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INVITED EDITORIAL

The Fluoride Dilemma

We consider fluoride to be an absolutely essential ingredient for development of sound teeth. At the same time it has been a proven fact that it reduces dental caries. Hence, fluoride is added to toothpaste to obtain the beneficial effects. What happens when fluoride is available in excessive amounts? Does it enhance the good effects? No. Absolutely not, this will lead to dental fluorosis with mottling where the teeth would be subjected to an unaesthetic discolouration ranging from chalky white to yellow to a brown or black discoloration. If fluoride is available in very high amounts it can affect the skeletal system with brittled bone leading to skeletal fluorosis where the patients can get even crippled! Currently, it has been also hypothesized on the evidence available that excess fluoride in addition to having bad effects on teeth and bone, to have a damaging effect on the kidneys. Several patients have been identified from endemic areas containing very high fluoride levels ranging over five ppm and above in the drinking water to suffer from kidney failure.

Going through the above facts, it is evident that fluoride has it's very good effects on teeth and at the same time serious bad effects on teeth bone and kidneys. In this context, fluoride in our own sinhala language can be considered as a " Athuwath barry Nathuwath barry " (ඇතුවත් බැරි නැතුවත් බැරි) element. But yet no one has absolutely come out with a strategy to prevent the bad effects. This as become a globel issue now. At a time where aesthetic and cosmetic dentistry have become the order of the day and since every patient expects his or her dentist to

give the best possible smile irrespective of one's age, gender or disease status, it is of paramount importance to minimize or eliminate these bad effects.

Considering the above, what has the profession or the health providers in Sri Lanka done to prevent the dangerous effects of fluoride. The SLDA has carried out much research project to find a simple an inexpensive method of treating the ugly, unsightly dental stain with promising results to some extend. But as the saying goes "prevention is better than cure", have we done everything possible to prevent and eliminate this unsightly stain and associated effects, specially from the North Central Province of Sri Lanka where the prevalence of dental fluorosis is the highest.

I am aware that at this point in time a research team consisting of various experts including chemists and dental experts and engineers are looking at ways and means of reducing the excess fluoride from the drinking water by constructing a defluoridation plant as a pilot project in Vilachchi in the Anuradhapura District. Hope this would bring successful results in the future and provide a solace to the affected community by eliminating the bad effects to counteract dental fluorosis, skeletal fluorosis and suspected kidney failure associated with high fluoride.

Be that as it may, until something concrete takes place, in this country can every household in these endemic areas be helped to constrct a fluoride filter (which has been extensively researched) like having a pounding stone (mirisgala) in every home.

This is food for thought for everybody.

R.L. Wijeyeweera

Letter to the Editor

Sri Lanka Dental Journal 2010; 40(02-03) 50-53

A future for forensic dentistry in Sri Lanka : Facing challenges, deconstructing skeptics and inciting reality

Induwara Goonerathne

There have been multifarious discussions at miscellaneous official local caucuses regarding the establishment of forensic dentistry as a specialty in Sri Lanka. These forums spanned from Sri Lanka Postgraduate Institute of Medicine (PGIM)-, Board of study in Dental Surgery, Board of Study in Forensic Medicine, Faculty Board of the Faculty of Dental Sciences, Sri Lanka Dental Association, to College of Dentistry and Stomatology of Sri Lanka. The expressions of interest of the penury to establish forensic dentistry as a specialty in Sri Lanka has been obvious, unambiguous and unanimous within all these responsible bodies. Such discussions have been in place, as far as the author can recall, since early 1990s. Professor BRRN Mendis, Dr. Ajith Ranasinghe, as Deans of the Faculty of Dental Sciences and late Prof. C J Babapulle, as the, then Professor and Head of Department of Forensic Medicine at University of Peradeniya have contacted the author and communicated the essence of these discussions. Due to several reasonable logistical grounds such moves faded away in the past: the preeminent reason being, the lack of experts in the field of forensic dentistry in Sri Lanka, at that time. Thus, the implementation process of the discipline of forensic dentistry in Sri Lanka suffered several repeated spontaneous "abortions".

The need for forensic dentistry in Sri Lanka was notably felt in the recent Tsunami disaster that eliminated a substantial number of Sri Lankans. While virtually all foreign victims deceased in Sri Lanka in the Tsunami waves, were positively identified, a majority of locals could not be recognized using standard but quotidian medico-legal techniques. Surprisingly, none of the victims could be positively identified using forensic dental techniques in Sri Lanka. In contrast, for example, a majority of victims in both 9/11 attack and the Katrina disaster in the US were positively identified with the avail of forensic dentistry, at a low cost. This exemplification overwhelmingly demonstrates the unpreparedness of our domestic strategy for such a disaster and a potential, acute forensic investigation. Even though the local scenario prevails malnourished, forensic dentistry plays a pivotal and a significant role in modern approaches to Disaster Victim Identification (DVI) and other medico-legal investigations.

It must be sadly discerned that all most all unidentified skeletal remains are dismissed "unidentified" in Sri Lanka. Further, virtually all forensic dental cases are handled by the JMOs who have no exposure to dentistry. Albeit it is envisaged theoretically that JMOs are expected to obtain an opinion from the local dental surgeon, it does not occur as expected, due to numerous dependable logistical and other variables in pragmatic grass root circumstances. This issue further deteriorates when a forensic dental opinion is provided by a non dental practitioner or a medico-legally untrained hospital dental surgeon. The courts eventually not perusing this

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reality, rely on the report submitted by the medical expert at most times, a non specialist medical officer/District medical officer. Unveiling "forensic dental specialty" in Sri Lanka will not only shed light in these gaps and rather obscure endeavors but also undoubtedly will provide necessary reforms in order to improve quality in both forensic investigations and justice administration.

Dental trauma analysis from a medico-legal standpoint and bite marks studies play a major role in crime investigation. It is surprising to note, up to date, that there has been no bite mark reported in Sri Lanka with its medico legal analysis in a criminal case. It does not connote that the Sri Lankan victims were never "bit" during crimes! This merely signifies that bite marks were undetected, misdiagnosed as mere abrasions/ other imprint abrasions or were not delivered much attention due to unacceptable reasons. Furthermore, abundant dental evulsions due to blunt or sharp trauma with a history of severe mobility and gross loss of periodontal attachment are being reported as "grievous injuries". These results the suspect to rigorous imprisonment, which could have been obviated if due forensic dental expertise were secured. Such approaches evade justice and warrant inequality. Sri Lankan courts are presumably unaware of dental/medical advancements unless they are reported by the attending expert witness. Such gaps hinder due process of administration of justice. Therefore it is necessary that dental cases with a significant relevance and medico-legal involvement are seen and reviewed by a specialist in forensic dentistry.

Not many are aware of the context, nature, application, depth and scope of forensic dentistry. Forensic Odontology (also known as Forensic Dentistry or Legal Dentistry), is a well established discipline of on its own stature. This art and science primarily involves, the proper examination, handling, interpreting and presentation of dental and oral evidence in a court of law, in the interests of justice. General Dental surgeons, specialist dental surgeons in other domains of dentistry and forensic pathologists and Judicial Medical Officers (JMO) do not possess special knowledge and skills in forensic density. Forensic dentistry is a subspecialty in dentistry on its own merits. Hence, the idea of establishing forensic dentistry in Sri Lanka has been moving from one table to the other wondering whom to entrust the responsibility.

Many universities around the world but Sri Lanka conduct formal post graduate courses, training and research to upgrade knowledge, skills and relevant attitudes in forensic dentistry to be in par with developing other medical sciences. Such a development in the discipline will undoubtedly improve quality and approaches in justice administration. Professional bodies and specialists boards for example American Board of Forensic Odontology and British Association for Forensic Odontology tend to maintain quality, professional standards and guidelines for practitioners in their respective states.

Unfortunately in Sri Lanka even in the 21st century, this discipline does not seem to be in existence. However, there is an increasingly growing demand and interest among dental practitioners, students and others in Sri Lanka.

Without any insight or training in this field, some professionals tend to make negative assertions that have no academic underpinning or argument. It is effortless and easy for someone who do not comprehend the nature, scope, depth and capacity in this subspecialty to hallucinate strong but negative skeptics. Culturally Sri Lanka does not warrant changes to the existing system, no matter how hard or bad it is. Ironically development of any dental discipline has faced challenges in the history. However, dental profession has had courage and motivation to face obstacles.

It appears that forensic dentistry gains gradual recognition in Sri Lanka. Globalization, internet, cable television stories, detective films, foreign

exposure, literature, recent disasters, and advocacy by forensic practitioners are significant contributors for this development.

Forensic dentistry was taught by a visiting JMO/ Medical officer under Ethics and Jurisprudence in the Sri Lankan traditional dental curriculum. Ethics and Jurisprudence often had a stepmothers' treatment in the dental curriculum as there was no academic staff trained in particular, to handle the subject. It was affixed in the Prosthetic Dentistry curriculum to which none could find a relationship other than to the fact that late Prof. Bambaradeniya from the prosthetic department used to coordinate the ethics and jurisprudence component at the time. Ethics and jurisprudence was continued to be a part of prosthetic dentistry examination for decades after the demise of Prof. Bambaradeniya.

Fortunately with the introduction of revisions to the dental curriculum, forensic dentistry became a compulsory module in oral pathology. Faculty board approval, for the contents in forensic dentistry for the new dental curriculum has already been granted. Although this can be viewed as an advancement of the discipline of forensic dentistry, it must be noted that only a limited theoretical dimensions of the subject could be imparted during the allocated short time table slots without any skill development in the learner. In contrast, the medical curriculum in Sri Lanka teaches only a further limited introduction to this subject within the forensic medicine curriculum.The medical fraternity expects the dental practitioners to handle forensic dental work. Ironically none of the post graduate dental or medical specialist programs have a detailed study of this discipline. Consequently, the domain of forensic dentistry is neglected totally within the Sri Lankan dental and medical education paradigm.

There were remarkable achievements in the field of forensic dentistry recently. As innovations, the ministry of health introduced two new cadre positions in forensic dentistry and recruited two dental officers. This is a significant move forward in terms of the implementation and applying forensic dental service function in Sri Lanka. Giving due consideration for the need for post graduate forensic odontology training in Sri Lanka, Boards of Study in Dental Surgery and Forensic Medicine at the PGIM, recommended formulating a special steering committee to study and propose a Post Graduate training program leading to board certification. On the recommendation of the two boards a special committee was appointed by the Director / PGIM. This development can be seen as a milestone in domestic forensic dental education.

Having perused all most all international post graduate training programs in forensic dentistry a unique proposal to establish a post graduate forensic dentistry training program leading to MSc and MD (Forensic Dentistry) and board certification through the Post Graduate Institute of Medicine (PGIM), University of Colombo, was designed, considering all relevant aspects and expectations of stake holders. This proposal was approved by the special committee and the board of study in dental surgery at PGIM.

It is essential to establish the forensic dental specialty in Sri Lanka. Dental surgeons in the ministry of health, military service, universities and private sector are potential candidates to follow these programs of study. Forensic Dental Specialists with adequate training should be board certified using the proposed PGIM program. These forensic dental specialists should be able to work independently and confidently in the ministry of health, military, university or at any other agency in a specialist capacity.

Parallel to the PGIM training programs in forensic dentistry, author of this article designed another master degree program in forensic dentistry to be conducted through the faculty of dental sciences which the faculty board of the faculty of dental sciences approved unanimously. This

study program will mainly cater to international candidates and interested local state or private candidates.

