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EDITORIAL

CONTINUING PROFESSIONAL DEVELOPMENT (CPD)

The leading article on Continuing Professional Development in the present issue of the SLDJ is an eye opener to the dental professionals in Sri Lanka in number of ways.

'Continuing Professional Development is the systematic maintenance, improvement and broadening of knowledge and skill, and the development of personal qualities necessary for the execution of professional and technical duties throughout one's working life'.

Why CPD is so important?
Science is constantly changing. What you learned yesterday may not be applicable or valid for practice any longer. Once a dental surgeon is trained and qualified, it used to be possible to sit back and just work away at any encountered problem. However, that is not what the society expects or the profession expects from you. If some one wants to sustain in the profession as a successful professional, the only way out is acquiring new knowledge, improve skills and familiarize with latest technology or in other words engage in continuing professional development (CPD) activities.

Where are we?
Most of the non medical professional organizations in Sri Lanka have already developed continuing professional development

activities to update their own professions. Medical profession is now actively engaged in professional development activities, even though it is still under volunteer basis. To my understanding, dentistry as a profession has not taken adequate steps in this regard. CPD is a compulsory and legitimate requirement in the western world. This is going to be the scenario in Sri Lanka too in the near future. As such without any further delay, we as a profession should work towards this both for the safety of the patients as well as the well-being of the profession.

Upul B Dissanayake
Editor

Leading Article

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Continuing Professional Development (CPD) programmes for dental professionals 'Today's Doctor-Tomorrow's Cure'

Jayantha Weerasinghe

Introduction

Continuing Professional Development (CPD) is the means by which members of professional associations maintain, improve and broaden their knowledge and skills and develop the personal qualities required in their professional lives. Several terms are in use giving more or less similar meanings of various aspects of CPD.

Continuing education is a term within a broad spectrum of post-secondary learning activities and programmes. Continuing Medical Education (CME) is a term used in the past for the process of life long learning acquired by medical personnel after graduation for updating their knowledge.[1] Formal educational activities have not been found to be making much of an impact on the physician's behaviour and the health care outcome.[2]

Some of the competencies expected by professional bodies such as American Board of Medical Specialties in addition to medical knowledge and patient care include: inter-professional and communication skills, professionalism, practice-based learning improvement and systems-based practice.[3] Over the years inclusion of topics related to professional skills which are considered beyond traditional clinical subjects has changed the concept of CME. Professional development refers to skills and knowledge attained for both personal development and career advancement. Professional development encompasses all types of facilitated learning opportunities, ranging from college degrees to formal coursework, conferences and informal learning opportunities situated in practice. CPD is aimed at changing the practitioner's skills and attitudes towards improving standards of patient care.[4]

CPD can also be defined as the conscious updating of professional knowledge and the improvement of professional competence throughout a person's working life. It is a commitment to being professional, keeping up to date and continuously seeking to improve. It is the key to optimizing a person's career opportunities, both today and for the future. It is continuing because learning never ceases, regardless of age or seniority; professional because it is focused on professional competence in a professional role; concerned with development because its goal is to improve personal performance and enhance career progression, which is much wider than just formal training courses.

World wide strategies

Continuing professional development may be essential for specialized professionals such as general surgeons.[5] Changing attitudes of dental practitioners towards participating in CPD courses have also been reported when the General Dental Council in U.K. planned to introduce revalidation scheme.[6,7] Let us now explore the strategies employed by different countries with regard to CPD. According to a survey conducted by Peak *et al*, (2000) programmes of CPD are being

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conducted by many organizations worldwide.[8] Although their approaches and methods vary they all contain topics covering a spectrum of clinical, professional and managerial skills. In this survey, it has been reported that countries including Canada, USA, U.K, Australia and New Zealand consider CPD as a mandatory exercise. These programmes are being organized by respective professional bodies worldwide. None of the countries consider holding examinations for this purpose. The CPD activity is used for recertification (renewal of license to practice) purposes by USA. In U.K participants receive incentives such as fees and promotions in addition to revalidation. Most countries publish a list of successful practitioners to make the participation in CPD programmes attractive. Most programmes offer an hourly based credit system. The advantage of this system is that the time devoted for activities can be measured and recorded. On the other hand it may not reveal the true picture on quality and relevance of the activity. The Kuwait Institute for Medical Specialization which has been conducting CME courses for medical and dental professionals since the year 2000 has published comprehensive guidelines for organizers of CME programmes.[9]

National trend

CPD activities have been identified to be conducted among various professional organizations to improve standards. The Institute of Professional Management of Sri Lanka (www.ipmlk.org) a leading organization in human resource management has published extensive guidelines for CPD.[10] In relation to the medical field, The National Centre for Continuing Professional Development in Medicine has initiated the 'National Programme of CPD for doctors and already made available a portfolio guidance book to be used for this purpose. This activity will be discussed in detail later in this article. There is a national need for medical personnel to get involved in CPD activities that will benefit themselves, the profession and more importantly the patients who seek treatments.

Availability of postgraduate medical programmes in Sri Lanka

A dental surgeon graduating in the present day will have ample opportunities to pursue higher education within the country. The Post Graduate Institute of Medicine (PGIM) conducts four MS/ MD programmes in dentistry (Oral and Maxillofacial Surgery, Restorative Dentistry, Orthodontics and Oral Pathology) and offers opportunities to obtain specialist qualifications. Two diploma programmes of the PGIM help senior dental surgeons to improve knowledge and professional skills. Namely, the Diploma in Hospital Dental Practice will benefit hospital dental surgeons while the Diploma in General Dental Practice is useful for private practitioners. The latter is a distance learning programme which enables a trainee to learn at his/her own place. The latest programme offered by the PGIM (offers from 2008) is a M.Sc programme in Biomedical Informatics. This is a discipline regarded to be formed by the intersection of Computing, Medicine and Biology. This will open a trusted career pathway for a doctor with special interest in Information and Computer Technology (ICT). In addition various M.Sc and Certificate courses are being envisaged in the future by the Faculty of Dental Sciences. However, all these postgraduate courses could only absorb less than hundred dental surgeons annually. Due to various commitments majority of senior members may not wish to enroll into this type of 'formal' training programmes. Therefore, there must be other opportunities for majority of members to get their knowledge updated. As clinical treatment modalities in dentistry are changing rapidly there is a demand among the profession to acquire new knowledge.

Role of professional organizations in CPD

CPD programmes conducted by the professional organizations include various seminars, workshops and other learning opportunities that are well attended by the members who seek new knowledge and experience. While the Sri Lanka

Dental Association is responsible for the CPD activities of dental practitioners, the College of Dentistry and Stomatology of Sri Lanka which is recognized by the PGIM as the umbrella organization for all associations related to dental subspecialties will be able to play a major role in certification processes of CPD activities of specialists. Web based distance learning programmes which is the tool of the modern concept of distant education gives an effective and cost-efficient opportunity for learners and tutors.[11] Some organizations provide online education websites via which the user will be able to obtain relevant knowledge and also to record CPD activities.[12,13,14] One other important aspect of CPD programmes is that it should be made accessible for all professionals including those work in rural areas.[15,16] However, the professional associations are required to play a major role in this regard through their provincial branch associations for providing their members with adequate opportunities of CPD.

CPD activities

Since CPD is a self managed and life long process of professional development, continuous exposure and acquisition of wide range of knowledge, skills and experience will make a professional member a successful individual in the society.[17,18] There are many motivating factors for joining CPD programmes. Some individuals may have an inherent desire to develop and update their professional knowledge and skills that will help them to demonstrate their professional standing to patients, employers and peers. It could also help them to improve career prospects and advancements. They could use the evidence of CPD as a protection in medical litigations. In future many professional associations would consider making CPD activities as a requirement in recognition of professional standing of a member.[19]

When an individual practitioner wishes to embark into CPD activities, first it is necessary to identify the achievements one has attaind so far by personal reflection and then to plan the kind of experiences and improvements that are needed for the future. It may be possible to apply principles of SWOT analysis (identifying Strengths, Weaknesses, Opportunities and Threats) in this regard. Most practitioners will be able to rank their knowledge and skills in comparison with their peers. At the same time it is necessary to identify weak areas that need improvements.

Some of the expected outcomes in CPD as stated in the CPD guidelines issued by the National Centre for Continuing Professional Development in Medicine are:

1. Systematic maintenance, improvement and broadening of knowledge:
   It will be possible to get the knowledge improved and updated through CPD activities. These can be achieved by participating at meetings or academic activities both designated, non-designated and individual events.

   Designated events are formal learning opportunities organized by various professional colleges or organizations including conferences, congresses, workshops, lectures, seminars, refresher courses and colloquiums.

   Non-designated events are local meetings, case conferences, journal clubs, non-routine teaching sessions.

   Individual events are undergraduate teaching, postgraduate teaching and supervision, undertaking examination components, educational administration, consultancies,and committee work etc.

2. Systematic maintenance and improvement of skills:
   CPD activities will also help to improve skills and capabilities of a professional. These can be achieved by participating at skill training or

any other similar activities such as practice drill on Cardio Pulmonary Resuscitation (CPR) etc. This also may include activities such as short term study courses on skill development followed at a tertiary education institution, special project work, staff training, developing transferable skills such as languages, IT, business/financial skills and management skills.

3. Development of personal characteristics, attitudes and behaviours expected of a healthcare professional:
   Evidence of organizing or participating in a voluntary service to the community such as health camps, letters or newspaper articles indicating public appreciation of the work of an individual etc.

4. Personal and professional development:
   These can be achieved by publishing research reports and papers in journals approved by an accredited provider, papers read at conferences or congresses/poster presentations and obtaining additional completed qualifications related to professional practice.

5. Develop into a self-directed reflective leaner:
   These could be achieved by methods such as assimilation of knowledge from self learning activities such as reading a journal article and writing a summary, distant learning programmes, self study on specific topics such as reflective writing about a professional experience etc. and use of internet, audio-video or other multimedia resources and library.

Recording of CPD activities- Personal Portfolio

A portfolio in CPD is a collection of evidence which provides proof of the achievements of knowledge, skills, attitudes, understanding and professional growth attaind through a process of self-reflection over a period of time. It can be a collection of A4 size papers in a ring binder. A portfolio will be a documentary evidence to prove an individual's commitment to obtain continued competence in the chosen filed. It also helps in career development and acts as an easy reference for building up curriculum vitae.

There are three main categories of records in a portfolio. Firstly personal learning logs are entries prepared by the learner that contain details of learning activities, description of the knowledge and skills obtained and areas for future learning. Secondly certificates awarded that show evidence of the learning activities. Thirdly any other evidence of personal development such as publications, letters from patients or newspaper articles appreciating the clinical work etc.

Award of credit points could be performed by the learner by adopting an approved scheme. At the end of a stipulated period the portfolio can be submitted for the authorized CPD centre to receive a certificate.

In conclusion it can be stated that is high time to createawareness among the members of professional associations regarding the advantages of involving in CPD activities which will help today's doctor to acquire the required knowledge, skills and attitudes in professional life that also benefit patients whom they treat with the modern treatment methods.

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Review Article

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Tuberculosis: a brief overview of dental perspective

J. A. M. S. Jayatilake

Abstract

Tuberculosis (TB) primarily caused by *Mycobacterium tuberculosis* is a chronic infection of lungs. It can also infect other organs including brain, liver, bones and kidneys. Resurgence of TB has been observed in the face of increasing immunocompromised conditions due to HIV infection, immunosuppressive therapy, malnutrition, poverty and crowded living. Spread of TB takes place via droplet nuclei of respiratory secretions. Many dental procedures generate aerosols and Dental Health Care Personnel (DHCP) often work more close to patients. As such, there is a potential risk of TB cross transmission during dental treatment. Moreover, TB can induce oral lesions such as chronic oral ulceration, gingival enlargement, osteomyelitis, alveolar bone loss and cervical lymphadenopathy. Proper diagnosis and prompt referral of such lesions by DHCP will help in the treatment and prevention of this infection which is easily curable. Moreover, DHCP should take adequate precautions with meticulous infection control practice to prevent cross transmission of TB in the dental clinics. This review explores current literature on TB with reference to general and oral manifestations of the disease, current trends of infection and prevention of cross infections in general dental practice.

