Sri Lanka Dental Journal

Volume : 38
Number : 03
December : 2008

SRI LANKA DENTAL ASSOCIATION

The Official Publication of the Sri Lanka Dental Association

ISSN 1391-07280

Sri Lanka Dental Journal

Volume 38 **Number 03** **December 2008**

The **Sri Lanka Dental Journal** is a refereed journal published three times a year by the Sri Lanka Dental Association in collaboration with the College of Dentistry and Stomatology of Sri Lanka, College of General Dental Practitioners of Sri Lanka and the College of Community Dentistry of Sri Lnka.

Editorial Office

Office of the SLDA,
Professional Centre,
275/75, Bauddhaloka Mawatha,
Colombo - 7.

Correspondence regarding editorial matters, articles, reviews and news items should be addressed to the Editor, SLDJ, Dr. Upul B. Dissanayake, Dept. of Oral Pathology , Faculty of Dental Sciences, University of Peradeniya, Sri Lanka.
Tele: 0812 385821/ 0812 387500/ 0812 2392068
0777 393318

Correspondence regarding advertisements and financial matters should be addressed to Dr. Malcolm Stanislaus , 50, Hekitta Cross Road, Hendala, Wattala.
Tele: 011 - 2930368

ISSN 1391 - 07280

Typeset & Printed by:

AK 2 PRO - Ethul Kotte, Kotte.
Tel/Fax : 011 - 2871899

CONTENTS

Sri Lanka Dental Journal

Volume 38 Number 03 December 2008

EDITORIAL
ARE WE ASLEEP

Lack of suitable material has become a major barrier in publishing our professional journal "Sri Lanka Dental Journal". Our members seem to have no interest in sharing their experiences with fellow practitioners by publishing interesting clinical material that they encounter in day to day practice. As professionals, this is one of the serious problems that have to be addressed promptly. What is the reason for this? How do we change this scenario? Has the culture of professional writing not been inculcated in the dental community or is this because of lack of interest? May be that dental surgeons have no time to spend on writing and publishing, because of their heavy workload? However, as members of this noble profession all of us have a moral responsibility of contributing towards improvement and progress in dentistry in Sri Lanka. Distribution of information and sharing knowledge play a very significant role in the development of a profession especially in the context of the modern day world. This is a vital need of our profession. Therefore, a dialogue on this issue among the members of the profession is the need of the hour. Let us all rise up to the occasion.

Upul B Dissanayake
Editor

Leading Article

Sri Lanka Dental Journal 2008; 38(03) 99-101

Competencies for a dentist in Sri Lanka

Nilmini Wanigasooriya

Most of us in the profession of dentistry, have an idea of what the general dental surgeon should be able to do, but there certainly is no universal agreement or consensus about it. A list of abilities on which there is consensus is yet to be formulated. The value of having such a list is well established and many developed countries have put forward such lists. A validated and agreed upon list of competencies for the dental graduate in Sri Lanka would be of immense value in all areas related to the profession of dentistry.

The words 'competence' and competency' are used interchangeably, and may be defined as 'the quality of being legally qualified or adequate'. In the context of professional education and training, the word "Competency" has two meanings. The first derives from the definition that competency 'addresses the ability of an individual to perform effectively in a given job. The second definition of the word is 'what is required of an individual for effective performance'. The second meaning involves definining what is important for effective performance, while the first deals with the degree to which an individual does what is important to a job. These two definitions are closely related, but are separate.

When considering competencies in the context of training or learning, two other terms that come to mind are 'Learning Outcomes' and 'Learning Objectives'. These terms from the vocabulary of educationists, which are used in the context of professional training also refer to qualities or attributes that teaching or training aims at. All three of these terms are defined from the point of view of the learner and are similar in meaning, and refer to qualities in a person. The differences between them are subtle, and due to this, some find themselves confused about their precise meaning and appropriate use. This calls for a clear explanation of the difference between these terms.

Their differences lie mainly in the level at which they are targeted. Learning objectives and learning outcomes may be used interchangeably and define a specific and circumscribed outcome, usually within a given domain of learning of knowledge, skills or attitudes. Competencies on the other hand define broad attributes that are complex and not limited to any one domain of learning. They lie at a higher level than learning objectives. One competency therefore, would invariably combine more than one objective, and encompass more than one educational domain, even all of them.

The following example may be used to clarify this.

Competency: The new dentist must be competent to manage caries in patients of all ages. This competency would consist of several key component competencies.

Dr. Nilmini Wanigasooriya Head, Unit for Development of Dental Education, Faculty of Dental Sciences, University of Peradeniya.

The achievement of this competency or one of the supporting key competencies would involve the achievement of many learning objectives.

Objective 1: The student should be able to identify carious teeth and tooth substance on visual examination, on exploration with a dental probe, and on radiographs.

Objective 2 : The student should be able to remove carious tooth substance, using rotary and hand dental instruments, without causing damage or injury to healthy tooth or other oral tissues.

In this example, two objectives that would be included in one competency is given, but it is obvious that these two are insufficient to cover the competency fully, and would have to be supplemented by several other objectives or learning outcomes of diverse domains and even disciplines in order to be complete.

There are many ways in which a clear statement of desirable competencies would be of use to a profession. Such a list in relation to dentistry would serve many functions. It would provide valuable information for dental educators, prospective and current dental students, dental health service planners and prospective patients among others. It would serve as a basis for planning of all areas in relation to oral health, from education and training to delivery of health care.