The PGIM training program leading to MSc and MD will play a main role in the ministry of health and in military service. The ministry of health and military service will only recognize a PGIM qualification for their promotions. Therefore, the need for a program via PGIM becomes very relevant. One should not advocate a training program only at a diploma level or at masters level and arrest from there, because with these qualifications (diploma or masters level) candidates will be frustrated without promotions or specialist recognitions in the Health Ministry, military or courts. This program should inculcate motivation, interest and professional development of the potential candidate. Therefore this program should necessarily lead to a MD. Educational programs should be designed to suit the professional culture, professional politics and the structural architecture in a country contextualizing the needs and potentiality.

It is the view of the author to implement these programs at least in the year 2013. Until then, necessary groundwork, logistics and development of training centers can be attended to. It may be a sound idea to have at least one specialist forensic dental surgeon in each province in Sri Lanka with at least two specialists posted in Colombo. At least one specialist forensic dental officer each should be in the three armed forces, police and university. After these specialists are trained, the PGIM could scrutinize the need for increasing further trainees or curriculum revisions. As a minimum standard, at least one specialist forensic dental officer is recommended per province initially. With this model, it is anticipated that this neglected dental discipline will flourish, create more dental cadres and job opportunities in the country. In addition improvements in quality in forensic investigations and justice administration are spontaneous. Inevitably, research and new knowledge in the field of forensic dentistry would be facilitated in Sri Lanka as these proposed study programs have an essential research component. Strong commitment and encouragements by the dental profession and medico legal community would be blessings for the development of this discipline which is a national need.

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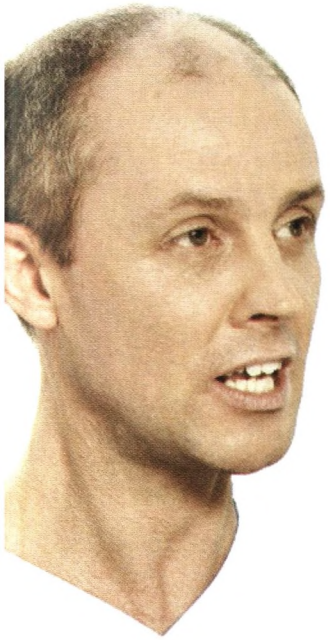

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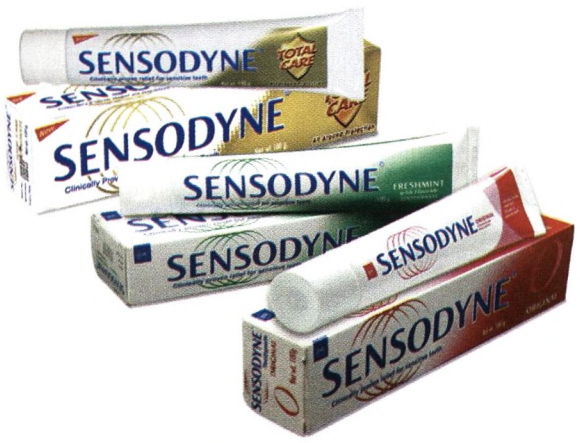

SENSODYNE

DAILY PROTECTION FROM THE PAIN OF SENSITIVE TEETH

1. Addy M. Int Dental J 2002; **52** : 367-75

Research Article I

Sri Lanka Dental Journal 2010; 40(02-03) 54-60

Prevalence of *Candida* in saliva and root canals of teeth associated with apical periodontitis

N.S. Soysa, H.N. Fernando, Neil Alles, R.L. Wijeyeweera, K.A .Wettasinghe, G.J. Panagoda

Abstract

Objectives: *Candida* is the commonest fungal pathogen isolated from the oral cavity. The occurrence of *Candida* in root canals has been reported by many researchers. The control and elimination of yeasts is very important during endodontic treatment because of their presence in dentinal tubules and root canals may lead to periapical diseases. Colonization of root canals by yeasts derived from oral cavity has not been established yet. Hence, the aim of this study was to determine the relative prevalence of *Candida* in the oral cavity and root canals associated with apical periodontitis in a group of patients who sought endodontic treatment from the Dental Hospital Peradeniya, Sri Lanka.

Material and methods: Experimental samples from root canals of the teeth associated with apical periodontitis and samples from oral rinses of the respective patients were obtained from 35 adults and 8 children. The medical history including antibiotic therapy and clinical/ radiographic data on teeth were recorded. The samples were then cultured on Sabouraud dextrose agar and *Candida* growth was identified by growth characteristics and colony morphology.

Results: *Candida* isolates were recovered from 16 oral rinse samples and 4 root canal samples in adults. The relative frequency of *Candida* prevalence in root canals was 11.42%. Presence of *Candida* in root canals was not significantly associated with their presence in saliva.

Conclusion: The prevalence of *Candida* in the oral cavity in the present group was 45.7% while prevalence of *Candida* in root canals associated with periapical periodontitis was much less. While the role of *Candida* in root canal infection and the effectiveness of routine clinical endodontic therapy in the presence of canal candidosis remain

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for further investigation, the role of *Candida* in the initiation and perpetuation of periapical disease remains to be determined.

Key words: *candida*, root canal, apical periodontitis

Introduction

Candida is by far the commonest yeast isolated from the oral cavity of both the healthy and the medically compromised adults.[1,2] Their presence usually does not result in disease unless there are associated local or systemic predisposing factors, such as, low salivary flow, poor oral hygiene, cigarette smoking, ill fitting dentures, diabetes, malignancy, cytotoxic therapy, antimicrobials or HIV infection[3,4,5,6,7] Oral yeasts have been isolated from dental plaque, dental caries, subgingival flora, as well as root canals.[8,9,10,11] The presence of *Candida* in root canals varies from 0.6%-10% in untreated cases and 3.7%-10% in treatment resistant cases.[12,13,14,15,] Using a variety of cultivation techniques a higher prevalence of yeasts ranging from 7% (in treated teeth) and 55% (in untreated teeth) have been demonstrated.[9,16] The majority of the yeasts recovered were *Candida* species and *Candida albicans* being the most prevalent followed by *Candida dubliniensis.*[16,17] Yeasts in the root canal space are reported to be served as a reservoir for the dissemination to the periphery via the blood stream.[18,19] Furthermore, they also cause periapical granulomas and systemic urticaria.[18] Hence, re-evaluating the presence of *Candida* in the oral cavity is utmost important from an endodontic point of view. However, factors influencing the colonization of root canals by yeasts derived from the oral environment have not been specifically investigated.[16] A number of predisposeing factors are appeared to be responsible for this process. For instance the use of intra-canal medicaments, local and systemic antibiotics and previous root canal treatments cause a reduction of specific groups of bacteria in the root canals providing the yeasts an opportunity to grow and predominate in a low nutrient environment.[15,20,21,22,23,24] Poor asepsis during root canal treatment also allows colonization of microorganisms in root canals. *C.albicans* is likely to grow on dentinal surfaces even in the absence of oral tissue fluids rich in supporting media.[24] They penetrate into dentinal tubules (dentinophilic) by growth patterns such as by forming either blastospores or hyphae. It is also well known that *C.albicans* can digest dentinal collagen, resulting in release of minerals, including calcium, from the crystal phase after the removal of the organic content of dentine.[25] In the presence of a mixed endodontic infection, *Candida* may interact with other microbes and microbial products. The co-aggregation reaction with bacteria can play a predominant role in candidal colonization of mucosal and hard tissue surfaces in the oral cavity by forming a complex biofilm.[26] Cell surface properties of *C.albicans* are rapidly modified in response to different growth conditions or environmental changes, and phenotypic switching may occur in a particular ecological niche.[27] Therefore, the dentinophilic properties and pathogenicity of *C.albicans* may be increased. In addition Nair *et al* (1990) observed budding yeasts cells of *Candida* in peri-radicular lesions refractory to the endodontic treatment.[28] Hence, the objective of this study was to determine the prevalence of *Candida* in root canals in patients with periapical periodontitis and to correlate it to the prevalence of *Candida* in the oral cavity.

Material and methods

Patient selection

Thirty five adult patients and 9 children who attended the Dental Hospital Peradeniya, Sri Lanka for non-surgical root canal treatments were included in this study. Previously untreated teeth associated with radiographic evidence of periapical pathology were selected for the investigation. Patients who were on drugs such as antibiotics, steroids or having diabetes and patients who wore dentures were excluded from the study. Informed consent was obtained from all patients before enrollment. The study design

and the procedure were consistent with the principles of the declaration of Helsinki.

Clinical data

The patient's medical history was obtained and none had a history of prolonged antibiotic or steroid therapy, anaemia, diabetes or any predisposing condition known to promote a *Candida* carrier state. The restoration margins where present were assessed using a dental probe to establish the presence or absence of restoration leakage. The presence of caries, fractured restorations or probe-able restoration margins was used as positive indicators of open canals. The nature of the canal content and the periodontal condition of the tooth were noted.

Mouth wash and root canal sampling

Oral yeast colonization was assessed by an oral rinse technique described by Samaranayake *et al* (1986).[29] Each subject was asked to rinse the mouth thoroughly, with 10 ml of sterile phosphate-buffered saline (PBS) for 1 min and it was collected into a sterile container before sampling the root canals. The target teeth were scaled, polished and isolated with a rubber dam. The sampling field was decontaminated as described else where with some modifications by scrubbing with 30% hydrogen peroxide (v/v) (Sigma, UK).[17] The decontamination procedure was repeated following access cavity preparation. Root canals were negotiated to their full length. The canal contents were soaked up using 2 sterile paper points, each being left in the canal for at least 1 minute and immediately transferred aseptically to the microbiology laboratory in PBS for microbiological analysis.

Laboratory processing of samples

The saliva and root canal samples were centrifuged and resuspended in 1 ml of new PBS and vortexed. An aliquot of mixture was inoculated into Sabouraud dextrose agar plates and incubated overnight at 37°C. Preliminary identification of yeasts from bacterial cell colonies was based on the unique growth characteristics and colony morphology of *Candida* grown on Sabouraud dextrose agar and CHROM agar.

Statistical analysis

Forty two cases were selected for statistical analysis. Statistical analysis were made with a computer program SPSS. Fisher's exact test was used to find the association of the presence of *Candida* in saliva and the presence of *Candida* in root canals.

Results

Table 1 shows the patients' data. Out of 35 adults who participated in this study 15 (15/35; 42.8%) were males while 20 (20/35; 57%) were females. Out of 35 root canals investigated in adults, 20 had some communication with oral cavity through caries, restoration leakages and fractures while 15 had no communication with oral cavity, which were considered as closed canals. Total of 35 oral rinses were taken from adults. *Candida* was more frequently recovered from oral cavities (16/35 or 45.7%) than root canals (4/35 or 11.42%) in adults (Table 2). All 4 *Candida* positive root canals in patients had no previous root treatment. All 4 patients with *Candida* present in the root canals were healthy. The root canal system of 4 teeth had some communication with the oral cavity, 3 canals via carious cavities and the other through fracture. *Candida* isolated from root canals was further cultured on CHROM agar to identify the species. *C.albicans* and *C.tropicalis* were the main *Candida* species isolated form root canals (Table 3). The association between the presence of *Candida* in root canals and mouth rinse was not significant (p=0.94). In the case of children, *Candida* was isolated only from mouth rinses and all were identified as *C.albicans*.

Discussion

The sample collection (imprint culture, swab, mouth rinse, and saliva) and growth techniques appear to influence the recovery of *Candida* as various techniques give different isolation rates. The oral rinse technique used in this study is one of the most sensitive methods for assessing

candidal carriage in individuals as it allows the study of intraoral distribution of *Candida*.[29] Most of the studies with non-selective blood agar have reported a lower prevalence of yeasts as compared to the present study (11.4%) and others (10%) who used Sabouraud dextrose agar.[17] Studying the prevalence of *Candida* in the oral cavity, Najzar-Fleger *et al* (1992) reported *Candida* in 55% of root canals, while Waltimo *et al* (1997) isolated a total of 48 fungal species from root canals of teeth with apical periodontitis.[9,16] The prevalence of yeasts in untreated cases in this study was 11.4% in contrast to 1.9%, 5.7%, and 26% reported in previous studies.[17,21,22] All 4 patients with *Candida* present in root canals were healthy and had no history of immunodeficiency or antibiotic treatment within the previous 12 months. The root canal system of 4 teeth had some communication with the oral cavity via caries or fracture. Though yeasts occur relatively infrequently in root canals, their presence in root canals was significantly associated with their presence in saliva.[17] In the present study, we found different species of *Candida* in root canals, namely *C.albicans* and *C.tropicalis*. suggesting that *C.albicans* does not seem to be the dominant species in the root canals. However, this cannot be conclusive due to the small sample size. In this study we could not find an association between the presence of *Candida* in root canals and mouth rinses.

