowson studied 906 mandibular second premolar teeth using radiographical examination and found only 0.4% of the teeth exhibited

Correspondence to:
Dr Dalia Abdullah,
Faculty of Dentistry,
Universiti Kebangsaan Malaysia,
Jalan Raja Muda Abdul Aziz,
50300 Kuala Lumpur,
Malaysia.
Telephone: +603 9289 7769 (O)
        +601 2225 7585 (Mobile)
Fax: +603 9289 7794
E-mail: daliaabd@medic.ukm.my,
    daliaabd@hotmail.com

three roots.[8] The information on multiple canals and multiple roots of this type of tooth could be found mostly from the case reports. Two clinical reports have been published recently to describe the infrequent cases of three-rooted mandibular second premolars with three canals presented with chronic apical periodontitis.[9,10]

The periodontal–endodontic (perio-endo) lesion can be defined as an infrabony, marginal pocket communicating with the apical area of the tooth with an infected pulp.[11] The lesion involves both periodontal and pulpal tissues and could mimic either the endodontic or the periodontal disease. Both primary endodontic and periodontal diseases are caused by bacterial infection. Studies have shown that great similarities exit between the microbiodata of endodontic and periodontal lesions (i.e. mostly of a mixed anaerobes).[12] The current literature contains no description on the microbiodata of the endo-perio lesion, and this may be due to the difficulty in obtaining the samples. With the similarities of bacterial flora when the lesion occurs independently, it is not surprising if the endo-perio lesion might not show unique microbiological profile.[12]

In 1972, Simon et al[13] classified the perio-endo lesion into the following group: (i) primary endodontic lesion with secondary periodontal involvement, (ii) primary periodontal lesions with secondary endodontic involvement and (iii) true/combined endo-perio lesion. Primary endodontic lesion with secondary periodontal involvement occurs when a tooth with an endodontic disease advances coronally to initiate marginal periodontitis. Clinically, this condition may mimic the presence of periodontal abscess especially with the formation of a deep pocket.[14] This pocket is narrower (more like a pseudo-pocket), unlike the pocket caused by periodontal disease, which is broader. Poor oral hygiene will lead to accumulation of plaque and calculus in the pocket causing progressive secondary marginal periodontal breakdown with eventual downgrowth of epithelium into the pocket complicating the condition further. Primary periodontal lesion with secondary endodontic involvement occurs when periodontal disease progresses apically to involve the pulpal tissue and initiates pulpal pathosis.[13] Although not much is known on the effect of periodontal disease on the pulpal tissues, there are reports on substantial pathological change and necrosis in the pulp tissues due to long-standing periodontal disease, especially in the presence of accessory canals. However, the results of these findings were controversial. Langeland et al studied the 60 non-carious teeth with various degrees of periodontitis and demonstrated that pathological changes do occur in the pulp in the presence of periodontal disease.[15] They found that as long as the apical foramen is not involved, the pulp remains vital. This shows that periodontal disease does not usually affect the vital functions of the pulp until the disease process has reached the apical area and disturbs the main blood supply flowing through the apical foramen. There are conflicting evidences on whether the periodontal treatment itself has untoward effect on the pulp due to the removal of the protective layer of cementum, thus exposing the dentinal tubules during the root planning.[16] Healthy pulp tissue is highly vascular and quite resistant to infection. The outward flow of the dentinal fluid on removal of the cementum could be expected to reduce the inward diffusion of noxious bacterial products and protect the pulp.[17] True perio-endo lesion refers to the existence of two disease entities on the same tooth. These two diseases can either exist separately or merge to form one large lesion.[13,16,17] Therefore, these lesions are often indistinguishable from an advanced primary endodontic lesion with secondary periodontal involvement and/or a primary periodontal lesion with secondary endodontic involvement.

Even with this classification and its description, a non-vital tooth with periodontal involvement at a late stage is a challenge to diagnose. Clinicians often find it difficult to determine the primary location of the infection, and especially to find in which direction the spread of the disease occurs. The lesion becomes indistinguishable and interpretation of the clinical findings may vary on which disease comes first. Finding the right aetiology however is essential so that correct therapy can be rendered. In most cases, root canal treatment needs to be completed and evaluated before deciding whether to proceed with periodontal treatment.[13]

This case report describes the clinical management of a three-rooted mandibular second premolar presented with a perio-endo lesion.