Key words: *Mycobacterium tuberculosis*, tuberculosis, dentistry, infection control

Introduction

Tuberculosis (TB) has been a worldwide health problem for centuries. It is a chronic bacterial infection primarily due to *Mycobacterium tuberculosis* and is responsible for more deaths world wide than any other infection. However, there are two other related organisms; *Mycobacterium bovis* and *Mycobacterium africanum* which are responsible for TB in some patients.[1] Each year about eight million people develop TB and two million develop latent form of TB while three million die of TB globally.[2] Strikingly, about 90% infected with tuberculosis remain asymptomatic, only about 5% develop the disease in the first five years while the other 5% develop the disease in later years.[2] In Sri Lanka, Tuberculosis is still a major public health problem and about 9000 patients are identified as infected with tuberculosis every year, of which around 60% are diagnosed as pulmonary tuberculosis cases.[3]

TB has emerged violently with the onset of the HIV epidemic in the late 1980s and this problem has aggravated with the isolation of multi drug resistant (MDR) mycobacteria and HIV co-infection.[1] This has also been facilitated by the migrant workers, deterioration of health care facilities, exploding population, smoking, war, crowded inhabitation, malnutrition and low socioeconomic conditions in many parts of the world. TB is classically a disease of poverty and

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many of the TB patient population live in low-income countries. Most significant feature of TB is that the infectivity could be arrested almost completely by starting proper antibiotic treatment even for a shorter period like two weeks.[4]

Aetiopathogenesis

Mycobacterium tuberculosis is a slender rod shaped bacterium having a waxy coat made up of fatty acids that prevents them being killed by phagocytes and harmed by antibiotics. Moreover, such a complex architecture of the cell wall prevents them from being decolorized by acids once stained with dyes. Therefore, they are better known as acid fast bacilli. On culture, this bacterium takes a long period to multiply (18-24 h) and takes months to form colonies. TB is an airborne infection that spreads via droplet nuclei generated in the respiratory tract. Droplet nuclei expelled from the lungs when the patients cough, sneeze, spit, sing, speak or laugh carry organisms to the external environment. These droplets are capable of surviving in the air for a long period so that they could be inhaled by another individual. However, coughing or sneezing at one instance usually brings out only a few organisms into the air. Therefore, prolonged exposure to an active TB patient is necessary for a person to catch the disease.[4] Sputum containing live bacilli from TB patients is highly contagious.

The primary site affected by TB infection is the lung alveoli and bronchioles. Once droplets are inhaled the organisms overcoming the innate immune barriers of the respiratory tract reach the alveoli and infect the lung tissue. Macrophages engulf the inhaled bacilli. Though some of the bacilli inhaled are killed by the macrophages some can survive within and proliferate inside the macrophage. Once the macrophages engulf and destroy some of the tubercle bacilli, T lymphocytes start production of chemical mediators and induce cell mediated immune response which is also known as delayed type hypersensitivity. Thus, the immune response to TB is predominantly cell mediated. If the immune system is strong enough the primary infection is contained within a chronic inflammatory granuloma known as the tubercle. However, in immunocompromised individuals such as children and elderly, HIV patients, and malnourished people tubercles can break down leaving TB bacilli spreading into various tissues such as meninges, kidneys, liver, bones and joints. On the other hand, live *Mycobacteria* can travel to other body sites within macrophages.

Clinical features

General features

Pulmonary TB is the typical presentation of the TB infection. Common features of this disease are chronic cough often with haemoptysis, general weakness, low grade fever, weight loss, ill health, and night sweating. There is lymphadenopathy and, it is mostly seen in the cervical lymph nodes. Occasionally, extra pulmonary lesions may appear in the form of primary or secondary lesions. Expectoration of the TB bacilli lead to self inoculation and tuberculous tracheitis, laryngitis, tonsillitis may result as secondary infections.

Orofacial manifestations

Naturally, oral mucosa serves as a barrier for *M. tuberculosis*. In a pioneering study, Gruber(1949) examined the hard and soft tissue changes in the oral cavity including teeth, mucosa, salivary glands etc, of the TB affected individuals and concluded that there were no pathognomonic oral signs of pulmonary TB.[5] Yet, TB lesions have been described in the orofacial region occasionally in the past decades and it is worthwhile for dental health care personnel (DHCP) to be aware of them owing to growing immunocompromised population prone to infections.[1] Currently, with the introduction of modern antituberculous drugs and improvements of the health care system, orofacial manifestations of TB are extremely rare.

Oral lesions of TB may be caused by primary as well as post-primary TB infections. If the oral lesion is due to primary TB, the initial site of

infection is the oral cavity. Secondary lesions occur subsequent to post-primary TB such as pulmonary or other TB diseases. Although there are a few reports of primary TB lesions in the oral cavity, oral TB results from self inoculation by the infected sputum subsequent to pulmonary TB. Organisms may penetrate the tissues via a traumatized mucosa or an extraction socket. Common oral manifestations of TB are superficial ulcers, patches, indurated soft tissue lesions, or osteomyelitis of the jaws and chronic ulcerative lesions.[2] These lesions are mainly seen on the tongue while lip, cheek, soft palate, uvula, gingiva and alveolar mucosa also can be affected.[2,6] Therefore, it has been suggested that TB should always be considered as a possible cause of oral ulceration.[7] Some of the important features of these ulcers are undermined irregular edges, minimum induration and the presence of a pseudo membrane over the basal granulation tissue.[2]

Alarmingly, incidents of primary oral TB appearing in unusual clinical guises apart from the usual oral ulceration have been reported especially in immunocompromised patients. For example, Karthikeyan *et al.*(2006) reported a case of primary tuberculous gingival enlargement in a ten year old girl.[6] This report has emphasized the need for considering tuberculosis in the differential diagnosis of gingival enlargements and ulcerations. In a similar case report, Revera *et al.*(2003) described about a patient having primary oral TB with maxillary gingival enlargement and alveolar bone loss.[8] These investigators have pointed out the importance of considering TB in the differential diagnosis of conditions causing gingival enlargement and alveolar bone resorption. Secondary oral TB lesions often have led to diagnosis of underlying pulmonary TB in many patients.[2,7,9] This examplifies the necessity for proper investigations in chronic oral ulcerations for possible TB infections.

Laboratory diagnosis and treatment

Sometimes, TB mimics other respiratory tract associated disease conditions such as pneumonia, lung abscess and tumors. Therefore, the diagnosis of TB is directly associated with proper history, signs and symptoms and radiological evidence. At the same time, several investigations including microbiology, histopathology, immunology molecular biology and radiology play a substantial role in the diagnosis of TB and the following investigations are currently in use in the diagnosis and screening of the infection.

Smear microscopy (Acid fast test)

In this simple staining technique, smears prepared from sputum or other clinical microbiological specimens are stained with the Ziehl Neelson stain (acid fast stain) and examined using light microscopy. Mycobacteria that are capable of retaining the primary stain in the presence of acid or alcohol decolourization are seen in pink. This inexpensive and efficient technique is extremely helpful in screening communities in resource-limited countries. However, the results depend on the skill of the operator and the proper collection and transportation of the specimen. Smear staining is the main technique to screen the patients in directly observed therapy (DOT) where patients are under direct surveillance for their infectivity and response to therapeutics.

Bacteriological culture

Culturing *M. tuberculosis* from the specimens in appropriate selective and nonselective media helps definitive diagnosis of the infection. However, cultures are time consuming and expensive. It requires various growth conditions for the organisms. Positive bacteriological culture is important in determining the state of TB infection and choosing appropriate therapeutics. Therefore sputum, tracheal aspirates, and urine etc, are sent to microbiological laboratories for culture and antibiotic sensitivity testing (ABST).

Chest radiographs

One of the important diagnostic tools of pulmonary TB is the chest radiography. The radiological evidences could vary according to the stage of the disease and common radiological features are

pulmonary consolidation, hilar and mediastinal lymphadenopathy and cavitations.[10]

Tuberculin skin test/ Mantoux test

Purified protein derivative (PPD) of *M. tuberculosis* is injected intradermally to the forearm and examined after 48-72 hours. Redness and swelling due to delayed type hypersensitivity reaction in response to PPD at the injection site demonstrates previous exposure to TB. But it is not a definite indicator of active disease status. Once, there is significant reaction for the tuberculin test other investigations should be utilized for the confirmation of active infection.

Histopathology and immunology

Mycobacteria present in the tissues as well as the associated chronic inflammatory response could be demonstrated using histopathology. This method has become popular as it helps presumptive diagnosis with the availability of the organism specific stains used in immunohistochemistry. Moreover, immunological tests such as enzyme linked immunosorbent assay (ELISA) against antibodies to TB bacilli are also currently available.[6]

Molecular biology

Molecular biological investigations using polymerase chain reaction (PCR) have also been introduced to identify TB specific DNA in patient samples. This technique has been used by Eguchi *et al* (2003) to examine the recovery rate of *M. tuberculosis* from different oral samples including saliva, dental plaque, dental caries and denture plaque.[11] These investigators concluded that PCR is a highly useful and sensitive technique for the detection of *M. tuberculosis* especially in oral samples. At the same time it is a rapid and relatively simple diagnostic tool. PCR has been used to confirm oral tuberculosis in various occasions recently by several investigators.[6,8]

TB is a disease which is preventable and curable easily. With the introduction of new treatment strategies like directly observed therapy (DOT), management of TB has become easier than before. Chemotherapy is the prime approach to treat infected patients while nutrition and other general hygiene improvement play an additional role. Antibacterial drugs are used in combination for quick elimination of organisms and to prevent development of resistance. The commonly used dosage regimen of antituberculous drug therapy is with isoniazid, rifampin and pyrazinamide administered daily for two months followed by isoniazid and rifampin given daily, twice a week or thrice a week for four months.[12] Ethambutol or streptomycin would be added in the presence of antibiotic resistance. However, different dosage regimens are applicable with reference to patient conditions and the microbial virulence. Immunization with the live attenuated *M. bovis* (Bacille Calmette-Guerin: BCG) is the worldwide method used for the prevention of TB.

Relevance to clinical dentistry

Being a respiratory tract infection primarily spread via air borne droplet nuclei, TB poses a threat of cross transmission in the dental clinic. In a survey to assess the impact of TB on the hospital dental practice in the USA where 132 dentists responded, 12% had reported at least one tuberculin skin test conversion by DHCP within a year while five respondents reported DHCP contracting TB through patient care.[13] On the other hand, 120 dental patients who were unaware of their TB status were investigated using PCR on salivary samples, revealed that 30(25%) were positive for *M. tuberculosis.*[14] Although this study did not demonstrate infectivity of the patients it shows that DHCP are frequently exposed to this pathogen. Collectively, these data elucidates the importance of appropriate precautions in avoiding the spread of TB during dental treatments.

Considerable risk of getting TB from patients lies on dental health care personnel (DHCP) due to their closeness to patients and they are constantly exposed to the respiratory secretions and oral secretions. In fact, DHCP and their patients share

the same air space for significantly longer time when compared to other clinical settings. Moreover, many dental procedures including dental scaling and restorative procedures induce clouds of droplets and aerosols immensely. One of the serious threats in dental aerosols is *M. tuberculosis* infection.[15] Centers for disease control (CDC) also has pointed out that the risk of TB cross transmission in dental clinics is higher when compared with other health care settings.[16] Therefore, it has been recommended that every oral health care facility should have a TB infection control plan as a part of its written infection control protocol. Suspected TB patients should be sent immediately for medical opinion and management prior to dental treatments. Emergency dental treatment should be performed in operating rooms where negative pressure and adequate air exchange systems are available.[17] Active TB poses a risk of infecting not only the DHCP but people in the waiting room as well. People with active TB must receive emergency dental care only in a hospital and should not receive elective dental care. If TB is inactive after proper medical treatments, elective dental care can be offered with no special considerations. Preprocedural mouthwash with chlorhexidine has shown to minimize the bacterial load in the aerosols significantly and would be used as a precaution in treating suspected patients.[18] Phenolic agents (5% phenol in water) are excellent disinfectants for TB bacilli containing sputum and secretions. Meanwhile, fresh household bleach (5% sodium hypochlorite) can also be used as a general disinfectant in the dental clinic and the laboratory. Importantly, ordinary surgical masks do not protect against TB infection because of bigger porosity. Harrel and Mollinary in an extensive review on aerosol and splatter formation and its relevance to infection control in dentistry suggested that use of universal barrier precautions, preprocedural oral rinse, rubber dam isolation and high volume evacuation would greatly help to prevent air borne infection transmission at the dental set up.[15] Similarly, proper ventilation and direct sunlight also reduce the existence of droplets and the bacteria in the environment.