A fundamental aim of root canal treatment is to eliminate microbial infection of the root canal system. This cannot be accomplished by chemo-mechanical preparation of the root canal alone, because microorganisms are generally present in the root canal and the dentinal tubules after root canal instrumentataion. Several studies have evaluated the effect of endodontic irrigants against *C.albicans*. For instance Sen *et al* (1987) evaluated the antifungal properties of chlorhexidine and sodium hypochlorite (NaOCl) and found that *C.albicans* is more resistant in the presence of the smear layer.[24] NaOCl alone show antifungal activity in the absence of a smear layer after 30 minutes. Hence, antimicrobial effectiveness of irrigating solutions should be evaluated in patients predisposed to oral candidiosis.[24]

Conclusion

The prevalence of *Candida* in the oral cavity in the present group was 45.7% while prevalence of *Candida* in root canals associated with periapical periodontitis was 11.42%. The role of *Candida* in root canal infection and the effectiveness of routine clinical endodontic therapy in the presence of canal candidosis remain for further investigation.

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We thank all the patients who participated in this study, and all the nursing staff and technical staff at the Department of Restorative Dentistry and Division of Paedodontics, Dental Hospital, Faculty of Dental sciences. This work was supported by a research grant of the University of Peradeniya (rg/2002/43/d) to NS Soysa.

Table 1. Characteristics of the sample and relevant clinical information

Adults	Children
n=35	n=8
Age range	Age range
16-52 Mean (30)	9-15 Mean (11)
Sex	Sex
Male n= 15	Male n= 5
Female n= 20	Female n= 3
Root canal status	Root canal status
Open n= 20	Open n= 5
Closed n=15	Closed n=3
Open canals n=20	Open canals n=5
Caries n=9	Caries n=1
Restoration leakage n=7	Restoration leakage n=0
Fractures n= 4	Fractures n= 4

Table 2. *Candida* recovered from oral rinses and root canals.

	Total number of root canal			
	Open canal (20)	Closed canal (15)	Open canal (5)	Closed canal (3)
Mouth rinse	10	6	2	1
Root canal	4	*	*	*

* (denotes no *Candida* isolation.)

Table 3. *Candida* species in root canals and mouth washes from adults.

Candida species	Mouth rinse	Root canal
C.albicans	13	2
C.tropicals	*	2
C.glabrata	3	*

* (denotes no *Candida* isolation)

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Research Article II

Sri Lanka Dental Journal 2010; 40(02-03) 61-69

Ultimate Tensile Strength of the dentine-enamel junction region of normal teeth and teeth affected by dental fluorosis

K.A. Wettasinghe, T. Nikaido, P.G.K. Waidyasekera, D.D.S. Weerasinghe, J. Tagami

Abstract

Objectives: The objective of the present study was to compare the ultimate tensile strength (UTS) of the dentine-enamel junction (DEJ) region of normal teeth and teeth affected by dental fluorosis.

Material and methods: The study sample consisted of upper first and second molars extracted from patients living in areas of endemic fluorosis. They were classified according to the modified Thylstrup-Fejerskov index (TFI) into normal (N, TFI 0), mild fluorosis (ML, TFI 1-3), moderate fluorosis (MD, TFI 4-6) and severe fluorosis (SV, TFI 7-9). Three teeth from each group were sectioned vertically into two halves in the mesio-distal direction after sectioning the roots. A 6 mm thick composite resin block was bonded to both dentine and enamel sides to facilitate handling and testing. The resulting blocks were serially sectioned into several 0.7 mm thick slices, perpendicular to the DEJ. Each slice was then trimmed to an hour glass shaped specimen with a superfine diamond bur, in order to reduce the DEJ region to approximately 1 mm in width. Ten specimens from each group were tested for ultimate tensile strength (UTS) at a crosshead speed of 1 mm/min. The fracture mode was determined by examining the fractured specimens under a digital microscope at 50X magnification. Data were analyzed with one-way ANOVA, Tukey post hoc comparison test and the Pearson Chi-Square Test.

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Results: The UTS mean values (n=10) were [MPa(SD)]: N-45.9(13.7), ML-38.6(13.7), MD-33.0(6.5) and SV-28.0(5.5). There were statistically significant differences between N and MD ($p<0.01$), N and SV ($p<0.001$), and ML and SV ($p=0.032$) groups, respectively. Results of the present study show a definite and statistically significant reduction in the ultimate tensile strength of the dentine-enamel junction region with increasing severity of dental fluorosis.

Conclusion: There was an inverse relationship between the ultimate tensile strength of the dentine-enamel junction region and severity of dental fluorosis.

Key words: Dental fluorosis, dentine-enamel junction, ultimate tensile strength

Introduction

Dental fluorosis is a tooth malformation caused by chronic ingestion of fluoride during tooth development.[1,2] The prevalence of dental fluorosis (DF) ranges between 7.7% and 80.9% in the areas with fluoridated water and between 2.9 and 42% in the areas without water fluoridation.[3,4,5] In the fluorosed teeth, the surface layer of enamel is highly mineralized. It is composed of a mixture of large, flattened, hexagonal, crystals and extremely small, irregularly shaped crystals. The hypomineralized area, extending inwards from the surface layer, contains sparsely arranged, flattened and hexagonal crystals with either perforated centers or defects extending from the perimeter.[6] However, no difference has been demonstrated in crystallite size between normal and fluorotic dentine.[7] On the other hand, areas of interglobular dentine has been observed in dentine following high fluoride administration.[8,9]

The dentine-enamel junction (DEJ) is a complicated structure in the human tooth which plays a critical role in maintaining the unity between the hard, brittle enamel and softer, tougher dentine.[10] In maturity it appears to be critical to the biomechanical integrity of the tooth, because it joins these two structurally diverse calcified tissues.[11] Despite being exposed to millions of cycles of masticatory loading, even parafunctional and impact loading over a human life time, the enamel is hardly seen to delaminate from dentine.[12]

With the structural differences in enamel and dentine in fluorotic teeth, which frequently exhibit chipping of enamel as a clinical finding, a difference in the ultimate tensile strength (UTS) of the DEJ region can be expected. Recent studies have investigated etching and bonding properties of fluorosed enamel and caries susceptibility of human fluorosed enamel and dentine.[13,14,15,16,17] Previous studies have also shown the ultimate tensile strength of the DEJ region in normal human teeth.[18,19] However, no previous studies have investigated UTS of the DEJ region in teeth exhibiting fluorosis. Therefore, the purpose of this study was to compare the UTS of the DEJ region of normal and fluorotic teeth of different severity. The null hypothesis was that severity of fluorosis did not have any significant effect on the UTS of the DEJ region.

Material and methods

2.1 Preparation of specimens

Human upper first and second molars from subjects between the age of 35 and 54 years living in endemic areas for fluorosis in Sri Lanka were used for this study. Teeth were caries free and were extracted for periodontal reasons. Before extraction, the patients were told that their teeth would be used for research purposes and their informed consent was obtained. Teeth were cleaned in tap water immediately following extraction, wrapped in moist cotton wool and stored individually in containers at 4° C in a refrigerator, with the details of date of extraction and age of the patient. The teeth were used within 4 months of extraction for the present study. The teeth were cleaned and the buccal and palatal sides were polished to remove any stains with non fluoridated prophylaxis paste (Pressage,

Shofu Inc, Kyoto, Japan) with a bristle brush on a slow speed hand piece. They were dried quickly and the severity of dental fluorosis was recorded according to the modified Thylstrup-Fejerskov Index (TFI).[20] This was done by two investigators independently. The inter examiner variation gave a Cohen's *k* statistic of 0.95.[21] The teeth used in the present study were classified into 4 categories. TFI 0; normal (N), TFI 1-3; mildly fluorosed (ML), TFI 4-6; moderately fluorosed (MD), TFI 7-9; severely fluorosed (SV) with 3 teeth in each category. Having assessed the severity of dental fluorosis, the teeth were sectioned using a slow rotating diamond disc (Isomet, Buehler, Lake Bluff, IL, USA) under water lavage. First the roots were sectioned 2 mm below the cemento-enamel junction and the crown was sectioned into two equal buccal and palatal halves by making a vertical cut in the mesio distal direction (Fig. 1). Two 6 mm thick resin composite (Clearfil AP-X, Shade A3, Kuraray Medical, Tokyo, Japan) blocks were bonded to the enamel and dentine seperatly using an adhesive system as shown in the figure 1. (Clearfil SE Bond, Kuraray Medical, Tokyo, Japan). Composite resin was cured in three, 2 mm thick increments to form an extension to facilitate further slicing and testing. The mid portion of the resulting block was then sectioned into 0.7 mm slices with a slow rotating diamond disc horizontally, perpendicular to the interface of dentine enamel junction (DEJ). The slices were trimmed to an hour glass shape with a superfine diamond bur (ISO #545, Shofu Inc, Kyoto, Japan) under air-water irrigation in an air rotor hand piece in order to reduce the width of the DEJ area to approximately 1 mm, resulting in a cross sectional area of less than 1 mm^2. Ten specimens from each group were used for microtensile bond testing.

2.2 Microtensile bond strength

Each specimen was fixed to the flat grips of the universal testing device (EZ-test, Shimadzu, Kyoto, Japan) using cyanoacrylate glue (Zapit, Dental Vent. Am., Corona, CA, USA) with care to avoid spreading of glue to the test region of the specimen. Micro tensile testing was carried out at a crosshead speed of 1 mm/min until the specimens fractured. After fracture, the specimen was carefully removed from the testing apparatus with a scalpel blade and the cross sectional area at the site of fracture was measured with a digital caliper (CD-S15C, Mitsutoyo, Kawasaki, Japan) to the nearest 0.01 mm.

2.3 Fracture mode

The fracture mode of the specimens was determined following examination under a digital microscope (VHX-500, Keyence, Osaka, Japan) under 50X magnification. The fracture mode was classified into 3 types; cohesive failure in enamel only, in both enamel and dentine through the DEJ and in dentine only.

2.3 Statistical analysis

The UTS was calculated by dividing the imposed force(MPa) at the time of fracture by the area (mm^2). Statistical differences were examined using one-way ANOVA and Tukey LSD post hoc comparison test. The Pearson Chi-Square Test was used to explore the association between the failure modes and type of fluorosis. The data were analyzed by using SPSS for windows Version 11 ($p = 0.05$).

Results

3.1 Ultimate micro-tensile bond strength

The mean UTS of the DEJ region of normal and teeth with fluorosis are given in Table 1. There was a trend for the UTS to reduce with increasing severity of fluorosis. One-way ANOVA indicated a statistically significant difference among different sample groups ($f=5.226$). The Tukey LSD post hoc comparison showed statistically significant differences in UTS between normal teeth (N) and teeth with moderate fluorosis (MD) ($p< 0.01$), between normal (N) and teeth with severe fluorosis (SV) ($p<0.001$), and also between teeth with mild (ML) and severe fluorosis (SV) ($p= 0.032$). Results of the present study show a definite and statistically significant reduction in the UTS of the DEJ region with increasing severity of dental fluorosis.