Case Report

A 54-year-old male Malay patient with a non-contributory medical history was referred to the endodontic specialist clinic. At his first visit in the primary screening clinic in July 2005, he complained of discharging pus from both lower right and left quadrant. He was under the care of a periodontist at one of the government clinics, from which he was taking treatment for his periodontal disease. Based on the case note, examination revealed the presence of discharging pus from the area of teeth 45 and 33. Radiographical finding from an orthopantomograph (brought by the patient) revealed the presence of periodontal lesion distal to teeth 45 and 33. Unfortunately, vitality testing was not noted in the case note. No other radiograph was taken. A diagnosis of localized chronic periodontitis was made and drainage of the pus by scaling with an ultrasonic and with hand scalers was carried out at the area of teeth 45 and 33. The patient was given an appointment for follow-up, but failed to attend. In September 2005, the patient came again to the clinic complaining of severe sensitivity from the lower right region before it progressed to severe spontaneous pain. Then he noted that his right mandibular second premolar was painful on biting, and there was swelling with recurrence of pus discharging from the gum. Upon examination, it was found that tooth 45 had sound clinical crown. It was non-responsive to vitality testing. The attending dental officer carried out the pulp extirpation procedure, followed by deep scaling around the tooth.

Patient was seen in endodontic clinic after 5 months in the waiting list. At the first visit, he complained of occasional pain in tooth 45. Clinical examination revealed an occlusal access cavity in tooth 45. Tooth was tender to percussion with Grade II mobility. The surrounding gum was inflamed with pus discharging from the distal area. Periodontal probing revealed 8 mm pocket at the distal and mid-buccal area with 4 mm gum recession buccally. Further probing at the buccal area revealed a Grade I furcation involvement. Radiographically, the root canal morphology of tooth 45 displayed an abnormal anatomical configuration with the presence of three roots surrounded by periradicular radiolucency (Figure 1). A vertical bony defect extending to the apices of the roots was present at the distal aspect of
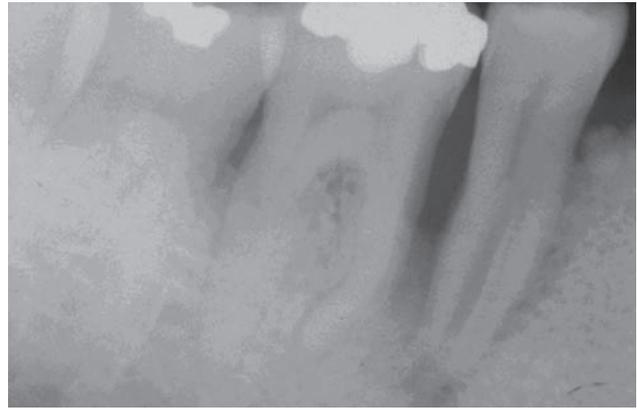

Figure 1. *Preoperative radiograph of tooth 45.*

the tooth. General examination of the mouth revealed that the patient was missing teeth 16 and 44, with localized periodontitis associated with accumulation of plaque and calculus.

Differential Diagnosis

A diagnosis of an advanced perio-endo lesion of tooth 45 (primary periodontic in origin) was made. It was decided that the non-surgical root canal treatment would be carried out, because the patient was undergoing periodontal maintenance. The patient strongly wished to save the tooth and consented to the treatment after being informed about the treatment plan, the procedures and the prognosis of the tooth.

Treatment

Root canal treatment was carried out in two visits and a surgical microscope (OPMI Pico Zeiss Dental Microscope, Germany) was used during the treatment. After administration of local anaesthesia (2% lignocaine 1:100,000 adrenaline), the tooth was isolated with rubber dam, and the temporary dressing was removed. The shape of the pulp chamber was found to be similar to a taurodont tooth: that is, a long/tall pulp chamber with the furcation positioned apically. Three orifices were found on the pulpal floor: mesiobuccal (MB), distobuccal (DB) and lingual (L) orifices. Cavity was modified to ensure a straight-line access. Coronal flaring of the canals was carried out with Gates Glidden drills size 2 and 3 (Dentsply Maillefer, Baillaigues, Switzerland), and working

lengths were determined with the use of Elements Diagnostic Unit (SybronEndo, Orange, CA). Sodium hypochlorite 5.25% was used as the irrigating solution. The canals were then cleaned and prepared with ProTaper hand files (Dentsply Maillefer, Baillaigues, Switzerland) according to its sequence (from the shaping files S1, S2 to finishing file, F1). Apical preparation was established at file size 20 (F1) for all canals. All canals had separate portal of exits and patency was maintained with a size 10 K-file. Calcium hydroxide (UltraCal XS, Ultradent, USA) was used as intracanal medication and the access cavity was filled with IRM (Caulk, Dentsply, USA) between appointments.