Considering the current trends in *M. tuberculosis* diseases globally, CDC has laid down guidelines for prevention of TB in health care settings in 2005.[16] These guidelines focus on proper methods of patient screening and health care with safe working environment. Consequently, essential elements of an effective TB control plan include three levels of hierarchies; administrative, environmental and respiratory protection controls.[16]

Administrative control

The main administrative goals of TB control are early detection of TB infection and prompt isolation in order to prevent cross transmission. It is the most important level of TB control and intends to reduce the risk of exposure to patients who might have infectious TB. This applies to several aspects including TB exposure risk assessment, implementation of infection control program, identification of patients and provision of health education. TB exposure risk assessment should be an ongoing evaluation based on the data obtained from the patient population as well as local and state health departments. Infection control programs should aim to control generation of air borne droplet nuclei. It is important to maintain a high index of suspicion and rapid implementation of precautions to prevent bacterial transmission. Prompt identification of suspected patients, their isolation and proper referrals are part and partial in the administrative control of TB transmission. Ultimately, routine health education programs should include facts on TB transmission, control and appropriate training for both patients and the DHCP. Simultaneously, screening for TB infection of DHCP and post exposure management should be conducted regularly.

Environmental control

This entails physical or mechanical measures that are used to prevent the spread of droplet nuclei

in 1-5 ìm diameter in ambient air in the health care settings. Air borne infection isolation rooms (AII room), with negative pressure or operating rooms provided with air circulation through high efficiency particulate air (HEPA) filters will be effective in eliminating the infectious particles in the environment.

Respiratory protection controls

Use of respiratory equipment to control exposure will be helpful to prevent inhalation of droplet nuclei by the DHCP. For this purpose DHCP should wear disposable respirators when attending infectious TB patients with action disease. These respirators will act as air purifying apparatus and eliminate the infectious particles from the breathing air.

Recently, Porteous and Terezhalmy (2008) in an extensive survey of the literature described that the strategies to prevent and minimize the risk of TB transmission in the dental setup depends mainly on the standard precautions stipulated in CDC guidelines in 2003.[19] Yet, the latter precautions are inadequate to control the spread of organisms through droplet nuclei 1-5 ì m and additional measures and specific precautions regarding *M. tuberculosis* are now available in guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health care facilities issued by CDC in 2005.[16,20] These guidelines elaborate the importance of written infection control protocol to control TB in health care settings including dental clinics keeping with the existing trends in infectious diseases and available resources. Following are some important steps that would be helpful to minimize the risk of TB transmission within the general dental practice environment.

- Obtaining a proper and comprehensive medical history from each patient and continuously updating it in the consecutive meetings. Check for previous TB infection, signs and symptoms of active TB. Also enquire whether there is recent contact with TB patients.
- If the medical history is suggestive of active TB immediate referral for medical treatment is necessary. No dental treatments or procedures should be commenced prior to medical opinion and appropriate treatment for the active TB.
- Suspected patients should cover the airway and the mouth with a handkerchief or surgical mask when they are waiting at the dental clinic and the clinic should be spacious with adequate ventilation and sun light to eliminate infectious droplets.
- Elective dental procedures should be postponed until a patient is confirmed noninfectious. In case of providing urgent dental treatments for a person having active TB which is infectious, the procedures should be performed in a place where TB isolation facilities are available. Attending dental staff should use respiratory protection such as high efficiency particulate air (HEPA) filtration masks/respirators.
- Educate the DHCP and the patients regarding the orofacial manifestations of TB and review infection control policies and protocols regularly.

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Research Article I

Sri Lanka Dental Journal 2008; 38(01) 15-23

Root canal morphology of permanent incisors and canines in a Sri Lankan population

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Abstract

Objective: The main purpose of this study was to investigate the root canal morphology of permanent incisors and canines of Sri Lankans and to determine the affinities of these morphological variations with reference to people of European and Asian origin.

Material and methods: Two hundred and fifty nine maxillary and 170 mandibular permanent incisors and canines were examined. Root canal morphology (number and configuration) was studied using a clearing technique. The examination of root canal systems of the teeth were based on Vertucci's classification.

Results: Maxillary central and lateral incisors and canines typically presented with a single canal of type I canal configuration. In mandibular teeth, most of the central incisors showed type I (57.4%) canal configuration. Type III was found in 37.0% of the cases. The incidence of type III canal configuration in mandibular lateral incisors was 55.9%. The remainder was distributed mainly between type I and II. In addition, mandibular canine teeth typically presented with a single canal.

Conclusions: The root canal morphology in mandibular incisors and canines is variable in different population groups. Mandibular incisor and canine root canal morphology of Sri Lankan people have closer affinities with those of people of European than East Asian origin.

Introduction

The study of the anatomy of root canals has a clinical and an anthropological significance.[1,2,3,4,5,6,7] Many investigators have examined the root canal configurations of the human dentition using number of methods such as macroscopic sections, polyester resin casts, transparencies of previously stained samples and radiographs of extracted teeth.[8,9,10,11] These studies have shown differences in the shape and the number of root canals among different population groups and differences are reported to be genetically determined.[3,4,5,6,12] For example, Walker (1988) reported the occurrence of a second canal in the mandibular first and second incisors was infrequent in people of East Asian origin but more frequent in people of European origin.[4] This finding was later confirmed by Peiris (2008) who reported a lower incidence of two canals in mandibular incisors and canines in East Asian populations.[6]

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All the available studies have been mainly carried out in European, North American and East Asian populations. The only study available for South Asians is that of Peiris (2008) who described the root canal morphology of human permanent teeth in Sri Lankan and Japanese populations and concluded that the Sri Lankans have a higher incidence of two canals in mandibular incisors and canines than the Japanese.[6] However, the information regarding the variations of root canal morphology in different population groups (the human dentition) is insufficient from anthropological and clinical perspective.

Therefore, the present study was carried out to investigate the root canal morphology (configuration and number) of permanent incisors and canines of Sri Lankans and to compare the findings with those of European and Asian population groups.

Material and methods

Total of 259 maxillary (86 central, 87 lateral incisors and 86 canines) and 170 mandibular (54 central, 59 lateral incisors and 57 canines) permanent incisor and canine teeth were used in the study. All subjects enrolled in this research responded to an informed-consent protocol approved by the Ethical Committee of the Nihon University School of Dentistry at Matsudo which conforms to the provisions of the Declaration of Helsinki in 1995 (revised in Edinburgh 2000). Teeth were collected from patients who came for extractions due to reasons such as caries, periodontal diseases, prosthodontic and orthodontic treatments, at three dental hospitals in the central province of Sri Lanka from 2003 to 2005. Ethnic origin of the subjects was confirmed, analyzing the pedigrees up to three generations. Mixed parentage was excluded during the interview. All the subjects in the study were Sinhalese males and females and the age range of the sample was 17-79 years.

Teeth were washed in normal saline immediately after extraction and stored in 10% formalin until the collection was completed. Teeth were then boiled in 5% NaOH for 5 minutes and cleaned with 10% NaOCl for 40 minutes in a super sonic cleaner to remove organic debris. Deposits (calculus) and bone fragments were removed by scaling and polishing.

Injection of China ink into the canal system was done following vacuum injection protocol described by Yoshiuchi *et al.* (1972) for visualization of the root canal system.[13] Teeth were then cleaned with water to remove any stains on the surface and demineralized for five days in 5% Nitric acid at room temperature (The Nitric acid solution was changed regularly). Teeth were tested for softness by inserting a needle in the coronal region to assess the reliability of the demineralization. After demineralization, the teeth were rinsed in running water for 24 hours and then dehydrated using graded ethanol for 5 days. The teeth were rendered transparent by immersing in Benzene (24-48 hrs) first and then in a solution containing Bezoic acid mixed with Benzene and Methylsalycylate (1:5) for another 24-48 hrs.

The specimens were then examined under a dissecting microscope (Olympus, SZ 40) at ×10 magnification and the number and configuration of root canals were recorded. Classification described by Vertucci (1984) was used to grade the configuration of the root canals (Fig.1).[1] After studying different techniques, Vertucci (1984) and Omer *et al.* (2004) have reported that three-dimensional evaluation of root canal morphology by demineralization and staining is superior over other methods.[1,14]

Intra observer variation in the assessment of the root canal morphology was tested by re-examining 100 randomly selected mandibular central and lateral incisors and comparing results with the initial canal assessments (Mandibular incisors were selected as they showed the most variable canal morphologies). Concordance rate was 99%. JMP (Version 3 - SAS Institute) software was used to analyze the data. Pearson

X square test was applied to show the significance. P values of less than 0.05 were considered significant.

Results

Tables 1 gives the data regarding the root canal configuration in maxillary and mandiblular incisors and canines, studied using the classification given by Vertucci (1984).[1] Percentages of type I canal configuration in maxillary central (I^1) and lateral (I^2) incisors and canines were 100%, 96.5%, and 94.2%, respectively. In mandibular teeth, 57.4% of the central incisors (I_1) showed type I canal configuration while type III canal configuration was found in 37.0%. The mandibular lateral incisors (I_2) showed the type III canal configuration in 55.9% of teeth while type I canal configuration was found in 35.6% of teeth. Mandibular canines, too showed a type I canal configuration in 70.2% of teeth while type III and type II canal configurations were detected in 17.5% and 8.8% of the cases, respectively.

Table 2 gives the number of root canals found in permanent incisors and canines of the sample. It was evident that almost all Sri Lankan maxillary central and lateral incisors and canines had one root canal, whereas in the mandibular incisors and canines, it varied from one to two canals. Meanwhile, none of the mandibular and maxillary incisors and canines showed three root canals.

Tables 3,4,5 and 6 give the comparison of root canal morphology (number and configuration), studied in mandibular incisors and canines of seven different population groups namely American Caucasians, Turkish, Japanese, New Zealanders, Mexican, Israeli and Chinese with that of findings of the present study.[1,2,4,6,11,15,16,17]

Discussion

Previous studies have shown that there is a variation in the root canal morphology of mandibular incisor and canine teeth when compared with those of the maxilla.[1,2,5,6] Table 3, 4, 5 and 6 compare the root canal morphology, studied in mandibular incisors and canines of seven different population groups namely American Caucasians, Turkish, Japanese, New Zealanders, Mexican, Israeli and Chinese with that of findings of the present study.[1,2,4,6,11,15,16,17]

In Sri Lankans, the number of root canals in I_1 does not differ significantly from Turkish (X^2=3.08), American Caucasians (X^2=3.64), New Zealanders (X^2=3.92) and Israeli populations (X^2=2.58). Root canal number in I_2 in Sri Lankans is in agreement with Turkish (X^2=0.02) and New Zealanders (X^2=0.02). Nevertheless, our findings were not in agreement with those of American Caucasian (P<0.0001, X^2=29.30) and Israeli populations (P<0.002, X^2=8.90) (Table 3).[2,1,15,16]

Meanwhile, I_1 have shown highest prevalence of type I canal configuration in Turkish, American Caucasians and Sri Lankan population groups. In addition, there is no difference in the incidence of type III canal configuration between Sri Lankans and Turkish/American Caucasian population groups (Table 4). In the case of I_2, prevalence of type I canal configuration in Sri Lankans is in accordance with that of Tukish but not with American Caucasians. Type II and type III canal configurations are common in Turkish people while type III is the most abundant canal configuration in Sri Lankans (Table 5).

Comparison of root canal number in Sri Lankans with that of Mexicans indicates that there is a significant difference in the frequency of I_1 and I_2 having two canals (I_1; P<0.02, X^2=4.91, I_2; P<0.0001, X^2=30.94). It has been reported that Mexicans are predominantly Asian in origin (Mongoloid) with Caucasoid admixture.[3,11] Moreover, the number of canals in I_1 and I_2 in Japanese (I_1; P<0.0001, X^2=20.63, I_2; P<0.0001, X_2=16.83) and Chinese (I_1; P<0.001, X^2=10.05, I_2; P<0.0001, X^2=19.26) differs significantly from that of Sri Lankans (Table 3). This suggests that there is a variation in the root canal number in Asians. In addition, Japanese have shown a lower prevalence of type III and a higher prevalence

of type I canal configurations in I_1 and I_2 when compared with those of Sri Lankans (Table 4 and 5).

Number of canals in Sri Lankan mandibular canines are in agreement with those of American Caucasians (X^2=1.29) and Turkish (X^2=0.642).[1,2] Low prevalence (4.7%) of two canals in mandibular canines has been reported for Japanese population.[6] Further, the number of canals in mandibular canines in Japanese (P<0.0001, X^2=20.41) differs significantly from Sri Lankans (Table 3). It infers that there is a low frequency of occurrence of mandibular canines with two canals in East Asian populations.

Turkish, American Caucasian and Sri Lankan populations have shown highest incidence of type I canal configuration in mandibular canines. The occurrence of type II and III canal configurations is also comparatively higher in these population groups. In Japanese, almost all canine teeth, studied have shown type I canal configuration (Table 6).