3.2 Fracture mode

The fracture modes of the specimens are given in Table 2. Pearson Chi-Square analysis revealed no significant association between mode of failure with the type of fluorosis (X^2=3.314, p=0.768). The photomicrographs of the specimens with typical fracture modes are presented in Figure 2. A majority of the specimens had fractured through enamel. Only three specimens indicated fracture through dentine. Two specimens fractured through dentine/enamel.

Discussion

In the present study, recording of dental fluorosis was done according to the modified Thylstrup and Fejerskov index, which is based on the clinical changes and coincides with pathological changes in fluorosed teeth.[20] This is the index of choice for assessing the severity of dental fluorosis.[22]

Since its introduction by Sano in 1994, microtensile technique has proven to be extremely useful to measure bond strengths as well as to determine the ultimate tensile strength of both materials and tooth structure.[18,23,29] The major advantages of the microtensile technique include improved stress distribution during testing and the ability to perform the test in very small specimens.[24] It is more labour-intensive than conventional testing but holds great potential for providing insight into the strength of adhesion of restorative materials to clinically relevant sites and substrates.[30]

Previous studies have attempted to measure the UTS of the normal DEJ with the microtensile method.[18,19] The UTS values of the DEJ in the normal teeth were approximately 45-50 MPa, which is similar to our results for the normal teeth. However, this study showed a definite reduction in the UTS of the DEJ region with increasing severity of dental fluorosis. In addition, almost all the specimens fractured within the enamel adjacent to the DEJ in both normal and fluorosed teeth. Therefore, the reduction in the UTS of the enamel adjacent to the DEJ can only be explained by the changes in the microstructure of fluorosed enamel.

Generally, hardness of enamel is attributed to it's high mineral content, and the brittle property of enamel is due to it's high elastic modulus and low tensile strength.[31,34] Therefore, enamel is easier to fracture than dentine when loaded in a direction perpendicular to the orientation of prisms.[28] On the other hand, the collagen phase of intertubular dentine contributes to a lower modulus of elasticity than enamel, while the lower mineral content is associated with a decrease in dentine micro hardness compared to enamel.[19] These factors clearly elucidate the tendency of enamel to be susceptible for fractures rather than dentine under a tensile force. In addition, collagen fibrils of DEJ complex play a critical role in resisting crack propagation from enamel into dentine.[11,12]

In fluorosed teeth, the subsurface, hypomineralized region of enamel is known to contain sparsely arranged hexagonal crystals.[6] The pores in the subsurface enamel are occupied by water as well as enamel secretory proteins which are retained due to the effect of the excessive fluoride level on ameloblasts.[35] The extent and severity of the subsurface hypomineralization is in keeping with the TFI scores. Thus in teeth with highest TFI scores, the width of the lesion extends almost to the DEJ and the pore volume of the subsurface area exceeds 25%.[35] Residual enamel matrix, water and microstructural organization of minerals affect the micromechanical properties of the enamel. Recent literature shows data regarding the mechanical properties of DEJ that has been associated with variations of mineralization and the associated collagen to mineral ratio within the DEJ compared to dentine and enamel.[36] Therefore, the structural aberrations seen in fluorosed enamel with a low Ca level may affect the integrity of the enamel of the DEJ region of fluorosed teeth, thus making it less resistant to tensile force and leading to immature fractures.

Conclusion

The UTS of the DEJ region was found to be approximately 45-50 MPa for normal teeth with

fractures occurring on the adjacent enamel. The UTS of the DEJ region was less in fluorosed teeth and it had an inverse relationship with severity of fluorosis. Further studies are necessary to understand the mechanical properties of dental structures of fluorosed teeth.

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Table 1. The mean UTS of the DEJ region of normal and teeth with fluorosis (MPa±SD).

	UTS (MPa)
Normal (N)	45.9 – 13.7[*, **]
Mild (ML)	38.6 – 13.7[***]
Moderate (MD)	33.0 – 6.5[*]
Severe (SV)	28.0 – 5.5[**, ***]

n=10 for each group. Same superscript letters show statistically significant differences.

Table 2. The fracture modes of specimens.

Tooth type	Dentine	Dentine/enamel	Enamel
Normal	1	-	9
Mild fluorosis	1	1	8
Moderate fluorosis	1	1	8
Severe fluorosis	-	-	10

n=10 for each group.

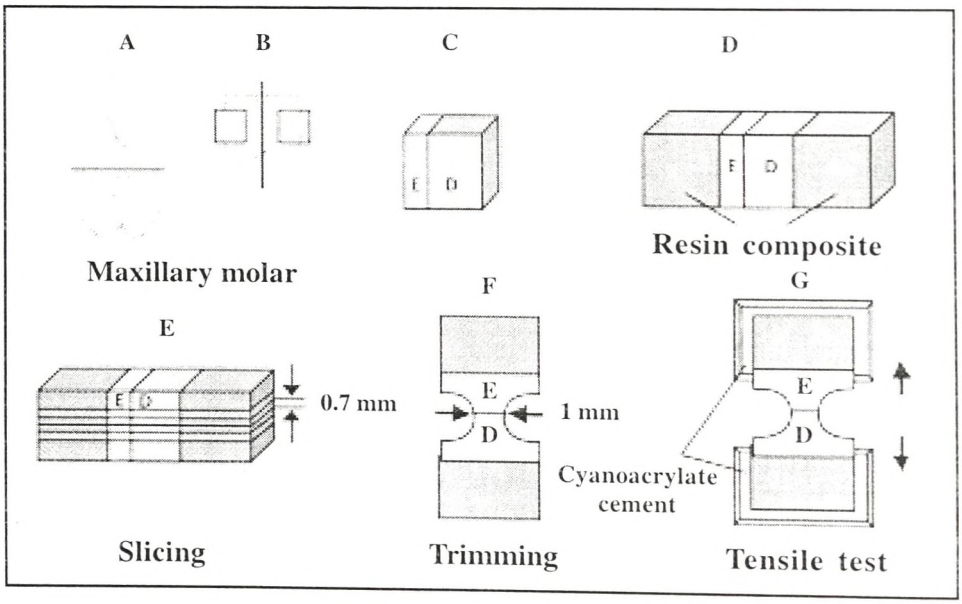

Figure 1. Schematic representation of specimen preparation. A: Sectioning of roots. B: Splitting of tooth mesio-distally to two halves. C: Block containing enamel and dentine sides. D: Resin composite bonded to enamel and dentine sides E: Slicing of block at 0.7 mm thickness. F: Slice trimmed with superfine diamond bur to hour glass shape, approximately 1 mm at DEJ region. G: Trimmed specimen fixed to grips of testing device with cyanoacrylate glue.

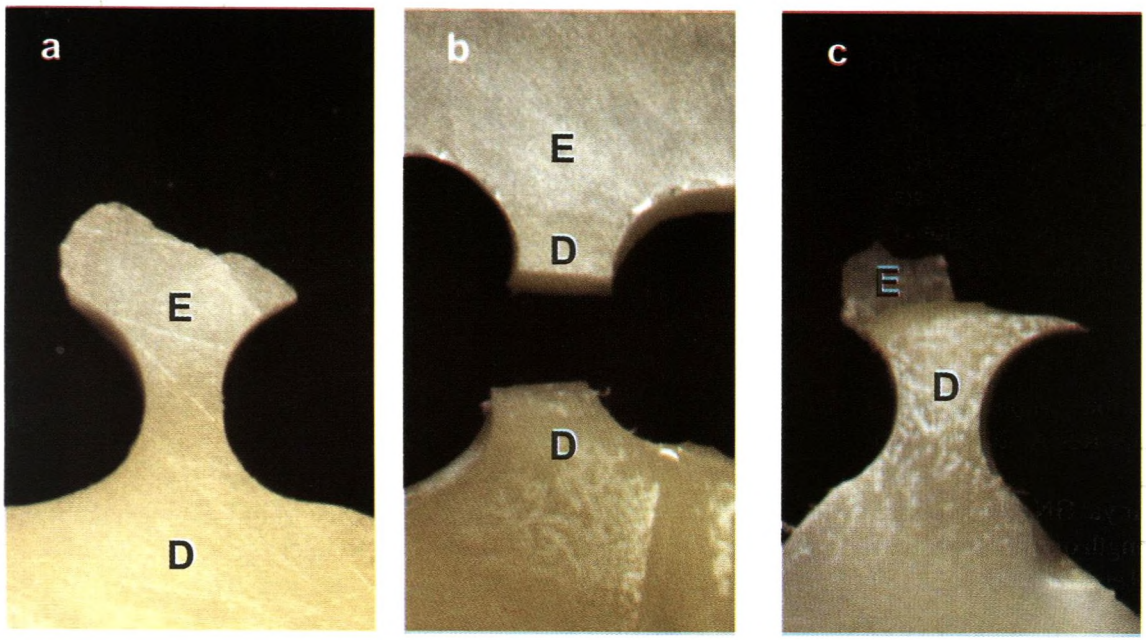

Figure 2.

(a) A specimen where the fracture line passes through enamel
(b) A specimen where the fracture line passes through dentine
(c) A specimen showing the fracture line passing through dentine-enamel junction

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Research Article III

Sri Lanka Dental Journal 2010; 40(02-03) 70-75

Salivary β - Glucuronidase as a marker for chronic periodontitis

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Abstract

Objectives: The objective of the present study was to evaluate the usefulness of salivary β-glucuronidase in predicting periodontal disease severity.

Material and methods: Sample consisted of 100 subjects (50 in the (experimental group and 50 in the control group) of whom 64 were males and 35 were females with the mean age of 35 years, ranging from 28-60 years. Saliva was collected from both groups and assayed for β-glucuronidase activity. The results were statistically analyzed using Pearson's parametric correlation analysis.

Results: The mean β-glucuronidase level was 0.25 for the experimental group and 0.14 for the control group. The 'p' value was significant (<0.01), which implied that there was an increase in the level of β-glucuronidase in the experimental group compared to that of the control group.

Conclusion: The results of the present study indicate that salivary β-glucuronidase is a dependable marker of periodontal disease. This marker can be use to screen for periodontal diseases and as a means of monitoring the response to therapy.

Introduction

The diagnosis of active phases of periodontal disease and the identification of patients at risk for the active disease is a challenge for both clinical investigators and clinicians. In general, clinical parameters including probing depth, attachment level, bleeding on probing, plaque index and radiographic loss of alveolar bone are used to assess disease severity. Further, a complete periodontal examination is time consuming and requires highly trained personnel. β-glucuronidase in GCF which as been proposed as a promising, non-invasive means of determining the tissue changes in the periodontium. However, collection of GCF insertion of filter paper into the crevice may cause tissue damage and alter crevicular fluid flow. It has long been realized that a rapid and simple diagnostic test can provide a reliable evaluation of periodontal disease and identify patients at risk for active disease which would be of value to both clinicians and patients.

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The usage of saliva for periodontal diagnosis has been the subject of interest since many years.[1] Enzymes in saliva have been studied as markers of periodontal disease.[2] Saliva is a fluid that can be collected easily and contains locally and systemically derived markers of periodontal disease. Salivary enzymes are derived from cells in the salivary glands, oral microorganisms, polymorphonuclear leucocytes (PMNLs), macrophages entering the gingival sulcus and epithelial cells.

PMNLs are a critical component of local inflammatory response in periodontal disease. PMNLs comprise 90% of leucocytes present in the gingival crevice and these cells reflect the degree of tissue inflammation. β-glucuronidase is an enzyme which actively involves in the degradation of proteoglycans and the ground substance. This enzyme is an indicator of PMNL influx into the gingival sulcus. Studies have demonstrated that β-glucuronidase activity in GCF correlated well with clinical parameters like pocket depth, attachment loss and bleeding on probing.[3] The present study was conducted to assess the relationship between β-glucuronidase activity in whole saliva and severity of periodontal disease.