Four weeks later, the patient was pain-free and pus exudates had disappeared. The tooth, however, was still mobile and tender on biting hard food. The canals were irrigated again to remove calcium hydroxide. Both EDTA solution (17%) and sodium hypochlorite were used in the final rinse before obturation. The canals were dried and were fitted with ProTaper non-standardized gutta percha points (F1). Warm vertical condensation technique was used to obturate all canals, and an amalgam core was placed inside the pulp chamber. A radiograph was taken to assess the quality of obturation (Figure 2).

Patient was seen again for review 1 year after root canal treatment and remained asymptomatic (Figure 3). The probing depth was at 4 mm at the distal aspect of the tooth with Grade II mobility. The patient was on periodontal maintenance phase and exhibited good oral hygiene. At 2-year recall, there was evidence of healing with increased bone density between teeth 46 and 45 (Figures 4 and 5).

Discussion

Anatomical variations of mandibular premolars are well documented in the literature, and the

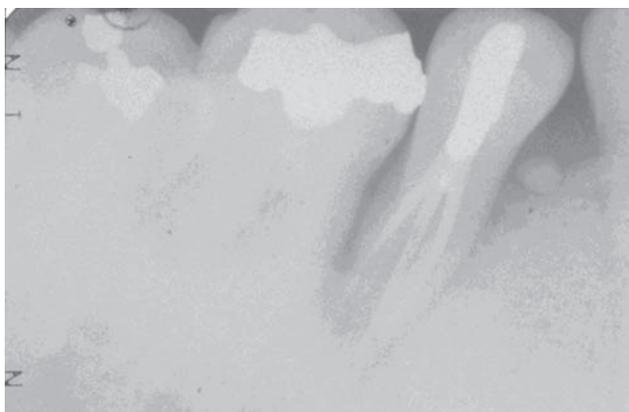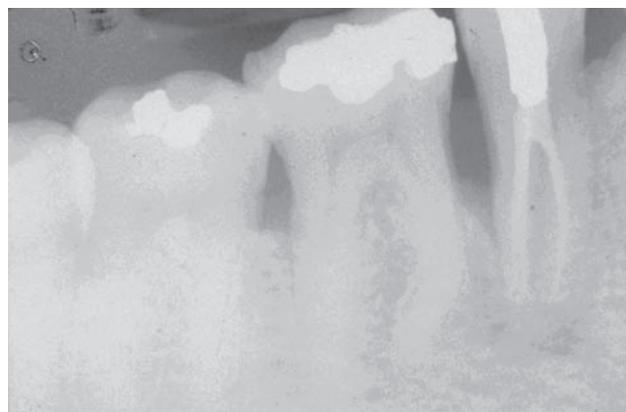**Figure 2.** *Periapical radiograph after obturation.*

Figure 4. *Periapical radiograph of 45 at 2-year recall.*

Figure 3. *Clinical picture of tooth 45 at 1-year recall.*

Figure 5. *Clinical picture of tooth 45 at 2-year recall.*

three-rooted mandibular second premolars appear to be a rare occurrence. Variations in root canal morphology had been associated with racial groups. Trope et al in a radiographical study of the root canal morphology of mandibular premolars found a significantly higher incidence of multiple roots in Black patients compared with that of the White population.[18] Walker (1988) and Lu et al (2006) studied the root canal morphology of the mandibular first premolars in Chinese population and found that Asians have different percentages of canal configurations than those reported in Caucasians and Africans.[19–21] It could be expected for the mandibular second premolars to show the same preponderance. There is, however, a lack of available information on the root canal morphology on Malaysians (comprises three main races of Malay, Chinese and Indian) that needs to be addressed.