The present comparison showed that the mandibular incisor and canine root canal morphology of Sri Lankan people have closer affinities with people of European origin than that of East Asian origin. It also shows that the root canal morphology is variable in different population groups. However, the studies on root canal morphology are very limited in different populations groups. As such, more studies in different population groups are needed to make a comprehensive comparison.

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Table 1. Root canal configuration in permanent incisors and canines in Sri Lankans

Canal configuration	Canal type	Maxillary			Mandibular		
		I^1 (n=86)	I^2 (n=87)	C (n=86)	I_1 (n=54)	I_2 (n=59)	C (n=57)
1	I	86(100)	84(96.5)	81(94.2)	31(57.4)	21(35.6)	40(70.2)
2-1	II	-	-	2(2.3)	1(1.9)	5(8.5)	5(8.8)
1-2-1	III	-	1(1.1)	2(2.3)	20(37.0)	33(55.9)	10(17.5)
2	IV	-	-	-	-	-	-
1-2	V	-	1(1.1)	-	2(3.7)	-	2(3.5)
2-1-2	VI	-	-	1(1.2)	-	-	-
1-2-1-2	VII	-	-	-	-	-	-
3	VIII	-	-	-	-	-	-
Additional canal types	A	-	1(1.1)	-	-	-	-

Figures in parentheses denotes percentages
I^1; maxillary central incisor, I^2; maxillary lateral incisor, I_1; mandibular central incisor, I_2; mandibular lateral incisor, C; canine

Table 2. Number of root canal in permanent incisors and canines in Sri Lankans

No of root canals	Maxillary			Mandibular		
	I^1 (n=86)	I^2 (n=87)	C (n=86)	I_1 (n=54)	I_2 (n=59)	C (n=57)
1	86(100)	84(96.5)	81(94.2)	31(57.4)	21(35.6)	40(70.2)
2	-	2(2.2)	5(5.8)	23(42.6)	38(64.4)	17(29.8)
3	-	-	-	-	-	-

Figures in parentheses denotes percentages
I^1; maxillary central incisor, I^2; maxillary lateral incisor, I_1; mandibular central incisor, I_2; mandibular lateral incisor, C; canine

Table 3. Number of root canals in mandibular incisors and canines in different population groups (%)

Population	One canal			Two canal		
	I_1	I_2	C	I_1	I_2	C
Turkish[2]	32.5 (200)	37.0 (200)	76.0 (200)	67.5 (200)	63.0 (200)	24.0 (200)
American Caucasian[1]	70.0 (100)	75.0 (100)	78.0 (100)	30.0 (100)	25.0 (100)	22.0 (100)
New Zealanders[15]	57.0 (172)	64.1 (114)	-	43.0 (172)	35.9 (114)	-
Israeli[16]	67.5 (400)	57.5 (400)	-	32.5 (400)	42.0 (400)	-
Chinese[4]	78.0 (100)	68.0 (100)	-	22.0 (100)	32.0 (100)	-
Japanese[17]	82.9 (615)	79.7 (526)	-	17.1 (615)	20.3 (526)	-
Japanese[6]	86.2 (94)	66.0 (100)	95.3 (107)	13.8 (94)	34.0 (100)	4.7 (107)
Mexican[11]	72.4 (179)	76.2 (184)	81.5 (187)	27.6 (179)	23.8 (184)	18.5 (187)
Sri Lankan[a]	57.4 (54)	35.6 (59)	70.2 (57)	42.6 (54)	64.4 (59)	29.8 (57)

a present study
Figures in parentheses denotes sample size
I^1; maxillary central incisor, I^2; maxillary lateral incisor, I_1; mandibular central incisor, I_2; mandibular lateral incisor, C; canine

Table 4 Inter population comparison of root canal configurations in mandibular first incisor (I_1).(%)

Population	N	Type I 1	Type II 2-1	Type III 1-2-1	Type IV 2	Type V 1-2	Type VI 2-1-2	Type VII 1-2-1-2	Type VIII 3
Turkish[2]	200	32.5	28.0	27.0	9.0	1.5	2.0	-	-
American Caucasian[1]	100	70.0	5.0	22.0	3.0	-	-	-	-
New Zealanders[14*]	-	-	-	-	-	-	-	-	-
Israeli[15*]	-	-	-	-	-	-	-	-	-
Chinese[4*]	-	-	-	-	-	-	-	-	-
Japanese[16*]	-	-	-	-	-	-	-	-	-
Japanese[6]	94	86.2	-	10.6	-	2.1	-	1.1	-
Mexican[11*]	-	-	-	-	-	-	-	-	-
Sri Lankan[a]	54	57.4	1.9	37.0	-	3.7	-	-	-

[a] Present study
[*] Data is not available for comparison

Table 5 Inter population comparison of root canal configurations in mandibular second incisor (I_2) (%)

Population	N	Type I 1	Type II 2-1	Type III 1-2-1	Type IV 2	Type V 1-2	Type VI 2-1-2	Type VII 1-2-1-2	Type VIII 3
Turkish[2]	200	37.0	26.5	26.0	10.5	-	-	-	-
American Caucasian[1]	100	75.0	5.0	18.0	2.0	-	-	-	-
New Zealanders[14*]	-	-	-	-	-	-	-	-	-
Israeli[15*]	-	-	-	-	-	-	-	-	-
Chinese[4*]	-	-	-	-	-	-	-	-	-
Japanese[16*]	-	-	-	-	-	-	-	-	-
Japanese[6]	100	66.0	-	30.0	-	3.0	-	1.0	-
Mexican[11*]	-	-	-	-	-	-	-	-	-
Sri Lankan[a]	59	35.6	5.8	55.9	-	-	-	-	-

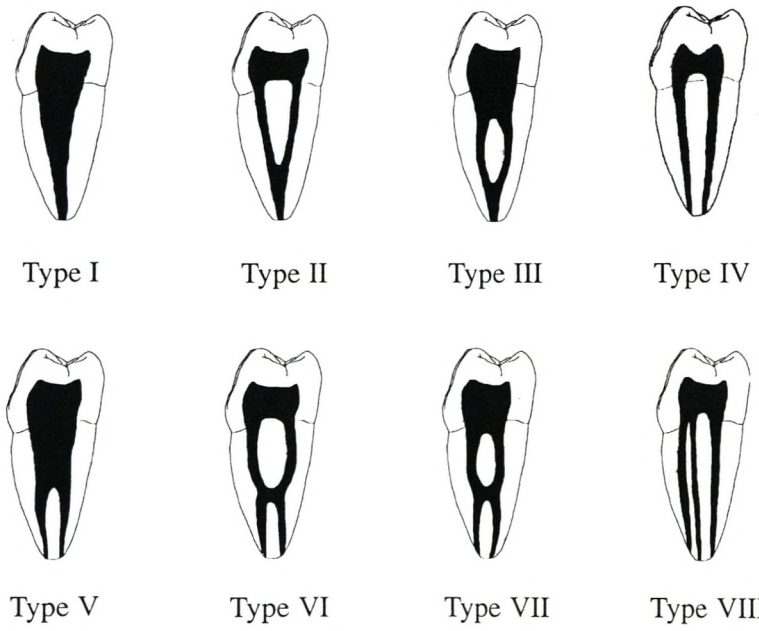
[a] Present study
[*] Data is not available for comparison

Table 6 Inter population comparison of root canal configurations in mandibular canines (%)

Population	N	Type I 1	Type II 2-1	Type III 1-2-1	Type IV 2	Type V 1-2	Type VI 2-1-2	Type VII 1-2-1-2	Type VIII 3
Turkish[2]	200	76.0	15.5	6.5	2.0	-	-	-	-
American Caucasian[1]	100	78.0	14.0	2.0	6.0	-	-	-	-
Japanese[6]	107	95.3	-	4.7	-	-	-	-	-
Mexican[11*]	-	-	-	-	-	-	-	-	-
Sri Lankan[a]	57	70.2	8.8	17.5		3.5	-	-	-

[a] Present study
* Data is not available for comparison

Figure 1 Vertucci's classification of root canal types.



Type I	–	One canal extending from the pulp chamber to the apex.
Type II	–	Two separate canals extending from the pulp chamber and joining to form one canal before the apex.
Type III	–	One canal leaving the pulp chamber, and dividing into two within the root, and merging to form one canal at the apex.
Type IV	–	Two separate and distinct canals extending from the pulp chamber to the apex.
Type V	–	One canal leaving the pulp chamber and dividing into two separate canals before the apex and opening separately at the apex.
Type VI	–	Two separate canals leaving the pulp chamber, merging within the the root, and re-dividing before the apex to exit as two canals.
Type VII	–	One canal leaving the pulp chamber, dividing and then rejoining within the body of the root, and finally leaving as two separate canals.
Type VIII	–	Three separate canals extends from the pulp chamber to the apex.

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Research Article II

Sri Lanka Dental Journal 2008; 38(01) 24-29

Evaluation of depth of cure and light intensity of visible light curing units used in Sri Lankan dental practices

U. U. K. P. C. Perera and K. A. Wettasinghe

Abstract

Objectives: The purpose of the study was to evaluate the efficiency of visible light curing units of Sri Lankan dental practices by measuring the intensity of light and depth of cure.

Material and methods: Total of 100 light curing units from 96 dental practices out of 9 randomly selected districts were included in the study. The light intensity of the curing unit and ambient light intensity of the clinic was measured using a digital visible light meter. Depth of cure was measured using light activated submicron hybrid composite shade A3, packed into a mould and cured for 20 seconds using the light source of the practice. Assessment of the awareness of the dental surgeon on issues pertaining to light curing unit and relevant other information was performed using a specially designed questionnaier.

Results: Thirty one percent of the curing units had a light intensity above 300 mW/cm^2 and a depth of cure of more than 3 mm. Thirty six percent showed an intensity ranging from 150 mW/cm^2 to 299 mW/cm^2 and a depth of cure between 2-3 mm. In the remaining 33%, the intensity was less than 149 mW/cm^2 and depth of cure was less than 2 mm. Fifteen units had an intensity of less than 100 mW/cm^2 and 7 of them were less than 50 mW/cm^2.

Conclusions: Approximately three fourth of the units tested were not functioning properly. Also there was substantial lack of awareness among the General Dental Practitioners of the need for maintenance and regular checking of the light intensity of their units and its significance.

Key words: Light cure, curing depth, light cure composit

Introduction

Visible light cured composite resins were introduced to use in dentistry more than three decades ago. Since then improvements in these materials and bonding techniques have resulted in their widespread use in dentistry, with expanded clinical applications.[1] Recent studies reveal that partial curing of the composite resins can cause water absorption and increase solubility, resulting in reduction of the hardness and eventual failure of the restoration.[2, 3]

The optimal output of a light curing unit significantly reduces with usage due to deterioration of the halogen bulb and the reflector. Also the filter can become inefficient due to blistering and cracking.[4] In wand type machines, fibers in the fiber-optic cord could break easily which may cause more reduction of light intensity than the gun type. However, gun type machines

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are widely used presently.[5] It has been shown that fluctuation of line voltage too reduces the intensity of light.[6,7] Procedural factors such as direction of light, size of curing tip, movement of tip, method of placement and increment addition, curing time, shade, translucency and the temperature of the material, distance from the tip of the light source to the surface of the material and post curing time also can cause reduction in polymerization.[8,9] It has also been documented that gluteraldehyde containing disinfectants damages the surface of glass rods of curing units, causing reduction in out put.[8]

Since composites are extremely technique sensitive, their success rate greatly depends on proper polymerization by a light curing unit with the correct wavelength and light intensity with adequate time of curing of proper increments.[10,11] If the material is not cured properly, it results in the failure of the restoration by causing sensitivity, non-vitality, secondary decay, discoloration and fracture of the tooth.[11,12,13]

A bright light may not necessarily be effective if it doesn't have the correct wave length for curing.[14] The use of a light meter is very important in assessing the light curing unit at regular intervals and particularly at the time of changing the bulb.[15] But in Sri Lankan practices it is not routinely done. Therefore, the quality of light cured composite resin restorations may reduce and become unpredictable.

The objective of the present study was to evaluate the efficiency of visible light curing units of Sri Lankan dental practices by measuring the light intensity and depth of cure.