Material and methods

Subject selection

The study was conducted between February and July 2009. The subjects for the study were selected from the patients visited the Department of Periodontics, Mamata Dental College and Hospital, Khammam within the given period. A total of 100 subjects (65 males and 35 females) with a mean age of 35 years, ranging from 28-60 years, who fulfilled the selection criteria given below were recruited for the study. Ethical committee clearance was obtained and consent was taken from all patients who agreed to take part in the study. The subjects were divided into two equal groups – an experimental group and a control group.

Criteria for group division

Experimental Group (n=50)

Total of 50 subjects with the following criteria were included under the experimental group - clinical attachment loss (CAL) more than 6mm; pocket depth 5 to 8 mm; bleeding on probing.

Control Group (n=50)

Total of 50 subjects with the following criteria were included under the control group - pinkish and firm marginal gingiva, visually free of inflammation and no bleeding on probing; sulcus depth 3 mm or less.

Subjects who had a minimum of 24 teeth excluding third molars were only included in the study.

Patients with following features were excluded from the study.

1. History of periodontal treatment in the preceding 6 months.
2. History of use of antibiotics, NSAIDs and antihypertensive drugs or other drugs known to affect the periodontium.
3. Smokers.
4. Known systemic diseases.
5. Xerostomia.

A gingival index score was determined for both groups using the Loe and Silness gingival index.[4]

Collection of unstimulated saliva

The patient was seated comfortably in an upright position with the head bent slightly downwards in a dental chair. He/she was asked not to swallow or move the tongue or lips till the collection of saliva was completed. After two minutes, the patient was asked to spit the accumulated saliva into a sterile container.

Biochemical laboratory procedure (β-Glucuronidase Assay)

The unstimulated saliva samples were kept frozen at - 20°C until assayed. Then the saliva was pipetted out from the sterile container by using an automated variable cap pipette and the analysis was performed at 25°C.

For the analysis two tubes were taken, one labeled as 'control' (blank) and the other labeled 'test'. 50µl 0.9% saline control and 100µl of 0.008mM 4-methyl umbelliferone as substrate standard was added to the control (blank) tube and was incubated for 15min at 25°C.

50µl of saliva and 100µl of 0.008mM of 4-methyl umbelliferone as substrate standard was then added to the test tube and was incubated for 15 min at 25°C. The reactions were halted by adding 2ml of 0.2M glycine buffer at a pH of 11.7.

Within 60min after mixing the chemical reagents in both tubes, fluorescence was measured. The absorbents of β-glucuronidase enzyme were recorded in a systronics digital spectrophotometer at 360 nm.

Saliva was used for β-glucuronidase assay, while a complete blood count analysis was performed on the whole blood by a clinical laboratory only in experimental group.

Results

The descriptive statistics was performed to calculate mean and range of the selected parameters (Table 1). The Pearson's parametric correlation analysis was carried out to study the correlation between β-glucuronidase activity and selected parameters (Table 2).

Our data indicates a significant positive relationship between increase in β-glucuronidase activity with increase pocket depth and attachment loss. In addition a significant correlation was found between neutrophil count and β-glucuronidase activity. There was no significant relationship between gingival index and β-glucuronidase activity. Monocytes and lymphocytes were not significantly correlated with β-glucuronidase activity in saliva.

The students 't' test was done for comparison of β-glucuronidase level between the experimental group and control group (Table 3). The model was verified statistically using logistic regression analysis to estimate the probing depth by β-glucuronidase level. This model confirms the test since the standardized coefficient is 0.85. R2=0.730 i.e. 73% of the probing depth variants are explained by the β-glucuronidase level.[5] For e.g. If a persons β-glucuronidase level is 0.257, then PD=2.4375+18.8891(0.257) = 7.28mm, i.e. for a person with β-glucuronidase value of 0.257, we can expect a mean probing depth of 7.28mm.

Discussion

The diagnosis of periodontal disease currently relies primarily upon clinical and radiographic parameters. These parameters are useful for detecting evidence of past disease or confirming periodontal health, but provide only limited information. Although periodontal disease can be site specific, studies have suggested that when monitored longitudinally, a small percentage of patients demonstrate the majority of active sites.[6]

The alternating chronic and episodic nature of periodontal disease, the wide range of disease severity affecting different teeth within the same subject and prolonged requirement for repeated treatments makes it necessary to augment traditional diagnostic aids like clinical examination, probing depth, attachment loss, radiographs and by biochemical assays utilizing biochemical diagnostic markers.

During inflammation, the extracellular release of PMNLs in the host gingiva may contribute significantly to the degradation of the gingival tissues and the pathogenesis of the periodontal disease.

The correlation between β-glucuronidase activity in saliva and periodontal health has been previously investigated. Whole saliva of the adult periodontitis demonstrated the highest enzyme activity, while whole saliva of the healthy controls demonstrated the lowes.[7]

β-glucuronidase is a lysosomal acid hydrolase enzyme which is capable of breaking down connective tissue ground substances. During the inflammatory process β-glucuronidase is released by the degranulation of activated PMNLs and is involved in the degradation of glycosaminoglycans which are the integral part of the connective tissue matrix.

Measurements of salivary β-glucuronidase activity may be useful in excluding healthy patients and referring patients at risk for more thorough periodontal evaluation. Collection of saliva is cost effective, and atraumatic with better patient convenience. Further, repeated sampling is not inconvenient and therefore this can be employed in screening large population groups. Omori has shown that analysis of the enzyme β-glucuronidase in saliva may be a measure of crevicular neutrophil influx for the whole mouth.[8]

The present study showed a significant relationship between increased β-glucuronidase activity and increased mean pocket depth, which was in accordance with the studies conducted in gingival crevicular fluid by Lamster *et al,* (2003) and Kaufman *et al,* (2003).[1] Our study also demonstrated a positive correlation between increase in the attachment loss and increased salivary β-glucuronidase level. This result was similar to the studies conducted in gingival crevicular fluid by Layik *et al*, (2000).[10]

Increase in neutrophil count in experimental group of our study correlated with the result of similar study done by Lamster *et al*, (2003).[3]

The present study exhibited a high level of salivary β-glucuronidase activity in the experimental group than did the controls which was similar to the results of the study conducted by Nakamura *et al,* (1983), and Lamster *et al,* (1988).

The present study highlighted that the salivary β-glucuronidase is a potential biochemical marker of periodontal tissue destruction and therefore this could be used as a good predictor of periodontal destruction.[13]

Conclusion

The results showed a significant association between clinical periodontal parameters and salivary β-glucuronidase activity. Due to non-invasive and simple nature of saliva collection, the above association can be used as a screening test for periodontitis, and as a means of monitoring the response to periodontal treatment. Further, studies are needed to compare salivary β-glucuronidase level with PMNL chemo taxis to assess whether the increase in the enzyme activity is due to increased recruitment of PMNL, hyperactivity of PMNL or due to increased deregulation of PMNL.

Table 1. Descriptive statistics with mean and range of the selected parameters for Both Groups

Parameter	n		Minimum		Maximum		Range		Mean	
	E	C	E	C	E	C	E	C	E	C
Age	50	50	28	28	60	60	32	32	41.10	41.1
Gingival Index	50	50	1.42	0.0	1.85	0.2	0.43	0.2	1.53	0.11
Pocket Depth	50	50	6.02	1.10	9.13	2.20	3.11	1.1	7.26	1.42
Attachment Level	50	50	6.14	1.20	12.62	2.3	6.48	7.1	8.13	1.51
Total WBC Count	50	50	9100	8500	10800	9500	1700	1000	10080	9500
Monocytes	50	50	1%	0%	3%	1%	2%	1%	1.6%	0.6%
Neutrophils	50	50	69%	45%	76%	55%	7%	10%	71.6%	61%
Lymphocytes	50	50	20%	18%	23%	20%	3%	2%	20.8%	18.8%
β-glucuronidase	50	50	0.173	0.012	0.324	0.031	0.151	0.019	0.255	0.148

n = Number of Patients, Range = Maximum - Minimum
E = Experimental group, C = Control group

Table 2. Results of Pearson's Parametric Correlation analysis between β-Glucuronidase and the selected parameters

Parameters	Correlation Coefficient	p Value
Pocket depth	0.858	<0.01
Mean attachment level	0.762	<0.05
Gingival index	0.085	0.814
Total WBC count	-0.063	0.863
Neutrophils	0.870	<0.01
Lymphocytes	-0.356	0.313

P < 0.05 = Significant, P < 0.01 = Highly Significant

Table 3. Comparison of β-glucuronidase level between the experimental group and control group

	N	β-glucuronidase				
		Mean	SD	t	df	p
Control group	50	0.1483	0.0160	6.0946	18	<0.01
Experimental group	50	0.2557	0.0533			

t = student 't' test value; df = degree of freedom; p<0.01 = significant

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Case Report

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Conservative approaches in the management of Amelogenesis Imperfecta - A case report

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Abstract

Amelogenesis imperfecta (AI) encompasses a complex group of conditions that demonstrate developmental alterations in the structure of enamel. The disease may result in poor development or complete absence of enamel in teeth, due to improper differentiation of the ameloblasts. A multi-disciplinary approach is necessary in the successful management of such conditions. Historically management encompassed extremely destructive crown and bridgework and occasionally total clearance and dentures. However, with the current understanding and efficient direct bonding techniques a very conservative approach is more feasible. The case describes the management of a patient suffering from amelogenesis imperfecta with orthodontic treatment, metal overlays to stabilize the occlusion and direct bonded composite veneers for improvement of aesthetics.

Introduction

Amelogenesis is the exquisitely orchestrated and genetically conserved process of enamel formation.[1] Normally ameloblasts or the enamel forming cells perform multiple functions depending on the stage of amelogenesis including the secretion of and processing of a unique extra-cellular matrix scaffolding that regulates crystalline growth.[2] This extra-cellular matrix is almost completely removed in the mineralization and maturation process, allowing crystallites to grow to their full size producing enamel.[3]

Amelogenesis imperfecta (AI) encompasses a complex group of conditions that demonstrate developmental alterations in the structure of enamel. The disease may result in poor development or complete absence of enamel in teeth, due to improper differentiation of the ameloblast. The anomaly generally affects both primary and permanent dentitions. AI is considered an inherited disorder.[4]

There is a wide variation in the reported incidence of AI. Studies done in Sweden reported the incidence to be 1:700, while other studies done in the US have shown it to be 1:14000 – 1:16000.[5,6] These variations primarily depend on the population of study as well as the criteria used in the diagnosis. As in any hereditary disorder, clustering of affected patients in certain geographic areas may occur and this may also lead to an increase in prevalence in those areas.[7] The inheritance pattern of AI may be autosomal dominant, autosomal recessive or X – linked.[8]

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Enamel defects are a result of genetic mutations associated with major enamel matrix proteins such as amelogenin, enamelin and ameloblastin. The genes coding for Amelogenin which is the most abundant enamel matrix protein is coded in the X chromosome (Xq24-q27). Enamelin is coded in the 4th chromosome (4q11-q21). A number of studies have shown genetic mutations in the above genes in patients suffering from AI.[9] Enamel deposition itself is a multistep process which involves elaboration of the organic matrix, mineralization of the laid matrix and maturation of enamel. Thus the defect can be in one or more of these steps.