Detection of possible existence of extra roots and complex root canal system is not always easy even using radiographical examination. When present, the additional roots are usually close to each other and sometimes appear as a bulky single root. The extra canals will usually bifurcate at the deeper part of the root and, most of the time, are not clearly visible in the radiographs. A single preoperative radiograph may not be able to accurately determine the root canal morphology, as it is only two-dimensional with the superimposition of the root images. Martinez-Lozano et al examined the effect of X-ray tube inclination on determining the root canal system in premolar teeth and found that inclining the tube 40° at horizontal angle would give the correct morphology of the mandibular premolars.[22] The radiograph should be of good quality and should be carefully examined. Use the image by tracing the external outline of the tooth to make out the number of roots, followed by tracing the internal pulp chamber and the canals. Any intersecting lines could indicate additional roots/canals. In the present case, the three roots of tooth 45 were clearly visible due to the absence of alveolar bone at the distal and inter-radicular area. The image of tooth trunk appeared to be separated into three roots with the lingual root visible in between the two buccal roots. The roots appeared convergent towards the apex and are close to each other.

Having the proper armamentarium is essential to locate all the orifices and negotiate into the root canals. In this case, the surgical microscope has been found useful to detect the orifices as the floor of the pulp chamber could be visualized and examined in detail, even though it was located halfway into the tooth. Lines on the pulpal floor connecting the root canal orifices could be traced clearly, indicating the position of the orifices. With the presence of extra canals, the shape of the pulp chamber in this premolar differed from the normal morphology, resembling the morphology of a maxillary molar. The shape of the access cavity was made more triangular than the usual oval-shape cavity. The orifices are located at the angles in the floor–wall junction, and the orifices were indicated as the mesiobuccal, distobuccal and lingual canal (Figure 6). Canal shaping was carried out using the nickel–titanium hand ProTaper instruments, with predictable results. As the roots appeared quite thin and slender, the apical portion was enlarged sufficiently with the finishing file, F1, with the tip diameter of size 20.

Tooth 45 was presented with an advanced perio-endo lesion. It is indeed difficult to distinguish which disease comes first in this situation. The dental history, clinical findings and the appearance of the lesion in the preoperative radiograph tend to suggest that pulpal necrosis occurred secondary to the periodontal lesion. The patient had previously been treated for his periodontal disease when he began experiencing symptoms of irreversible pulpitis. The patient, however, was not able to give the history of previous periodontal treatment in detail. At the first visit to our centre,
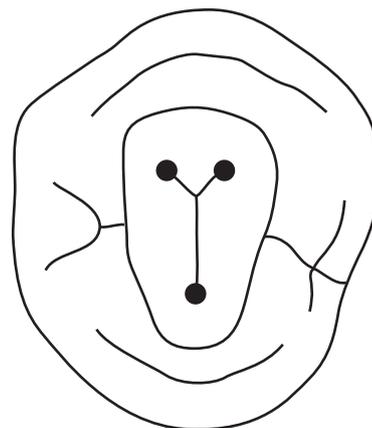

Figure 6. *Diagramatic drawing of the canal orifices in tooth 45 (two at the buccal and one at the lingual).*

tooth 45 exhibited sound and intact clinical crown indicating that the portal of entry for bacteria causing pulpal necrosis was not from the crown. Tooth fracture or crack was ruled out upon examination. Periodontal probing revealed 8 mm broad-based pockets at distal area that was characteristic of periodontal disease. Periodontal pockets at localized areas associated with accumulation of plaque and calculus could be seen on the contralateral side of the jaw as well. There was a 4 mm gum recession at the buccal aspect that exposed the furcal area. Vertical bone resorption could be seen in the preoperative radiograph extending from the coronal to the apex of the tooth, giving the appearance that the disease has spread to the apical foramen to involve the pulpal tissues. The apical radiolucency was directly due to process of inflammation of the canals. The presence of concavities and ridges in the inter-radicular area of the three convergent roots could have promoted the progression of the destructive periodontitis lesion apically. The exposed furcal region was a plaque retentive area and compromised the patient's ability to perform oral hygiene procedure, as this area was difficult to access for plaque removal. Mandibular second premolars have a relatively high incidence of possessing lateral canals. Vertucci reported that 48.3% of mandibular second premolars in his study presented with lateral canals, and these canals could be found at the cervical (3.2%), the middle (16.4%) and the apical part of the root (80.1%).[6] These canals potentially provide pathways in the exchange of bacteria and inflammatory products between the periodontal tissues and the pulp.