Material and methods

Total of 96 dental clinics, which were convenient to visit for the investigator from 9 randomly selected districts (districts in the war area were excluded) were chosen for the study. The selected districts were Colombo, Gampaha, Kegalle, Kandy, Nuwara Eliya, Batticaloa, Ampara and Kalutara. All the selected clinics were visited by the chief investigator. Total of 100 units were included into the study and the units were only of halogen bulb type (LED type units were excluded from the study). The sample consisted of 23 different models from 12 manufactures and units were grouped according to their light delivery system. There were 3 units made in Sri Lanka.

After obtaining the consent, the questionnaire specially designed for the study was completed and both intensity of the light and the depth of cure of the units were measured. Intensity of the light was measured using CURE RITE™ Digital light meter (CAULK–DENTSPLY). In assessing the intensity, the ambient light intensity of the room was recorded using the light meter. After that the light unit of the clinic was switched on for 10 seconds and three consecutive readings were recorded thereafter. The mean out of three readings was taken as the light intensity of the particular unit. Measuring the depth of cure was done by using a mould which was prepared from an extracted human mandibular molar tooth with a proximal box cavity (class II) of 3x 4x 8 millimeters (mm) in size with parallel walls. The tooth was then split longitudinally into two halves using a microtome (Leica Inc.).

Composite resin (Spectrum Dentsply™ hybrid light activated composite resin, shade - A3) was packed into the mould (after assembling the two separated sections of the mould together) with the help of a teflon coated plastic filling instrument and a condenser. Excess was removed and a metal contoured matrix band was placed and tightened around the mould. A piece of transparent matrix strip was placed over the occlusal surface to limit the length of the composite block. Light curing was done vertically through the occlusal surface. Before curing, the unit was switched on for 10 seconds to optimize the intensity. Then the metal matrix strip and the transparent strip placed occlusally were removed. Two parts of the mould was separated and the composite block was taken

out and placed on a glass slab. By using a plastic spatula, uncured composite material was removed following the scraping method. The length of the remaining composite block was measured by a calibrated verneer caliper. The depth of cure is taken as half the length of the hard composite block.[3,16] The rating scale proposed by Rueggeberg *et al*, (1993) was used in the present study to assess the intensity of light (Table 1).[12,17]

Light intensity (mW/cm^2)	**Increment thickness (in mm)**	**Time (in seconds)**	**Rating**
300 and above	2 -3	20 - 30	Adequate
299-200	2-3	20 - 30	Need additional time
199 - 0	2-3	20 - 30	Inadequate even with increased time

Table 1. Rating scale for light intensity measurements, used in the present study

Results

Most of the units used in dental clinics were gun type (97 %) and a very few were wand type (3 %). Thirty one percent of the curing units had a light intensity above 300 mW/cm^2, and the depth of cure was more than 3 mm. Thirty six percent showed an intensity ranging from 150 mW/cm^2 to 299 mW/cm^2 with a depth of cure of 2.1–2.9 mm. In the remaining thirty three percent of units the intensity was between 100 mW/cm^2 to 149 mW/cm^2 and depth of cure was less than 2 mm. Fifteen units had an intensity between 50 mW/cm^2 to 99 mW/cm^2. Seven units gave the readings of less than 50 mW/cm^2,which was very low in intensity (Figures I, 2).

Bulbs specified by the manufactures for light curing units were not used by 18% of the dentists when replacing. Proper voltage and wattage were not found in 12% of the replaced bulbs. Frosting and whitening of the bulbs were observed in 22% of the units.

Discussion

It has generally been assumed by dentists that halogen lamps typically operate at near optimum performance throughout their lifetime. However, studies carried out on halogen lamps show that another 36 units out of 100 curing could be improved by increasing the curing time. Nonetheless long exposure time escalates the wear and wasting of the bulb. This may in turn reduce the intensity of the light further. Remaining 33 units were not functioning properly and found to be needed maintenance. Out of the total of 33, 15 units had an intensity of light between 50 mW/cm^2 to 99 mW/cm^2. Intensity of light in 7 (out of 33 malfunctioning units) was less than 50mW/cm^2 which is a very low intensity.

Most of the Sri Lankan dentists had never replaced the bulb in the unit until it is burnt and no proper maintenance of the unit had been carried out. The filters and cooling fans have not been checked routinely and in some units, the cooling fans were full of dust and not working properly. The bulbs specified by the manufacturer were not used by some of the dentists (18%) since they were expensive. These dentists have used to adapt only a locally purchase bulb to the reflector. This can cause a substantial reduction of the output. Also they have not been particular about the voltage and wattage when replacing the bulbs of their units (12%). In some units, deterioration of the reflector and blackening /whitening of the bulbs were observed (22%). The frequency of

bulb replacement was very difficult to asses accurately because of lack of records. Caughman *et al,* (1995) reported that most of the new units initially give an adequate intensity to polymerize a 2 mm thickness of resin composite.[17] Heavy usage also quickens the deterioration of the bulb.[18] Investigations done by Friedman *et al,* (1991) showed that lamp deteriorates with usage.[19] Many practitioners assumed that halogen bulbs used in visible light curing units produce a consistent output until the bulb burns out.[20] Findings of the present study also confirm this. Regular checking and cleaning/replacement of the bulb as well as the filter, cooling fan and the fiber optic bundle is necessary to obtain the best output. The tip of the fiber optic rod should be checked for unwanted composite build ups. Most of the Sri Lankan dentists seem to be satisfied with the performance of their light curing unit and use them for restorations even with very low intensities of light less than 100mW/cm^2. There was substantial lack of awareness among most of the general dental practitioners on the need for maintenance, regular checking of the light intensity of the units and its significance. They had no idea about the proper intensity but their only concern was about the blue colour of the light (wave length). They were unaware that the outputs of their lights were inadequate to cure composite resins properly. Also in Sri Lanka, facilities (light meters) are not freely available to check the light intensity of a curing unit in a general dental practice.

Conclusion

The light curing unit of a dental practice should be monitored regularly to ensure the adequate intensity and output. Best way of overcoming these barriers is adapting a standard protocol for the maintenance of light curing units. Given below is a standard protocol to be adapted by the general dental practitioners.

Protocol recommended for maintaining light cure units (Shortall A and Harrington E. 1996)

- Output of the unit should be tested (at regular intervals) routinely with a light meter.
- The bulb, filter and the reflector should be inspected, if there is a deterioration in the light output.
- Lamps may require replacement after heavy use, if output has decreased and bulb shows evidence of blackening or frosting.
- If the filter has blistered or cracked it should be replaced and it should be free of dust.
- The tip of the guide should be routinely inspected for composite build-up and the accumulated materials should be carefully removed and repolish the light guide tip.
- Broken fibers of the core should be checked (which may show as black or darkened areas) by holding the distal end of the light guide tip to day light.
- Vacuuming of the exhaust port is needed to clean dirt from the fan.
- Unit should not be switched off until the fan stops running.

It is also recommend that the quality assurance report of a general dental practice should include the above mentioned protocol.

Acknowledgements

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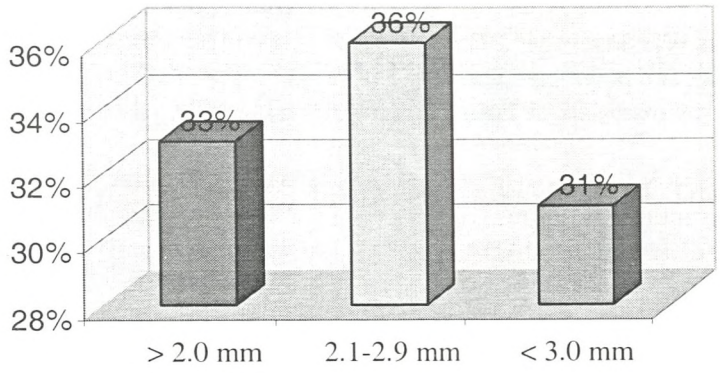

Figure 1. Depth of cure of the light curing units

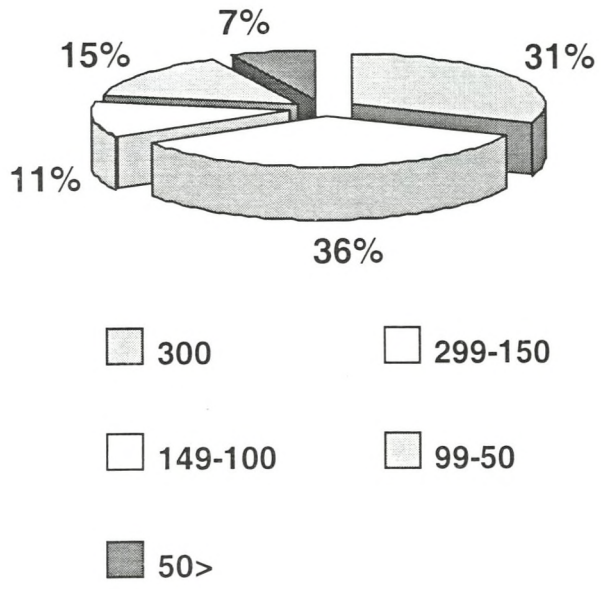

Figure 2. Distribution of light intensity of the units – (mW/cm^2)

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Research Article 111

Sri Lanka Dental Journal 2008; 38(01) 30-37

Bcl2 and Bax proteins expression in basaloid squamous cell carcinomas of the oral cavity

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Abstract

Objective: Basaloid squamous cell carcinoma (BSCC) is an uncommon variant of conventional squamous cell carcinoma (SCC) characterized by poor prognosis. Bcl2 and Bax are important anti apoptotic and pro apoptotic genes and deregulation of which may contribute to aggressive behaviour of the tumour. The aim of the study was to compare the expression of Bcl2 and Bax protein expressions in BSCC and SCC.

Material and methods: Nine oral BSCCs diagnosed at the department of oral pathology and nine age and site matched conventional oral SCC obtained from the archives were selected for the study. Immunohistochemistry was used to evaluate Bcl-2 and Bax protein expressions in the tumour samples.

Results: The Bcl2 protein was expressed in 55% of BSCC (5/9) and 33% of conventional SCC (3/9), while Bax protein was expressed in 22% of BSCC (2/9) and 33% of conventional SCC (3/9). Thus Bcl2 expression was found significantly more often in oral BSCC compared to conventional SCC ($p<0.001$), while there was no statistically significant difference in the Bax expression between the two groups of tumours. In addition, no correlation was found between the positive nodal status and Bcl2 or Bax positivity in BSCC or conventional SCC of the oral cavity.

Conclusion: In conclusion, the present study reveals that relatively higher numbers of BSCC express Bcl2 protein as reported previously for BSCC of other sites. However, apoptosis in BSCC may not occur as a result of pro-apoptotic gene Bax expression as relatively lower number of BSCC express this protein compared to conventional SCC.

Key words: Basaloid squamous cell carcinoma, apoptotic regulators, Bcl-2 and Bax genes.

Introduction

Basaloid squamous cell carcinoma (BSCC) is a poorly differentiated variant of squamous cell carcinoma, reported in 1986, for the first time. It

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predominantly occurs in the upper aero-digestive tract in the regions of oral cavity, hypo pharynx and larynx.[1] Histopathologically BSCC is defined as invasive carcinoma composed of closely packed cells with hyperchromatic nuclei and scanty cytoplasm, with foci of comedo-type necrosis within the tumour cell clusters.

Information available in the literature regarding the aggressiveness and outcome of BSSC of the upper aero-digestive tract is controversial compared to the conventional SCC. As such, there are reports to indicate poorer prognosis for BSCC compared to conventional SCC and also reports to indicate that there is no such difference regarding the aggressiveness and outcome between the two tumours.[2,3,4]

Deregulation of genes involved in apoptosis has been associated with tumour development and progression. The Bcl2 gene has been shown to prolong the cell survival by inhibiting apoptosis while Bax gene promotes apoptosis. Therefore, analysis of the function of Bcl2 and Bax proteins as a repressor and an effector in the molecular pathway of apoptosis may help us to determine the occurrence of lymph node metastasis and thereby the aggressiveness of BSCC compared to conventional SCC. Eventhough, there are reports available with reference to apoptosis regulator genes for BSCC of oesophagus and elsewhere, studies that exclusively deal with BSCC of the oral cavity are limited.[5,7] In addition, the inadequate sample size of previous studies involving BSCC excludes the possibility of meaningful comparisons between the two tumours.[5,7] The aim of the study was to compare the Bcl2 and Bax expression immunohistochemically in nine BSCC and nine age and site matched conventional SCC of the oral cavity.

Material and methods

The current study used a panel of nine BSCC of the oral cavity and nine age and site matched conventional SCCs retrieved from the archives of the Department of Oral Pathology, Faculty of Dental Sciences, University of Peradeniya, Sri Lanka. The carcinomas were resected between 2001 and 2005. The distribution of the clinical features of BSCC and conventional SCC used in the study is summarized in Table1.