Numerous classifications have been proposed for AI though an ideal classification has yet to be accepted. However, the classification proposed by Witkop which relies on the phenotype and pedigree is widely accepted. Accordingly, AI is divided in to 4 broad patterns; Hypoplastic (I), Hypomaturation (II), Hypo calcification (III) and Hypomaturation – Hypoplastic in combination (IV). These patterns are further subdivided depending on the clinical appearance and pattern of inheritance. Altogether 15 different sub types are reported.[10]

Clinical features depend primarily on the subtype and the pattern of inheritance. In the hypoplastic type there is inadequate deposition of enamel matrix. Any matrix present would calcify normally and appropriately. The condition presents as pin point to pinhead sized pits scattered mainly on the facial surfaces of teeth. These pits may be arranged in rows or columns. However, some may present as one large area of discoloured hypoplastic enamel surrounded by normal enamel. This effect is seen in X linked dominant smooth pattern AI (IE) as a result of activation of either the defective or normal X chromosome in different cells leading to alternative areas of sound and diseased enamel.[7,8]

In hypomaturation type AI the principle defect is in the maturation process of the crystalline enamel structure. Teeth appear normal in shape with a yellow to brown mottled colour. As there is defective maturation this enamel is soft and easily abraded. Cuspids show excessive wear compared with those of normal individuals. A lyonization effect is seen in X linked hypomaturation type AI (Type IIC). Radiographically, affected enamel lacks contrast with dentine.[7]

There is no significant mineralization of the appropriately laid down enamel in hypocalcified type AI. The teeth appear normal in shape at eruption with a brown-yellow shade. However, affected enamel is soft and could be easily lost with masticatory function. The only enamel left on the teeth may be in the cervical region which generally escape masticatory forces. Here too the enamel does not contrast with dentine Radiographically.[7] In addition, certain sub types show additional features such as multiple impactions, resorption of teeth, open contacts between teeth and an anterior open bite.

There are a number of aspects to be addressed in the management of any form of amelogenesis imperfecta. Consideration should be given into the management of tooth sensitivity associated with exposure of dentine. Facilitation of maintenance of good oral hygiene is of paramount importance given the irregular nature of affected teeth and resultant retention of calculus. Also the caries risk is no different to that of normal individuals and thus, preventive measures should also be given due consideration for the preservation of all available teeth. Finally, improvement of masticatory function, efficiency and the improvement of aesthetics which are prime concerns of patients should be addressed.[11]

A multi disciplinary team approach is necessary in the management of amelogenesis imperfecta. The team should cover Paedodontic, Restorative, Prosthedontic as well as Orthodontic disciplines. Additional help should be sought from a psychiatrist and a genetic counselor.[12]

The following case describes the management of a patient with hypoplastic type Amelogenesis imperfecta.

Case Report

A 26 year old female from Kandy was referred to the Department of Restorative Dentistry, Faculty of Dental Sciences Sri Lanka from the Orthodontic Unit, General Hospital Kandy, after completion of orthodontic treatment. She has been seen previously at the Advanced Restorative Clinic and was referred to the orthodontic unit for correction of her severe class II division I malocclusion prior to definitive management of her discoloured teeth. (Fig. 1, 2, 3). She was the youngest in the family. None of the other siblings or parents had a similar condition but she recalled that one of her maternal aunts having similarly discoloured teeth.

No record of her primary dentition was available but her mother complained of similar discolouration involving the primary teeth. She revealed an uneventful medical history.

Intra oral examination of this otherwise healthy patient revealed that teeth 15 and 25 had been extracted for orthodontic purposes (Fig 2) , and tooth 35 had been extracted due to caries at the age of 18years (Fig 3). Teeth 28, 38 and 48 had not erupted. All other teeth were present and appeared caries free, except tooth 45 which had a class II mesial amalgam restoration (Fig 2 & 3). There was generalized yellow to brown discolouration mainly concentrated on the incisal two thirds of the facial and oral surfaces (Fig 1). The surface was smooth and the discoloured area was very soft. There was moderate tooth wear affecting especially the occlusal surfaces of 16, 17, 18, 26, 27, 36, 37, 45, 46 and 47 (Fig 2 & 3). Her gingiva appeared healthy with a plaque score of 13%. A basic periodontal examination (BPE) was carried out which revealed scores of 2 in all sextants.

The patient had undergone upper fixed orthodontic treatment for correction of class II division 1 malocclusion with upper incisor crowding (Fig 4, 5, 6). She had a class I incisor relationship on a class II skeletal base. There was a positive overbite and 4 mm overjet. The Occlusal Vertical Dimension (OVD) was reduced by about 2 mm. (Fig. 4, 5 and 6)

Periapical radiographs were obtained in the upper anterior and molar region which showed poorly contrasting enamel from dentine (Fig. 7). From the clinical presentation, and radiographic evidence a tentative diagnosis of hypoplastic type amelogenesis imperfecta (Type ID/IE) was arrived at. As she could not give a detailed family history, the mode of transmission could not be evaluated.

The following treatment plan was formulated considering the patient's symptoms, treatment needs and the patient's expectations.

1) Educating the patient on the condition including the possibility of transmission to subsequent generations.
2) Preventive measures
3) Full mouth prophylaxis
4) Provisional raising of the OVD by means of light cured composites on the upper first molars to assess the tolerability of the possible raise in OVD
5) Placement of Cobalt Chromium (Co/Cr) metal overlays on 17, 27, 36, 46
6) Direct light cured composite veneers on teeth 14, 13, 12, 11, 21, 22, 23, 24, 34, 33, 32, 31, 41, 42, 43 and 44.

The patient was educated on the disease and the possible method of transmission. The possibility of subsequent generations suffering from this condition was also emphasized. The patient's main consideration was the appearance of her anterior teeth and thus the importance of following the formulated treatment plan was explained to her.

She had a DMFT of 2 and thus had a low to moderate caries risk. She complained of sensitivity on tooth 18 and a composite restoration bonded with a universal bonding agent (Clearfil SE Bond, Kuraray, Japan) was placed on the occlusal surface. A session of full mouth prophylaxis was undertaken in the next visit to remove calculus deposits especially in the lingual surfaces of her lower anterior teeth. A set of study models were taken. The extent of her smile was assessed and there was a display up to the first premolar in a forced smile.

Composite restorations were then placed on teeth 16 and 26 to raise the OVD in order to obtain a separation of 1-1.5 mm between the posterior teeth (Fig 8). The patient could tolerate this raise in OVD and it was then decided to construct Co/Cr overlays to this height. It was decided to place the overlays on teeth 17, 27, 36 and 46 as these teeth were in good occlusion. The selected teeth were prepared as per standard guidelines (Fig 9) and two stage putty-wash condensation silicone impressions (Speedex, Coltene Whaledent USA) were obtained. The raised OVD was transmitted to an articulator (Fig 10) and the overlays were fabricated in the laboratory (Fig 11 and 12). These were tried in the patient's teeth and contacts were relieved using articulating paper until bilateral balanced contact was obtained. The overlays were cemented (Fig 13 and 14) with resin cement (Panavia F 2.0 Kuraray Japan).

The patient was reviewed in one month to assess the tolerability of the new OVD. As she had adopted to the new OVD, Direct composite veneers (3M Filtrek Z-200) bonded with a dentine bonding agent (Clearfil SE Bond) was placed on all anterior teeth and the first premolars (Fig 15 and 16). A labial reduction of 0.5 mm was done on the labial surfaces of these teeth.

The patient was reviewed after completion of treatment in one month, three months, one year and two years. At the one year review she had dislodged the veneer on tooth 24 and this was replaced. No further complications arose within a four year review period.

Discussion

Management of a young patient with amelogenesis is a truly challenging task. Several factors have to be taken into consideration such as the quality of existing dental tissue, pulp status of teeth, periodontal condition of teeth, amount of lost tooth substance, presence of any malocclusions requiring orthodontic correction and age of the patient.[11] A multidisciplinary approach is generally required in effective management.[12,13]

Treatment is carried out in three phases. In the temporary phase, (undertaken during the primary and mixed dentitions) consideration is paid to oral hygiene maintenance, preventive aspects, alleviation of symptoms such as sensitivity and maintenance of the Occlusal Vertical Dimension. In the transitional phase, (carried out in the permanent dentition during adolescence) provisional aesthetic rehabilitation is carried out in addition to the above aspects. The permanent phase is performed in adulthood with definitive aesthetic as well as functional rehabilitation.[11] As the patient was an adult definitive aesthetic and functional rehabilitation was considered.

Alleviation of symptoms such as sensitivity and pain due to exposure of dentine or pulpitis should be the first priority. With modern adhesive techniques available, glass ionomers or light cured composites could be used as provisional or definitive restorations to cover exposed dentine. With severe attrition associated especially with hypocalcified types of AI pulp exposure may occur necessitating endodontic treatment of the affected teeth. Endodontics may also be complicated in cases where taurodontism is associated.[7] This patient did not show extensive tooth wear and dentine exposure and sensitivity was only witnessed in tooth 18 which was restored with a light cured composite restoration. All other teeth were asymptomatic despite the level of

attrition seen. Preservation of all available teeth is a very important aspect in management and acceptable preventive measures should be provided.

The periodontal status of teeth should also be carefully assessed prior to planning any definitive restorations. Due to the irregular surface of teeth, maintenance of oral hygiene may be hampered and this could lead to plaque deposits and subsequent gingivitis or periodontitis. Thus, after a periodontal assessment if found necessary meticulous plaque control measures have to be instilled. This patient had an acceptable level of oral hygiene and a session of oral prophylaxis was carried out to remove calculus deposits which were restricted to her lower anterior teeth.

Many patients with AI would present with certain malocclusions such as spacing due to altered morphology of teeth, impactions and hypodontia. In addition to this many cases present with an anterior open bite.[1,14] In severe tooth wear cases orthodontic extrusion may also be needed to obtain adequate crown height for the fabrication of prosthesis. Thus orthodontic intervention may be needed to correct such problems in order to facilitate a good aesthetic outcome. This patient had undergone fixed orthodontic treatment to correct malocclusions as mentioned in the case report.

Most patients suffering from AI would show some amount of attrition of especially the posterior teeth due to the diseased enamel being less harder than normal. Thus some form of occlusal rehabilitation is essential to provide adequate masticatory function and obtain inter occlusal clearance for placement of anterior restorations. A thorough evaluation of the amount of tooth wear as well as the available crown height is mandatory. If remaining crown height is inadequate orthodontic extrusion or surgical crown lengthening may be needed to gain good retention of restorations. Care must be taken in surgical crown lengthening in posterior teeth as exposure of furcation may predispose such teeth to long term periodontal problems.[15] If adequate crown height is available or obtained by above means, posterior restorations can be planned at a raised OVD. This should first be assessed prior to fabrication of definitive restorations by provisional composite buildups to assess the tolerability of the raised OVD.[11] If the patients could tolerate definitive restorations in the form of porcelain or porcelain fused to metal crown or bridgework, cast metal crowns or metal overlays could be provided.[16] Care should be taken in providing porcelain restorations especially if opposed by natural teeth as this could aggravate tooth wear.[15] Identification and treating the patients at an early age will minimize loss of OVD if consideration is given to maintain OVD by providing preformed stainless steel crowns in primary molars.[17] This patient was provided with cast metal Co/Cr overlays luted with a resin cement to obtain an inter occlusal clearance of 1-1.5 mm as she did not show her molar teeth even in a spontaneous smile and considering the possible benefits of having a supra gingival margin on periodontal health. The previously described teeth were selected as these teeth opposed each other due to previous extractions and orthodontic treatment. Due to financial constraints onlays on the other posterior teeth were not considered and direct light cured composites placed instead.

There are a number of alternatives for improvement of anterior aesthetics which include provision of all ceramic crowns, porcelain veneers, or direct composite veneers. The choice of material will depend on the available tooth tissue, availability of facilities and cost. The most predictable and durable aesthetic restoration of anterior teeth have been achieved by placement of full coverage crowns. As this approach requires removal of substantial amount of tooth tissue in compromised teeth, porcelain veneers have gained wide acceptance as a suitable alternative. However, disadvantages such as poor marginal adaptation, bonding problems and financial considerations do exist. With the advence

of efficient bonding systems coupled with aesthetic resin composites, direct restorations have also gained popularity as it offers acceptable aesthetics which involves minimal cost.[11,18] However, colour instability and inferior mechanical properties would require periodic replacement. Considering the financial implications the patient was provided with direct composite veneers to improve anterior aesthetics. Additional help should be sought from a psychiatrist in order to manage any psycho-social problems which arise as a result of poor aesthetic appearance and impaired masticatory function. Genetic counseling plays a role in identifying inheritance traits and educating such patients.[12] As the patient did not report any other relatives having a similar condition the mode of genetic transmission could not be determined.