The prognosis of perio-endo lesion depends primarily on the severity of the periodontal disease and the response to periodontal treatment.[17] With adequate endodontic therapy and efficacy of the periodontal treatment, this case demonstrated that it is possible to retain a tooth with a perio-endo disease. In describing the treatment concept of perio-endo lesion, Haueisen et al suggested that priority should be given for the endodontic lesion to heal before evaluating the periodontal tissues and initiating the treatment on residual periodontal defects by regenerative periodontal techniques.[23] Tooth should be reviewed at 6 month interval, and it can be definitively determined after about 18 months post-treatment whether the root canal treatment is successful or not.[23] This is to allow for regeneration of the bony defect induced by the endodontic lesion. In this case, the patient was seen twice for recall appointments for a period of 2 years. The endodontic lesion of tooth 45 subsequently healed as evident from the 2-year recall radiograph, which showed the absence of apical radiolucency as compared with the preoperative radiograph. The distal radiolucency, however, was not completely resolved. This is not a surprising outcome as non-surgical periodontal therapy would result in the arrest of disease progression with some attachment gain, but without significant bony regeneration.

Periapical radiograph plays an important role to monitor the progress of bony healing and should be of good quality for correct interpretation. Radiographical images, however, are subject to changes in angulation and contrast.[24] It should be noted that the pre- and postoperative radiographs in this case were taken with film-holders, but were not taken from the same angle giving the appearance of different bone heights especially at the distal aspect. Ideally, the radiographical technique should be standardized so that the images produced on the film would be at the same angle and would give a better interpretation. The angular appearance of the radiolucent area in this case is suggestive of a two-wall or a three-wall bony defect. Surgical periodontal regenerative therapy could be carried out to increase the distal bone height and improve the prognosis of the tooth. Improved clinical attachment levels and clinical furcation closure may be obtained with the use of several procedures, such as bone grafts, guided tissue regeneration and coronally positioned flaps. The surgical alteration of the root morphology to allow easy cleaning of the furcal area would not be possible due to the close position of all the three roots.

The patient had been regularly seen by our periodontist and was now very motivated in maintaining his oral hygiene. He had been given oral health education, oral hygiene instruction on brushing technique, followed by scaling and root planning of the localized areas. At the last evaluation, the patient has good plaque control and healing of the tissues was good as a result of the hygienic therapy. Prognosis of tooth 45 was initially questionable due to the presence of

persistent infection and deep pocket. With the regeneration of bone at the apical area, the bone support has increased dramatically. The exposed furcal area may in part play a favourable role as the patient had direct access to clean this area and brushing thoroughly would remove most of the plaque at least 2 mm subgingivally.

Conclusions

Diagnosing the types of perio-endo lesion is a challenge as the clinical and radiographical presentations are very similar. Thorough evaluation of the tissues must be carried out to determine the most probable cause, the course of disease and the most probable treatment. In the presence of pulpal disease, the periodontium must always be evaluated and, likewise, when periodontal disease exists, the state of the pulp must be ascertained.

In perio-endo lesions, when the endodontic treatment is adequate, the lesion of an endodontic origin will heal. Prognosis of these lesions often depends on the efficacy of the periodontal therapy. In this case, even though there have been improvements in the symptoms and evidence of bone healing radiographically, prognosis of the tooth remains questionable and depends mainly on the routine maintenance of the periodontal disease even when the patient exhibited good oral hygiene. Several longitudinal studies of treated periodontitis patients had unequivocally demonstrated that furcation-involved teeth are at high risk for extraction even in a situation where supportive periodontal therapy is provided.[25,26]

Acknowledgements

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Book Title: Fiber Posts and Endodontically Treated Teeth: A Compendium of Scientific and Clinical Perspectives

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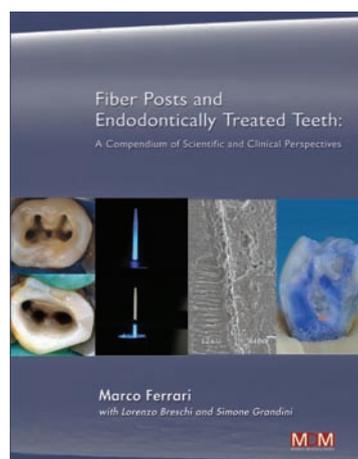
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Differences between the root dentine and coronal dentin and modifications that affect the dentine during post space preparation are highlighted as well as the interactions with the various adhesive systems. The prerequisites for successful post placement namely endodontic treatment, retreatment, and post space preparation are thoroughly covered. The authors also clearly explain how to take full advantage of the results published in research papers by understanding these actual research protocols and their limitations.