Immunohistochemical staining

Formalin fixed, paraffin- embedded BSCC and conventional SCC tissue samples were used for Bcl2 and Bax protein detection. Four µm thick sections were taken and mounted on silane coated slides and were subjected to deparaffinization with xylene and rehydration through an ethanol series. Microwave antigen retrieval was carried out by placing the slides in 10 millimol sodium citrate buffer in a microwave oven at 800 W for 10 minutes.

Immunostaining was performed using Bcl2 specific monoclonal antibody (Clone 124, Dako, Denmark) and Bax specific polyclonal antibody (P-19, Santa Cruz biotechnology, USA). The sections were visualized by adding 3-3'diaminobenzidine (0.05% DAB) and counter stained with Gills Haematoxylin. Bcl2 positive lymphoma tissue was used as the external positive control while positively stained lymphocytes present within the tumour tissue were considered as the internal positive control.

A minimum of 1000 cells from the positively stained area were selected and scored. Bcl2 and Bax expression was estimated semi quantitatively as a percentage of positive cells. Each sample was assigned to one of the following categories depending on the number of positive cells; 10-30% cancer cells stained = 1+, 31-50% cancer cells stained = 2+, >50% cancer cells stained =3+ and <10% cancer cells stained considered as negative (0).[8]

Eventhough, initially Bcl2 and Bax positivity was evaluated in four categories, the lesions were divided in to two groups i.e., Bcl2 and Bax positive and negative tumours due to small sample size

for the statistical analysis. The statistical analysis was performed with Chi square test ($p<0.05$).

Results

Five out of nine BSCC (55%) showed cytoplasmic expression of Bcl2 (Fig 1). Remaining four lesions were considered as negative for Bcl2 (Fig 2). Out of the four negative lesions one BSCC was completely negative for Bcl2 while three lesions had less than 5% of positively stained cells. Areas containing both basaloid cells as well as squamous cells showed haphazard Bcl2 over expression. However, most of the positive basaloid cells were not closely situated to areas of comedo type necrosis.

In contrast, three out of nine conventional SCCs (33%) showed cytoplasmic immunoreactivity for Bcl2. All the positive tumours had a staining in the category of 1+ (10%-30% positive cells). Therefore, upon comparison of Bcl2 expression more BSCCs tend to express Bc12 compared to conventional SCC.

With regard to Bax protein, 22% of BSCC (2/9) and 33% of conventional SCC (3/9) expressed Bax protein. Most of the Bax positive tumour cells were also distributed in a haphazard manner in BSCC. All the positive tumours in both groups of carcinomas demonstrated category 1+ staining with 10–30 % of tumour cells positivity. Upon comparison of Bax expression in BSCC and conventional SCC a relationship between the two groups were not observed. In addition, 22% of BSCC and 11% of conventional SCC expressed both proteins.

Data available following excision of the tumours with block dissections indicated lymph node metastasis in three BSCC patients and two conventional SCC patients. However, a relationship between nodal positivity and Bc12/ Bax expression was not obeserved in the two groups of tumours (Table 2).

Discussion

Even though, there is information regarding regulation of apoptosis for BSCC of other sites, studies that deal with the expression of apoptosis associated proteins in BSCC of oral cavity is limited.[5,7] The lack of information can be attributed to the fact that oral BSCC is relatively rare and also due to BSCC been a recently recognized variant compared to conventional SCC. Therefore, the current study was undertaken to gain an insight into expression of apoptosis associated proteins in BSCC of the oral cavity.

According to the results of the present study expression of Bcl2 protein is more frequent in BSCC compared to conventional SCC of the oral cavity. Similar results have been reported for BSCC of the oesophagus when compared to conventional SCC.[9,10] With regard to Bcl2 expressions in conventional SCC, contradictory reports exist in literature with some showing enhanced Bcl2 expression and others with reduced expression.[11,12,13,14] These contradictory reports have been attributed to the stage of disease. i.e. carcinogenic role of Bcl2 gene has been shown to be limited to the early stages of oral carcinogenesis.[15] Bcl2 expression reported in several previous studies do not significantly differ from our findings as 33% of conventional SCC of the present study expressed Bcl2.[8,9]

No statistically significant differences were observed for Bax expression in the two groups of tumours. Review of literature revealed that there is only one report avalable on Bax expression with a series of 17 BSCC of the oral cavity.[4] However, contrast to the findings of the present study, they have demonstrated 88% of BSCC with strong immuno-reactivity to Bax protein. This difference in the Bax expression in the two studies may reflect the differences in the molecular aetiopathology depending on the geographic variations.[4]

The Bax expression rates reported for conventional SCC in previous studies are similar to our results.[16] In addition, no correlation was found between the positive nodal status and Bcl2 or Bax expression in oral BSCC or in conventional SCC. In contrast, there are previous reports that have correlated Bcl2, expression in head and neck SCC with nodal negativity or nodal positivity.[8,17] The lack of correlation of OSCC in our study can be attributed to low sample size. Only nine conventional SCC were analyzed in the present study, as out of the 15 BSCC available in our archives, adequate tissue and patient's information were available for only nine BSCC.

Low sample size and use of only one set of anti-apoptotic and pro apoptotic antibodies can be considered as limitation of the present study. In addition, molecular pathological profile of OSCC from South East Asia significantly differs from OSCC of developed countries. Therefore, further studies from all parts of the world may be required to complete the molecular pathological profile of apoptosis related gene expression in oral BSCC.

The simultaneous finding of frequent apoptosis and strong expression of anti apoptotic Bcl2 reported in previous studies have been attributed to the activation of proliferation promoting proto-oncogenes particularly c-myc.[10] Therefore, a study is planned to evaluate c-myc expression in BSCC of oral cavity in the future. This will help to determine whether the molecular profile of BSCC of oral cavity is similar to BSCC of the oesophagus.

Finally, when considering the BSCC of the oral cavity used for the present study, there was no significant difference in the number of nodal positive tumours between oral BSCC and age and site matched conventional SCC. Therefore, this fact may support the findings of previous studies from other sites where they have concluded that aggressiveness of BSCC of the head and neck does not differ from that of conventional SCC.[2,3] In conclusion, the present study confirms the fact that relatively higher numbers of oral BSCC express Bcl2 protein compared to conventional squamous cell carcinoma similar to the findings of other sites. However, frequent apoptosis reported for BSCC in previous studies may not occur as a result of pro apoptotic gene Bax expression as relatively lower number of BSCC express this protein compared to conventional SCC. Further, we suggest that increased expression of Bcl2 may play a role in increased survival of tumor cells in BSCC.

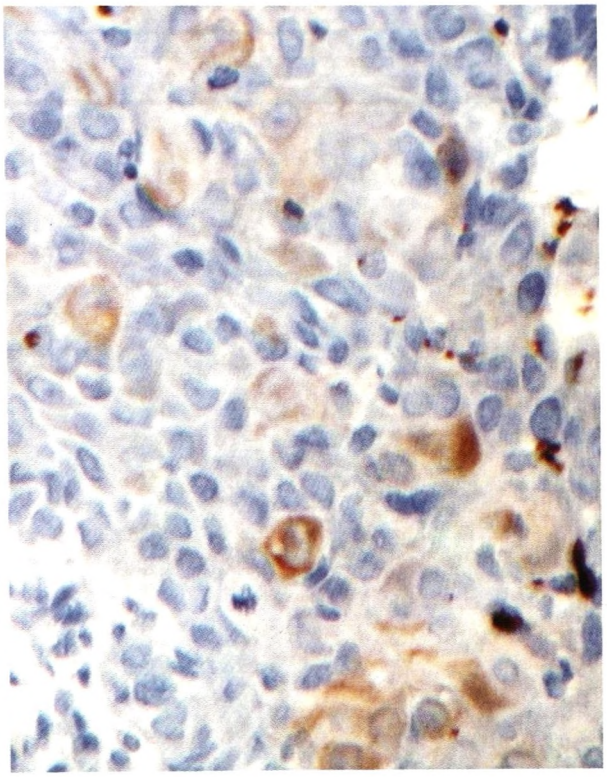

Figure 1 Photomicrograph shows a basaloid squamous cell carcinoma positive for Bcl2. Note: cytoplasmic staining of tumour cells (x20).

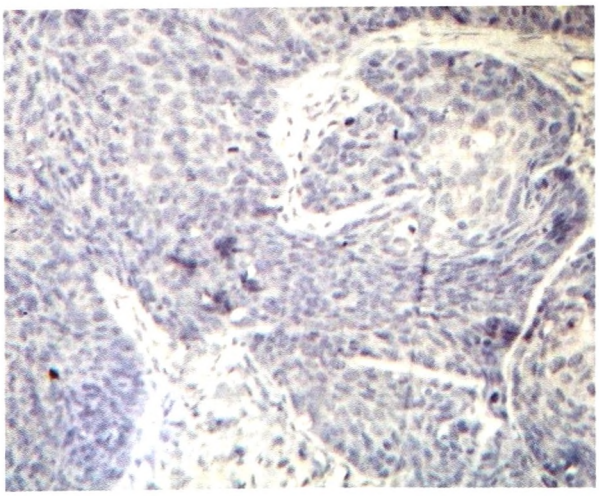

Figure 2 Photomicrograph shows a basaloid squamous cell carcinoma negative for Bcl2 (x10).

Table 1. Characteristics of the sample

Clinical features	BSCC	C.SCC
Age		
<50	1	1
>50	8	8
Gender		
Male	9	9
Female	0	0
Site		
Tongue	1	1
Floor of the mouth	2	2
Retromolar region	3	3
Alveolous	2	2
Soft palate	1	1
Size		
T1-T2	0	0
T3-T4	9	9
LN metastasis		
Present	3	2
Absent	6	7

Table 2. Bcl2 and Bax expression vs lymphnode metastasis

Bcl2 / Bax expression	BSCC		Conv. SCC		Chi square
	LN+	LN-	LN+	LN-	
Bcl2 Positive	1	4	1	2	P = 0.40
Negative	2	2	4	5	
Bax Positive					
Negative	1	1	0	2	P = 0.50
	2	5	2	5	

LN + = lymphnode metastasis present
LN - = lymphnode metastasis absent

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Case Report

Sri Lanka Dental Journal 2008; 38(01) 38-47

Dentin dysplasia type I – typical and atypical presentations

A. Ariyawardana and C. Herath

Abstract

This paper presents two cases of dentin dysplasia type I: one case with typical and the other with atypical presentation. Dentin dysplasia is a rare hereditary disorder of dentin formation, which is inherited as an autosomal dominant trait. Dentin dysplasia type I is characterized by clinically normal teeth, which exfoliate spontaneously or due to minor trauma. This condition undoubtedly impedes the physical and psychological well-being of the affected individual. Moreover, management of this condition poses serious difficulties for the dentist. Clinical and radiological features, theories of pathogenesis and the avenues of management are described in this paper.

Key words: Dentin dysplasia, developmental, aesthetics

Introduction

Inherited dentin defects that exclusively affect dentin have been divided into two main categories namely dentinogenesis imperfecta (DGI) types I-III and dentin dysplasia (DD). They share many features in common.[1,2] Dentin dysplasia (DD) is a rare hereditary disorder of dentin formation with a prevalence of 1 in 100,000.[2] This disorder is inherited as an autosomal dominant trait affecting both the deciduous and permanent dentitions.[3] DD was first described by Ballschmiede (1920) and the term "dentin dysplasia" was coined by Rushton in 1939.[4,5]

Witkop in 1972 has classified DD into two types: radicular DD as type I (DD I) and coronal DD as type II (DD II). In type I both the deciduous and permanent dentitions are affected.[3] The crowns of teeth are morphologically normal and the first sign of the disease is premature exfoliation due to minor trauma or even spontaneously.[6,9] The affected teeth show normal enamel and atypical dentin deposition resulting obliteration of the pulp chambers. The roots of the teeth are short, blunt or pointed with radicular cysts and periapcal abscesses without an obvious cause. Radiaologically DD type I is characterized by complete or partial obliteration of the pulp chamber, defective root formation with shortened and blunt roots and periapical radiolucencies.[8]

Based on the radiographic appearance, O'Carroll *et al,* (1991)[10] have proposed a classification for DD type I. They are type I-a to type I-d. Type I-a is characterized by complete obliteration of the pulp chambers, no root development and radiolucencies in periapical areas. In type I-b horizontal crescent shaped radiolucent pulpal remnants with few millimeters of root development and many periapical radiolucencies are seen. Type I-c is characterized by two horizontal crescent shaped radiolucent lines

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concave towards each other at cemento-enamel junction and significant but incomplete root development with or without periapical radiolucencies. Type I-d has root canals of normal length but may be bulbous in the coronal third where pulp stones are developed within the root canal.