Thus through early detection and systematic rehabilitation, the clinician's expectations such as aesthetics and near normal masticatory function could be achieved. An expectation of an improvement in dental aesthetics is of great concern to the patient. In essence a systematic approach would provide the patients suffering from AI an acceptable and a pleasant appearance without a compromise on their masticatory efficiency.

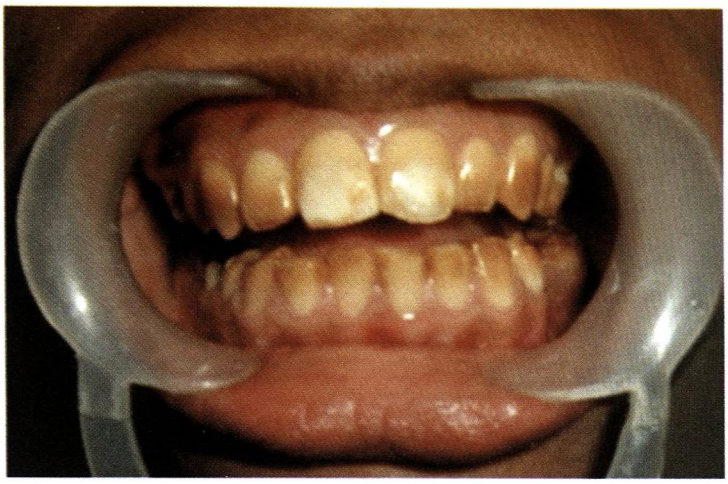

Figure 1. Pre Operative View

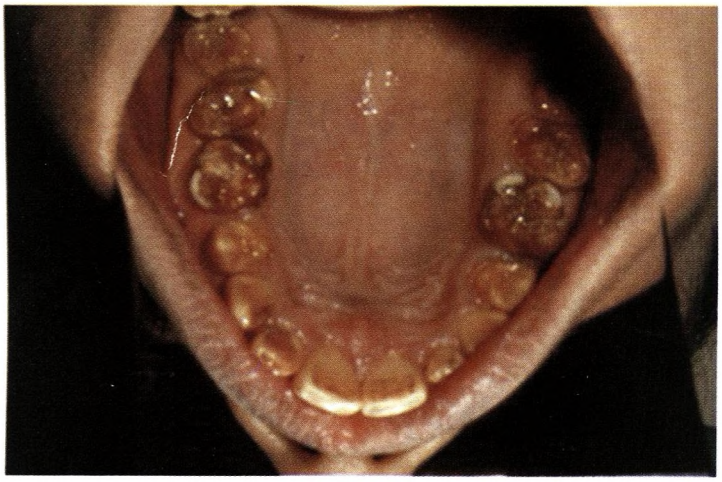

Figure 2. Pre Operative View (Upper Arch)

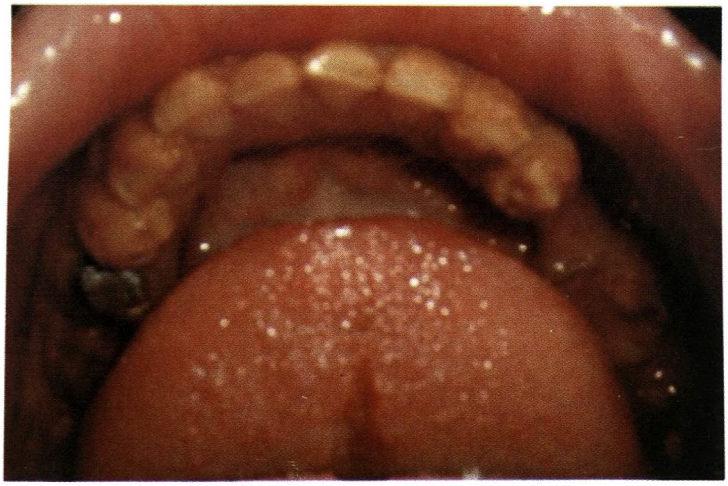

Figure 3. Pre Operative View (Lower Arch)

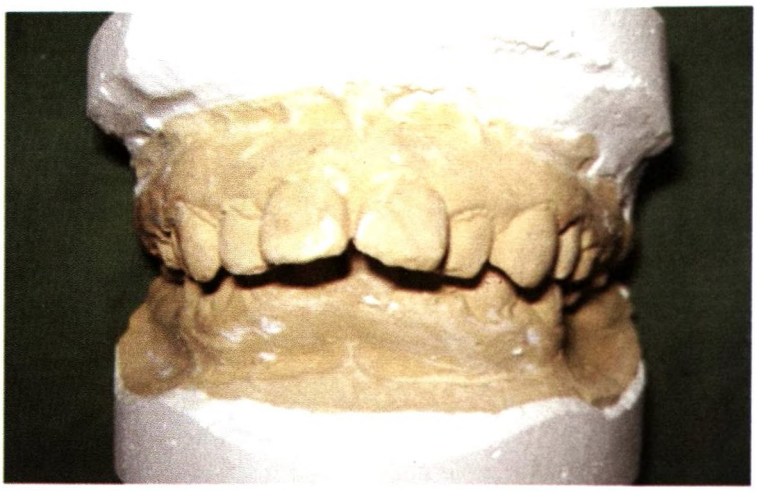

Figure 4. Pre Orthodontic study models

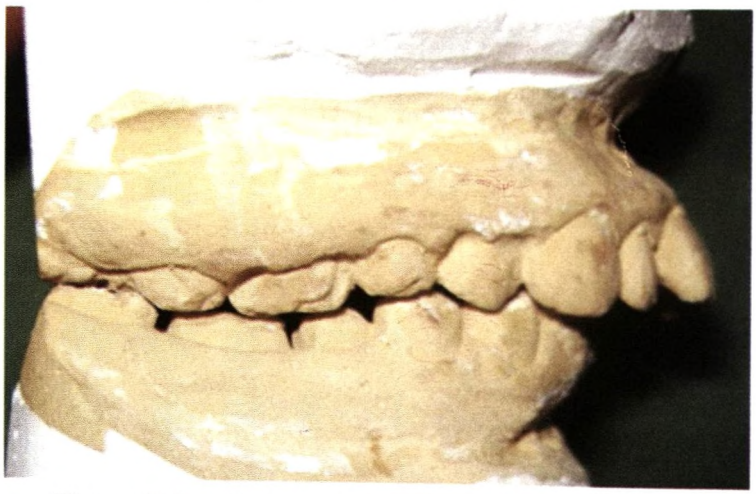

Figure 5. Pre Orthodontic study models (Right Side)

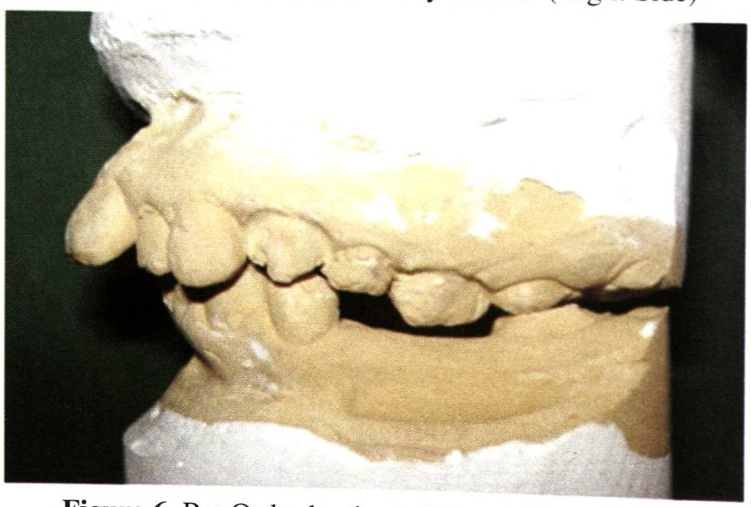

Figure 6. Pre Orthodontic study models (Lift Side)