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Finally, Professor Franklin Tay superbly discusses the changes in clinical practice that have been achieved with fiber posts and adhesive technology. This book has high-quality graphics, which is a valuable reference for those dentists who are always on the lookout to improve their clinical practice.

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• Abstracts should not be cited unless the abstract is the only available reference to an important concept.
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• Abbreviations for journals should conform to those used in *Index Medicus*.
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- Authors are responsible for the accuracy and completeness of their references and for correct text citation.

Examples are given below.

Journal articles:

1. Chew MT, Sandham A. An assessment of orthodontic treatment using occlusal indices. *Singapore Dent J* 2001;24:9–16.
2. Smith RN, Rawlinson A, Lath D, Elcock C, Walsh TF, Brook AH. Quantification of dental plaque on lingual tooth surfaces using image analysis: reliability and validation. *J Clin Periodontol* 2004;31:569–73.
3. Olszewski R, Reychler H. Limitations of orthognathic models surgery: theoretical and practical implications. *Rev Stomatol Chir Maxillofac* 2004;105:165–9. [In French]

Books:

1. Stevens J. *Applied Multivariate Statistics for the Social Sciences*, 3rd edition. New Jersey: Lawrence Erlbaum Associates, 1996.
2. Sapp JP, Eversole LR, Wysocki GP. Infections of Teeth and Bone. In: Sapp JP, Eversole LR, Wysocki GP. *Contemporary Oral and Maxillofacial Pathology*, 2nd edition. St Louis: Mosby, 2004:70–93.

Report:

1. Committee on Mercury Hazards in Dentistry. *Code of Practice for Dental Mercury Hygiene.* London: Department of Health and Social Security, 1979, publication no. DHSS 79-F-372.

Tables

Tables should be labelled in Arabic numerals and titled concisely. Number all tables in the order of their citation in the text. Tables should be typed double-spaced in as simple a form as possible. Abbreviations used in the table and not defined in the text should be defined in footnotes using these symbols (in order of appearance): *, †, ‡, §, ||, ¶.