DD type II, shows a distinct clinical and radiological appearance compared to DD type I. In DD type II deciduous teeth show a blue-to-amber-to-brown colour similar to that of dentinogenesis imperfecta (DGI). Radiologically teeth exhibit bulbous crowns, thin roots, cervical constriction and early obliteration of the pulp. Permanent teeth are morphologically normal but radiologically exhibit thistle shaped pulp chambers.[11] Most of the teeth show accumulation of pulp stones.[4] Moreover, no multiple periapical radiolucencies have been reported like in DD type I.[9]

Case I

An eighteen-year-old girl was referred to the Oral Medicine clinic, Dental Hospital (Teaching) Peradeniya, Sri Lanka for further management of mobile lower front teeth. She found difficulties in incising hard food due to the mobility of front teeth. She also experienced transient gingival swellings with discharge. Lower left central incisor had spontaneously exfoliated, few months ago. She did not give a history of any general medical condition.

The patient looked healthy. There were no extra oral swellings or cervical lymphadenopathy. Intra-oral examination revealed the presence of all teeth except lower third molars of both sides, upper right third molar and lower left central incisor. All teeth were caries free. An anterior open bite was noted with slight bimaxillary proclination (Fig.1). All anterior teeth were mobile and gingival recession was noted in relation to all the canine teeth on the labial aspect. Her oral hygiene was satisfactory and there were no soft tissue abnormalities.

The father of the proband stated that all his teeth had spontaneously exfoliated by the age of 20 years. He also indicated that none of his three sisters and six brothers was affected. The mother of the patient revealed no family history and she had all her teeth intact. The patient had one sibling: a 16 year old brother and on examination of his mouth it was revealed the presence of all permanent teeth except the third molars. There were no evidence of any clinical abnormality.

Radiological examination of the proband revealed rootless and pulpless teeth (Fig. 2). There were many radiolucencies in relation to root "stubs". The right mandibular third molar tooth was unerupted. Full blood count, an analysis of serum calcium, serum phosphorous and alkaline phosphatase levels were done and the results were found to be well within normal limits. Radiological examination of the sibling (this was done after obtaining informed consent) revealed no abnormality.

Clinical and radiographic findings were consistent with that of dentin dysplasia type I. The condition and its nature were explained to the patient and the parents. Dietary advice and instructions to maintain meticulous oral hygiene were given in order to prevent premature loss of teeth due to caries and periodontal disease. Furthermore, the patient was advised to prevent trauma to teeth especially when biting hard food. The patient was asked to attend for review regularly.

Case II

A four-year old girl was brought to the Paedodontic clinic, Dental Hospital (Teaching) Peradeniya, Sri Lanka with a complaint of abnormally shaped mobile teeth. According to the parents, the child had a history of delayed eruption and premature loss of some primary teeth. She had experienced intermittent pain in relation to both maxillary and mandibular teeth in the recent past. Her medical history was non-contributory. The patient was the only child and parents

revealed no family history of a disease of this nature.

Intra-oral examination revealed the presence of first and second molars of the primary dentition, permanent central incisors, right lateral incisor and first molars of both sides of the maxilla. In the lower arch, first permanent molar teeth were present. There were dentinal caries on all deciduous molars and upper permanent molars and they were restored.

Orthopantomogram (OPG) taken at this stage revealed the presence of developing permanent canine to second molar teeth in all quadrants of both jaws. It was also evident that the teeth had blunt and short roots some with obliterated pulp chambers (Fig 3). Based on these findings a tentative clinical diagnosis of dentin dysplasia of unusual form was made. The patient kept under review. Six months later the patient complained some discomfort in relation to the extremely mobile left central incisor.

On review at the age of 5 years it was noted that right lateral and left central incisors had exfoliated. At a subsequent visit when the patient was 6 years old it was noted that the maxillary first and second molars of the deciduous dentition were present. In addition, maxillary and mandibular first molars of permanent dentition were also present (Fig 4, 5). At this stage parents demanded for partial dentures to improve mastication and aesthetics. As the occlusal vertical dimension was not sufficient to insert dentures, stainless steel crowns were placed on all first permanent molars. Upper and lower partial dentures were provided and the patient was reviewed periodically (Fig 6). The dentures were adjusted according to the growth and eruption of teeth. At the age of 8 years it was noted that the second molars were erupting. At the age of 10 years it was noted that premolars also had erupted.

Discussion

In this paper two cases of DD type I: one with typical and other with atypical features of dentin dysplasia were described. The etiology of dentin dysplasia is not yet clearly elucidated though various theories have been put forward. Log an *et al.*, (1962) proposed degeneration and abnormal calcification of dental papilla leading to retarded growth and obliteration of the pulp space.[12] Later it was argued that invagination of the epithelial root sheath leads to deposition of abnormal dentine in the dental papilla.[13] Disagreeing with this theory Wesley *et al*, (1976) proposed an abnormal interaction of odontoblasts with ameloblast layer leading to abnormal differentiation or function of odontoblasts leading to abnormal deposition of dentin.[14] Witkop in 1971 has commented that atypical tubular and osteodentin in pulp chambers of the DD type I may have originated in developing dental papillae.[15] The knowledge of molecular etiology of human dentinal diseases has improved substantially over the past few years. The critical loci for DD-II, DGI-II, and the association of DGI-II to human chromosome 4q21 have been identified. Although a cluster of dentin/bone genes that includes osteopontin, bone sialoprotein, matrix extracellular phosphoglycoprotein, dentin matrix protein 1 and dentin sialophosphoprotein (DSPP) that are located in the common loci for these diseases have been identified, only mutations within dentin sialophosphoprotein gene *DSPP* (4q21.3) have been found to be associated with the development of DGI types-II and III and DD-II. [16] However, the molecular defect in DD type I remains unknown.[17]

The case number one shows typical clinical and radiological characteristics of dentin dysplasia type I. In type I, the pulp chambers are rapidly obliterated by the deposition of irregular masses of bizarre tubular dentin. The roots are significantly shortened that leads to premature exfoliation.[14] The presence of periapical radiolucencies without obvious cause is characteristic for this case.[7] The present typical

case of dentin dysplasia showed autosomal dominant characteristics too.

Although case II had several characteristic features of dentin dysplasia type I, there were several unusual features. Premature eruption and exfoliation of deciduous and permanent teeth were unique features in this case. Özer *et al.*, in 2004 reported two atypical cases of dentin dysplasia type I with delayed eruption of both upper and lower incisors.[18] The delay in eruption can be explained on the basis that the development of root is essential for eruption. Nevertheless, not many cases of dentin dysplasia with delayed eruption have been reported.[6]

The main clinical problem in dentin dysplasia type I is severe mobility and premature exfoliation of teeth. Periapical radiolucencies and granulomas could result from pulp necrosis or even spontaneously. [19] Close proximity of the vascular channels to the dentino-enamel junction may increase the risk of traumatic pulp exposure during cavity preparation. Therefore, preventive dental care is of great importance. Furthermore, periodontal diseases occurring in these patients may lead to rapid loss of attachment rendering teeth exfoliate prematurely.

DD type II was first described in detail by Shields *et al.*, in 1973 and it is virtually identical to DGI type II in the primary dentition with yellow-brown to blue-gray discoloration and pulpal oblietration.[7] In contrast, permanent teeth affected by DD II are normal in shape and colour or minimally discolored.

Little is known about the specific treatment of dentin dysplasia. The fundamental clinical problems in DD type I are periapical bone destruction and necrosis of the pulp leading to mobility and premature exfoliation of affected teeth. Periapical granulomas and cysts are the result of pulpal necrosis which could result from caries or even spontaneously. Close proximity of the pulpal vascular channels to the amelo-dentinal junction may result in traumatic exposure of the pulp during cavity preparation. Therefore, preventive approaches are imperative for these patients. Periodontal disease also can result in loss of attachment further compromising the crown root ratio. Hence, maintenance of meticulous oral hygiene is fundamentally important.[19]

Extraction is an option for those teeth with pulp necrosis and periapical pathology.[4, 20] Coke *et al.*, 1979 has attempted surgical endodontic therapy with retrograde amalgam filling in a DD type I case.[9] However, long term follow up was not available to evaluate the success of this modality. Ravanshad and Khayat (2006) have reported a case of DD type I that was followed up for four years after conventional endodontic therapy of multiple teeth and claimed to have achieved successful results.[21]

Malalignment of teeth is a common complaint in this disorder. However, routine orthodontic treatment should not be considered as short roots will not be able to withstand the forces applied in orthodontic tooth movement.[6]

Masticatory function and the facial appearance are badly affected when several teeth are exfoliated in a growing child. This could eventually deteriorate the physical and psychological well-being. Therefore, it is of utmost importance to rehabilitate the dentition if teeth are exfoliated. For the patient reported in case II, partial dentures were provided to maintain the function and aesthetics. Provision of dentures needs very close monitoring in a growing child as the denture becomes ill-fitting due to the rapid changes in growth. When the existing denture is no longer fitting it should be modified or new dentures should be provided accordingly.

Osseo-integrated implants are invaluable in restoring the function and aesthetics. However, atrophy of maxillary and mandibular bones is a major constraint in inserting the implants. Munoz-

Guerra *et al.*, (2006) reported a successfully treated case of DD type I, using onlay autogenous bone grafting and a sinus lift technique to overcome this problem.[22]

More clinical studies with long term follow up are needed to understand the pathogenesis, natural history and proper management strategies of DD I. Since there is no well established treatment to date, it is important to monitor patients carefully primarily with the view to prevent premature exfoliation of teeth.

What this paper adds?

This paper adds two more cases of dentin dysplasia to the literature. The paper describes a case of typical dentinal dysplasia type I and a case with unusual presentation. The paper includes a discussion on the current understanding of the aetiopathogenesis and treatment options available.

Why this paper is relevant to paediatric dentists?

Tooth mobility, periapical pathology and premature exfoliation of either primary or permanent dentition are the commonest presenting complaints of patients affected by dentin dysplasia type I. It poses serious difficulties in the management especially in maintaining aesthetics and function. As there is no satisfactory treatment to date it is of utmost importance to prevent premature loss of teeth by maintenance of good oral hygiene. Although rare, it is extremely important for the paediatric dentists to be aware of this condition.

Acknowledgements

We would like to thank, Ms. Damayanthi Ranaweera (nurse in-charge) and Ms. Vishaka Nayakarathne (nursing officer) of Oral Medicine clinic for their assistance in managing patients.