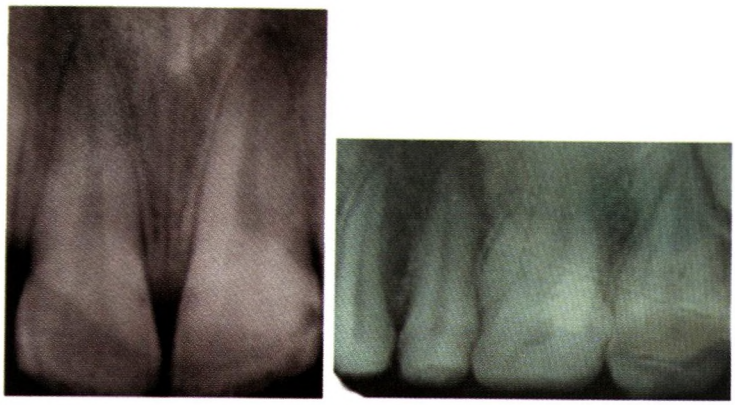

Figure 7. Periapical radiograph s of 11,21 & 13,15,16,17

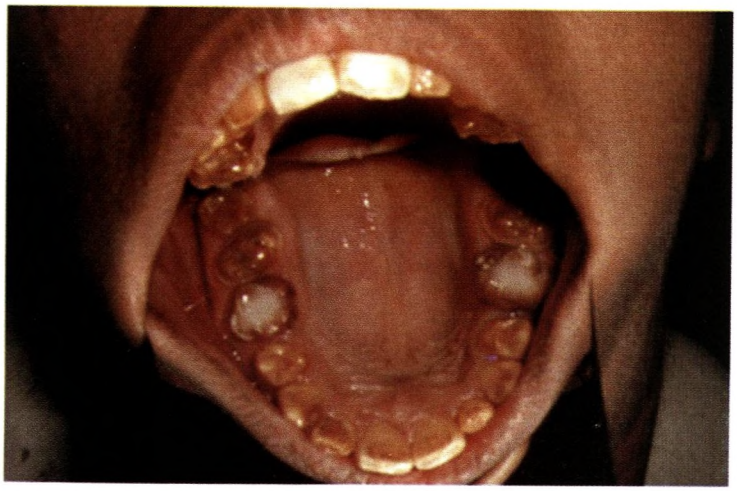

Figure 8. Provisional raising of vertical height using dentine bonded composites

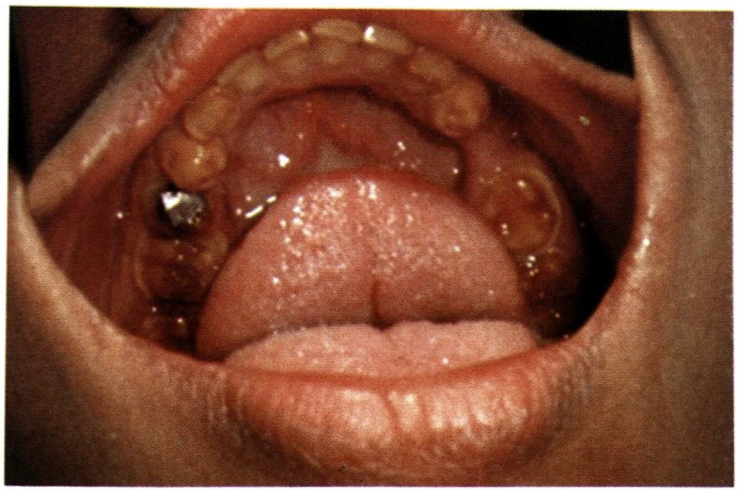

Figure 9. Preparation of 36 & 46

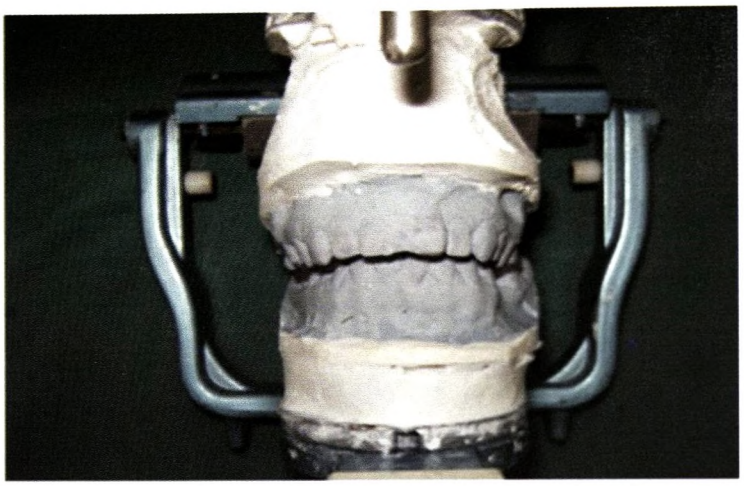

Figure 10. Casts mounted with a raised DVD

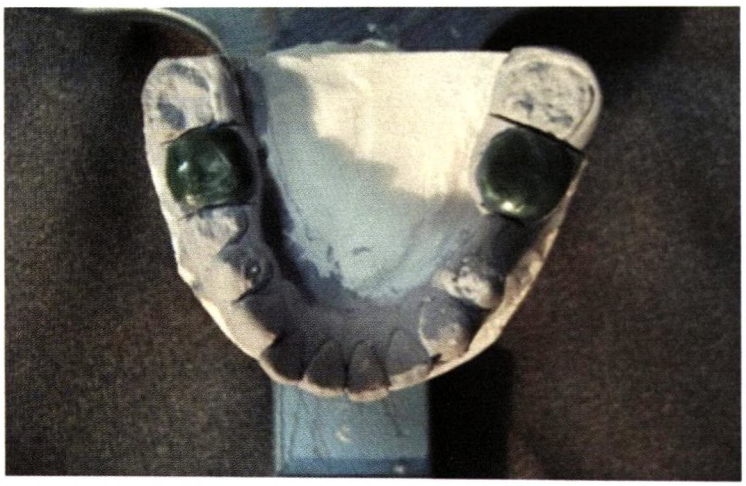

Figure 11. Wax pattern for lower overlays for 36, 46

Figure 12. Upper and lower metal overlays of 17,27,36 and 46

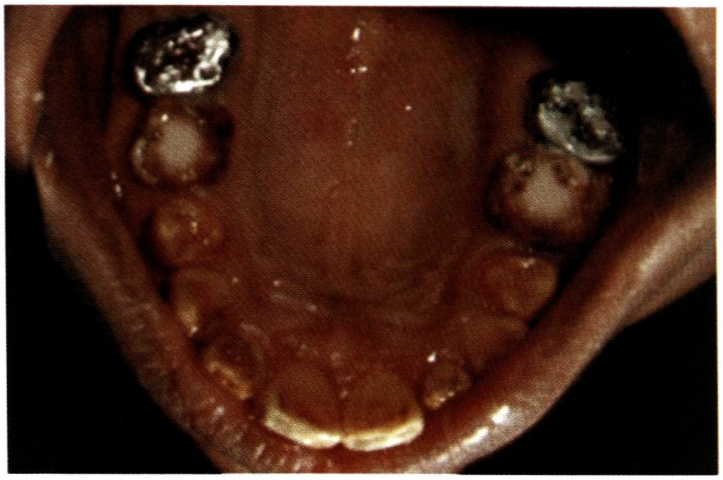

Figure 13. After attachment of Co/Cr overlays on 17 and 27

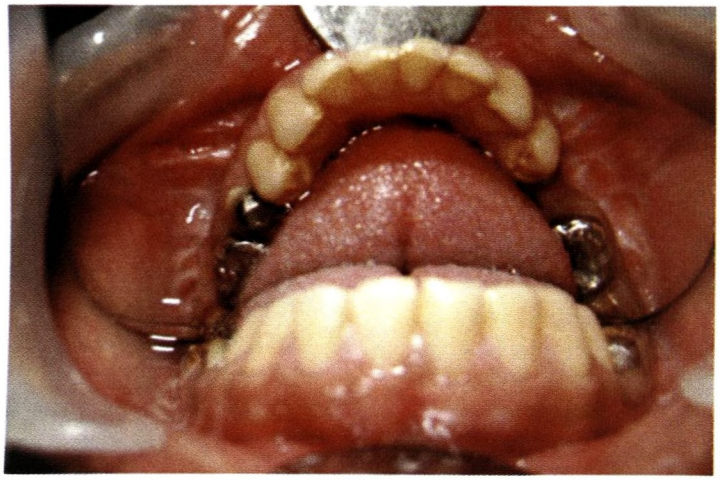

Figure 14. After attachment of Co/Cr overlays on 36 and 46

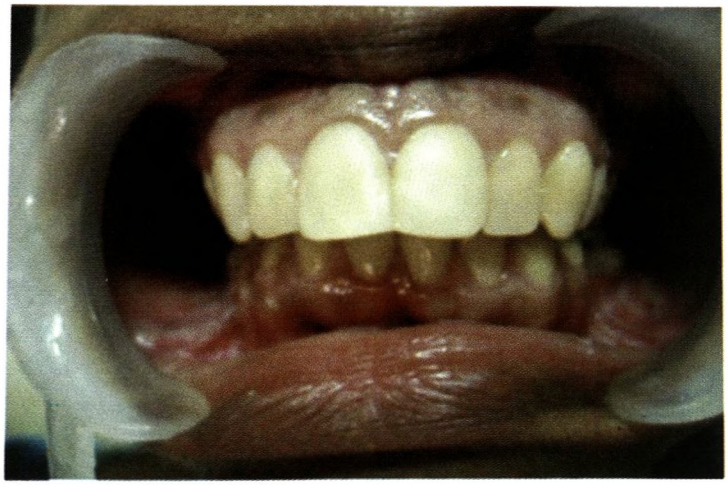

Figure 15. Following direct composite vaneers of 14 to 24 and 34 to 44

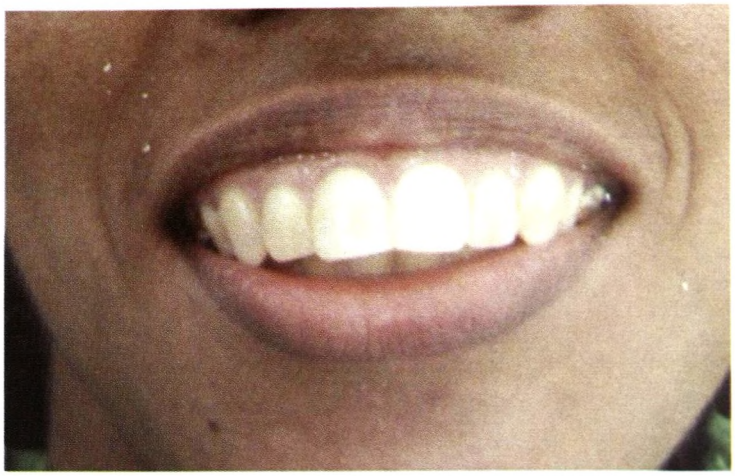

Figure 16. Post operative view the years later

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Barker DS. Lucas RB. Localised fibrous growth of the oral mucosa. J Dent Res 1965: in press.

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Pindborg JJ Atlas of diseases of the oral mucosa. 5th edition.. Copenhagen: Munksgaard, 1992: 50-66.

Chapter in book

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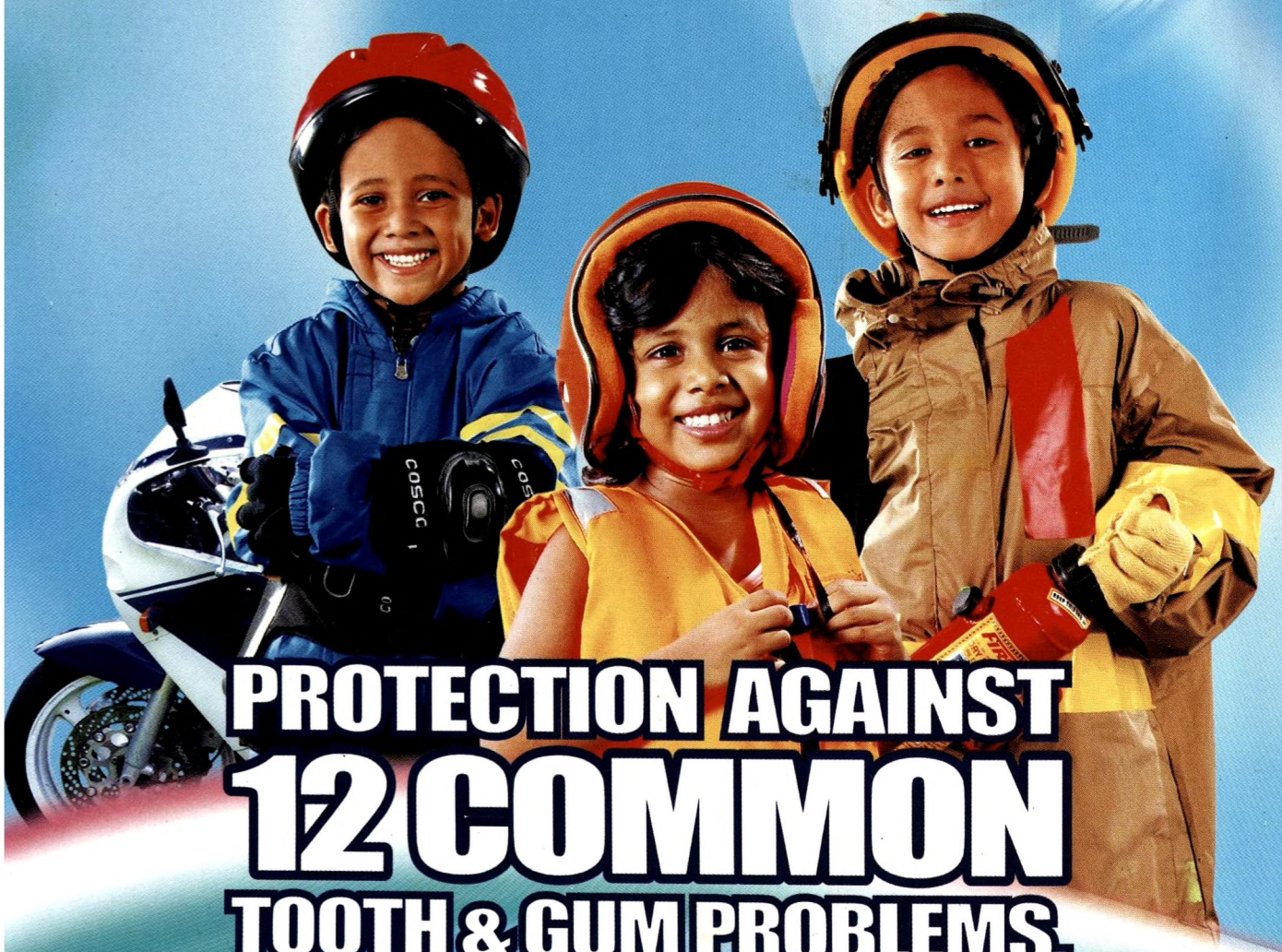

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