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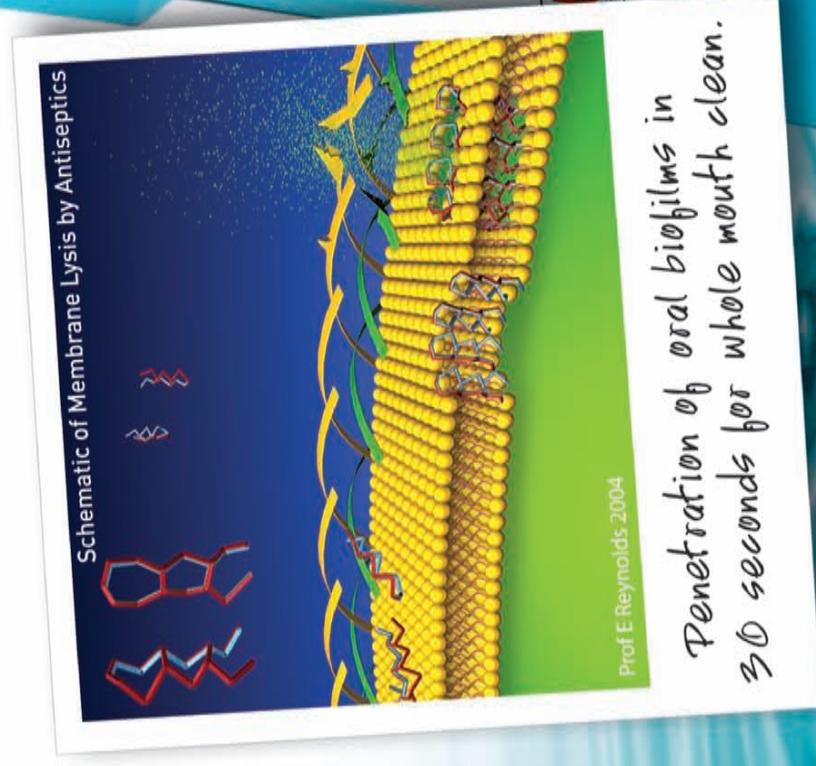
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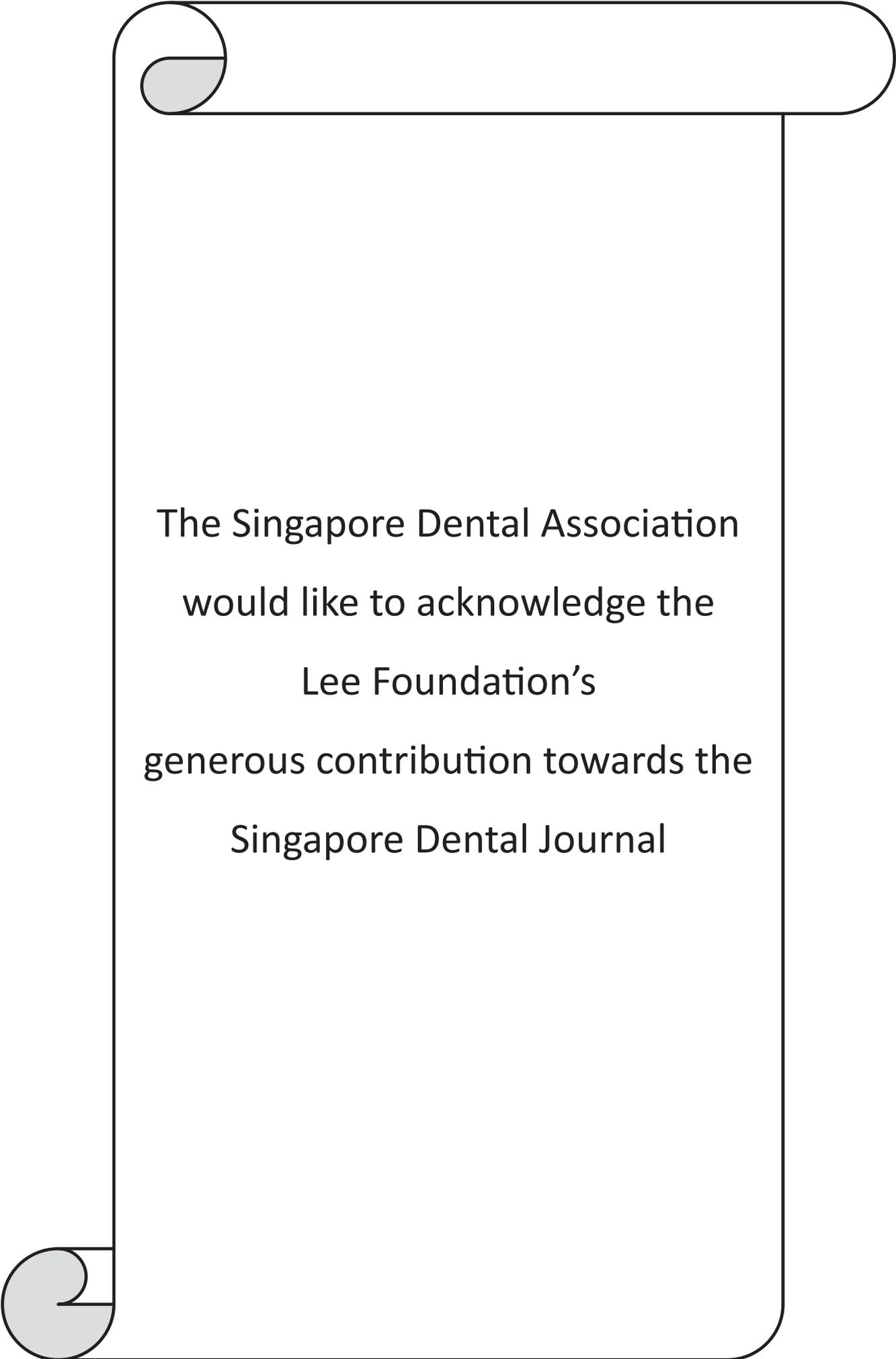
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1. Ouhayoun J-P. Penetrating the plaque biofilm: Impact of essential oil mouthwash. J Clin Periodontol. 2003;30:10-12.

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