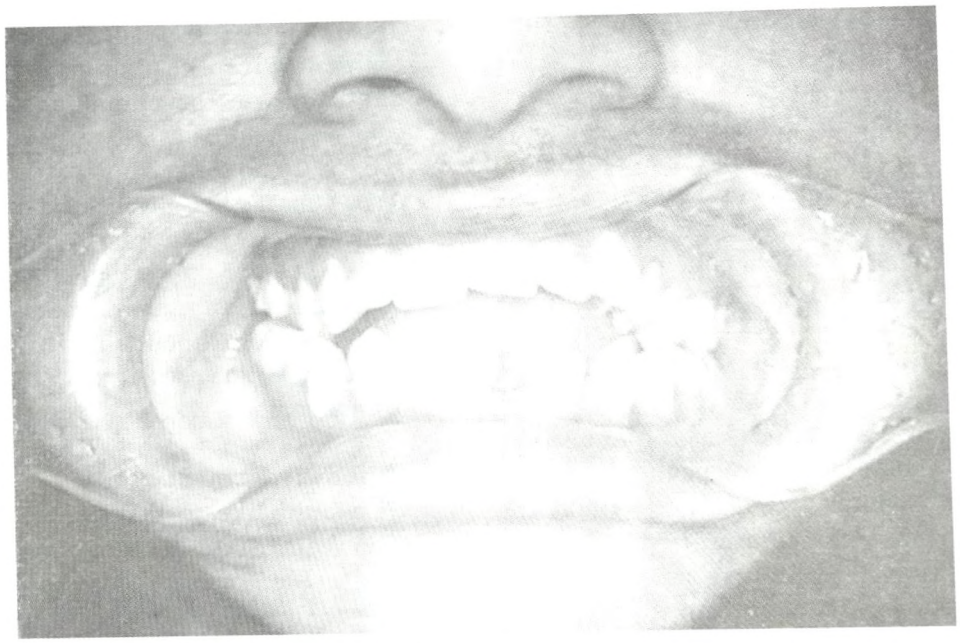

Figure 1. Photograph of the case I shows anterior open bite and bimaxillary proclination

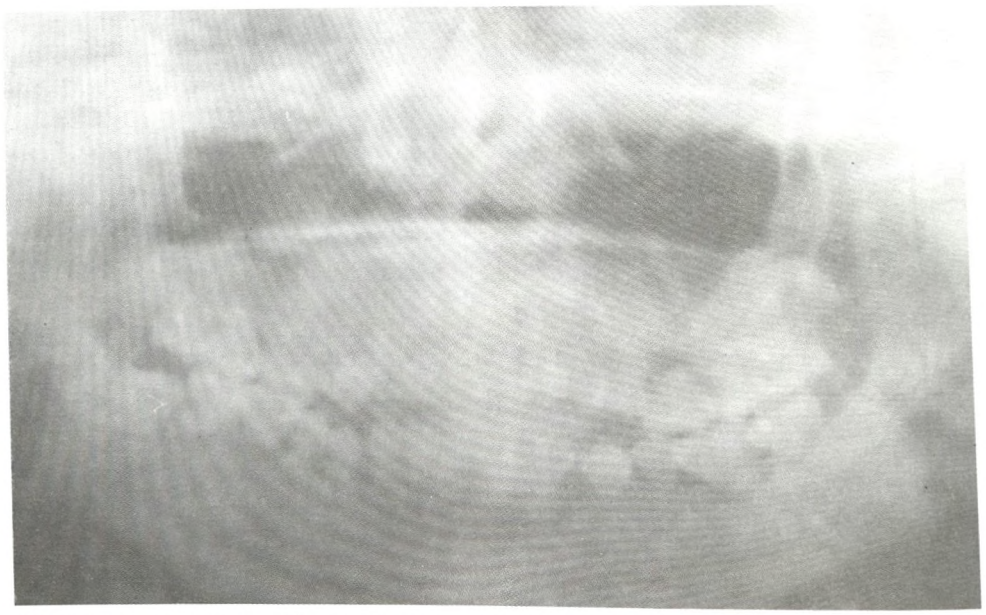

Figure 2. Dental orthopantomogram of case I shows rootless teeth with obliterated pulp chambers and multiple radiolucencies associated with root stubs. These features are characteristics of dentin dysplasia type I

Figure 3. Case II, orthopantomogram of the patient at the first visit.

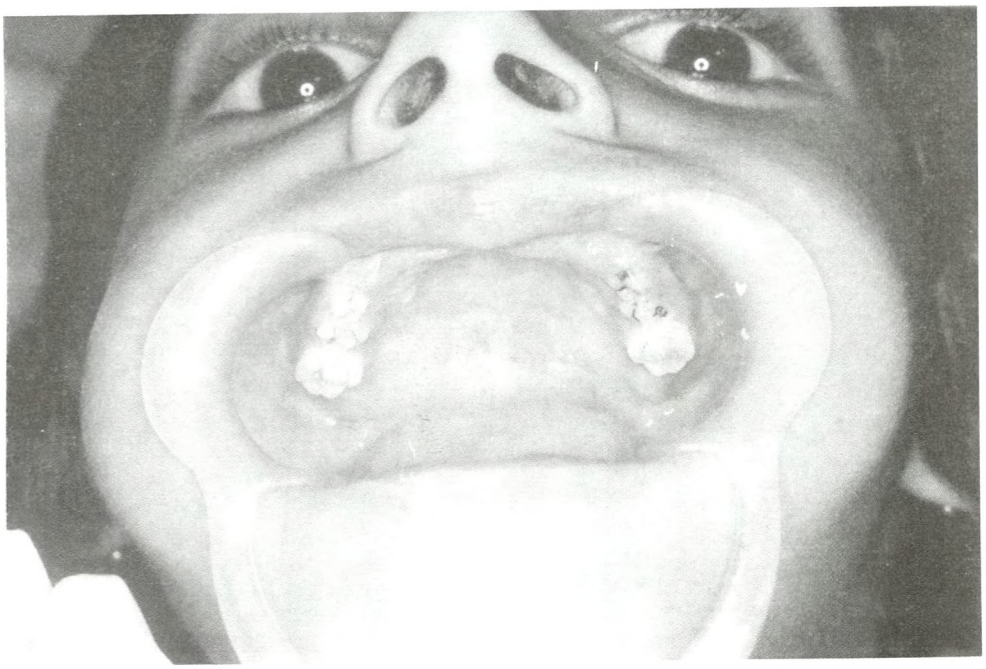

Figure 4. Case II, upper arch at the age of 6 years.

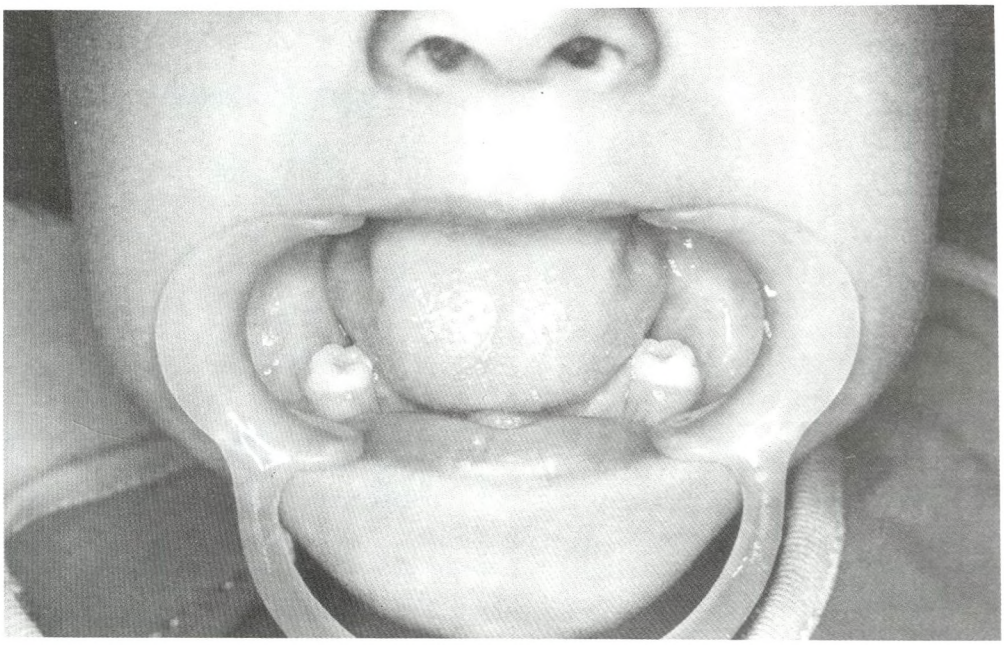

Figure 5. Case II, lower arch at the age of 6 years. Note the presence of only first molars of the permanent dentition.

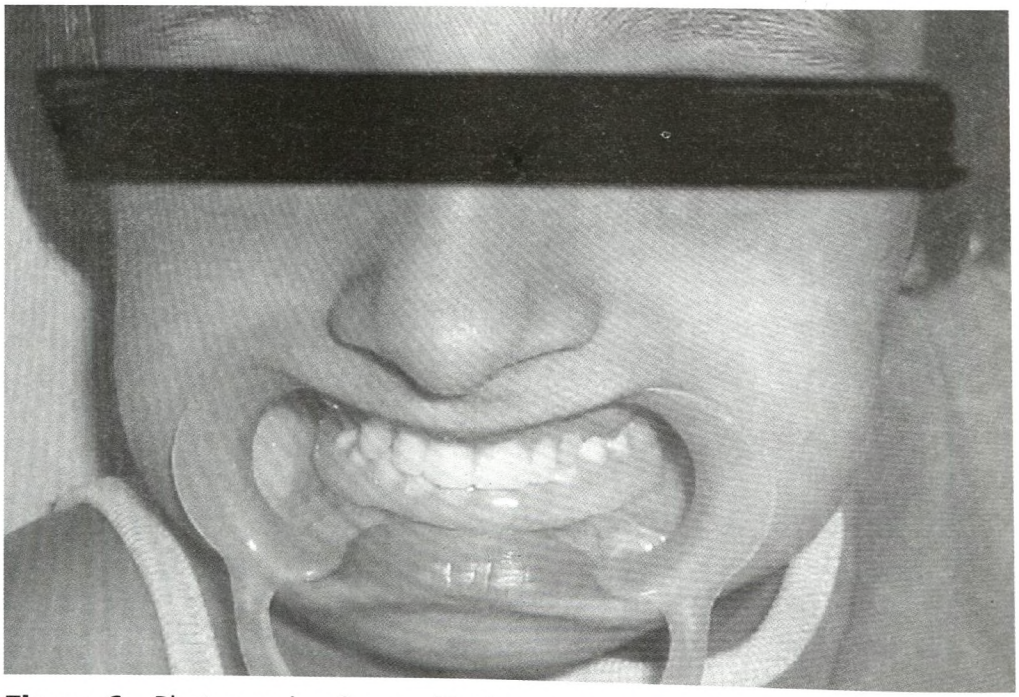

Figure 6. Photograph of case II shows upper and lower acrylic partial dentures which improved the aesthetics and function.

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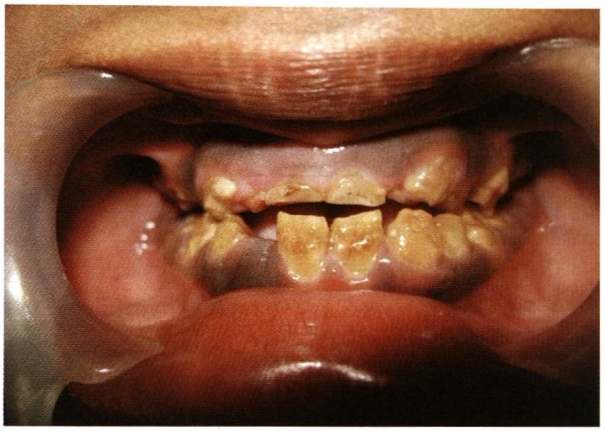
Self Assessment - Oral Diagnosis (SAOD)



This is a picture of a 12 year old girl with a complaint of discoloured teeth. Examination revealed the presence of all permanent teeth with overretained primary maxillary incisors.

1. What are the other findings necessary to come to a diagnosis?
   a. Drug history of the mother and the child
   b. Family history of affected siblings / relatives
   c. History of exposure to excess fluoride in drinking water
   d. IOPA and upper standard occlusal radiographs

2. What would be the differential diagnoses?
   a. Dentinogenesis imperfecta
   b. Chronological hypoplasia
   c. Amelogenesis imperfecta
   d. Dental fluorosis
   e. Amelogenesis imperfecta with syndrome

Radiological findings showed, impacted permanent maxillary incisors, taurodontic permanent molars, thin enamel and normal root morphology

3. What would be the definitive diagnosis?
   a. Dentinogenesis imperfecta
   b. Chronological hypoplasia
   c. Amelogenesis imperfecta
   d. Dental fluorosis
   e. Amelogenesis imperfecta with syndrome

4. What is the incorrect statement?
   a. Patient to be advised to avoid harder foods.
   b. Hypolpastic permanent first molar teeth can be treated with stainless crowns.
   c. GIC restorations are used to improve aesthetics of lower anterior teeth
   d. Bond strength would be affected with composite restorations
   e. Impacted maxillary teeth to be treated with exposure and bond ups.
   f. Definitive treatment for above condition is porcelain fused metal crowns

Answers

1. b,c,d
2. c,d,e
3. e
4. d

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Standard journal article

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* *Intake of acidic food and drink*
* *Frequent vomiting*
* *Gum disease*
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* *The use of dental products with abrasive ingredients*

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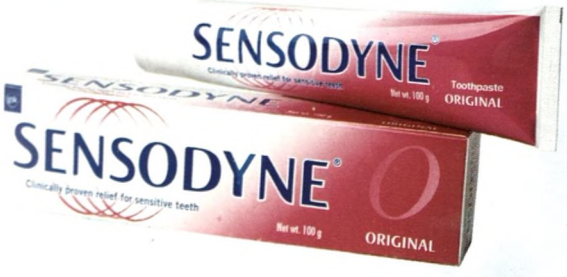

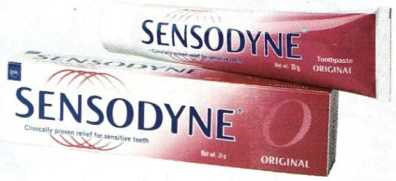

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