Where there is more than one level at which a profession is practiced as in dentistry, clearly defined lists of competencies would help to distinguish the role of one category of worker from others. As such, the role of the Dental Therapist, the Qualifying Graduate, and that of the graduate with Special Training in a specific clinical discipline, be clearly defined. This paves the way to plan Oral Health Care with minimum overlap between the categories, and could help the development of a seamless structure of oral health care delivery. Health care planners would be able to base their decisions on human resource projections on lists of competencies of the different categories workers. This would also help to ease difficulties that arise when considering new categories of possible auxiliary and other personnel. The clear definitions would allow the regulatory authorities to readily identify instances of overstepping of boundaries or working outside the particular worker's mandate.

In the sphere of education planners, this would enable the establishment of principles or key considerations in making decisions regarding training methods and assessment. Students, prospective students and educators would draw many benefits from a competency list. Prospective students would be able to learn the nature of the role of a dentist in the context of Sri Lanka, by competency statements. They would be able to determine their preference, based on much more reliable information than a lay understanding of the role of the professional.

In the learning situation, competencies serve to describe the outcome that is desired in the learner, in the real-life work situation. This helps the learner to target the learning, and assess progress. Where training constitutes several different disciplines, like it does in Dentistry, competencies would give direction and guidance to the educators as to the degree of relevance of the field in the professional role. In a given study area, educators can use competencies to determine the extent, and the depth at which to deal with a topic. They could highlight and emphasize the more relevant aspects, which would make the topic more stimulating to the students. This is particularly useful in relation to disciplines, the relevance of which is not obvious, and would help to retain the interest of the student.

Competencies would help learners to understand the relevance of a given discipline, and put the learning material in context, and perspective. They would be of particular value in complex learning situations, in helping the learner to identify his or her specific role and the learning need.

Competencies should be the guiding principle in assessments carried out for certifying student as professionally qualified. Constant reference to stated competencies when formulating assessment tools as well as during assessment sessions, would improve the validity of the exercise. Those responsible for certification would be able to ensure that the graduate receiving the certification is in fact able to perform in the specified role. This is a high priority in a field like dentistry, where graduating dentists are qualified to register with the Sri Lanka Medical Council, for independent practice, licensing them to attend on patients. A list of competencies is essential before the establishment responsible for this task could consider educational innovations like criterion-referenced methods to student assessment, which is superior to norm-referenced method currently used in relation to professional education.

For students this would act as a clear pointer to what is required of them when they are assessed for certification.

In planning the training of dentists, competencies have begun to precede the specification of learning objectives in many developed countries. Many lists specifying competencies for specific countries are available in the literature. On occasion one finds the need for many different lists being questioned. Indeed on one level one may visualize an "ideal" dentist who possesses all desirable competencies and argue that all dentists should be equally and uniformly competent.

Regardless of the effects of globalization, there still exists wide variation in social, economic and political environments in different parts of the world. These variations influence and determine the kind of work a dentist is called upon to engage in. Another major factor which gives rise to variations in the work is the prevalent disease patterns. A list of competencies that would cover all possible diseases conditions and situations would be very long indeed. Training dentists to such a uniform and 'ideal' list of competencies would require a much longer period of training and will also result in dentists being in the possession of many redundant competencies which they may rarely or never be called upon to exercise. The very existence of many such lists is proof of the need for different lists for different situations. As long as social, economic and political differences and different disease pattern remain in different environments, the need for different and specific competencies in relation to them will also remain.

The position of Sri Lanka in the social, economic, cultural and political context is a unique one. Compiling a list of competencies for the qualifying graduate in Sri Lanka, would be of use in many ways including those discussed here. Particularly in the field of education, if we are to train the kind of dentists that suits our particular situation and if we are to go about their training and assessment in a meaningful and valid way, compiling such a list of competencies, for a dentist working in the Sri Lankan context is an important and urgent responsibility of the profession and of all stake holders.

References

1. Boyd AM, Gerrow JD, Chambers DW *et al.* Competencies for dental licensure in Canada. J Dent. Educ. 1996: 60: 842-846.

2. Plasschaert AJM, Holbrook WP, Delap E *et al.* Profile and competences for the European dentist. Eur J Dent Educ 2005: 9: 98-107.

3. American Association for Dental Schools. Competencies for the new dentist. Proceedings of the 1997 AADS House of Delegates, aooebdux2 J Dent Educ 1997: 71: 556-558.

4. General Dental Council. The first five years. A framework for undergraduate dental education 2nd edition London. GDC, 2002.

Review Article

Sri Lanka Dental Journal 2008; 38(03) 102-114

Drug-associated oral candidiasis

J. A. M. S. Jayatilake, N.S. Soysa, A.N.B. Ellepola

Abstract

Candidiasis is by far the leading oral fungal infection in human due to multiple factors including HIV infection, diabetes mellitus and poor oral/denture hygiene. Oral candidiasis could also manifest as an adverse effect of drugs such as antibiotics, corticosteroids and cytotoxics. Therapeutics including anticholinergic drugs, antacids, and contraceptives have also been found to induce this opportunistic infection in some patients. There is an alarming increase in the use of therapeutics currently, with the increase of the compromised population, change of life patterns and modern treatment procedures such as tissue transplant therapy, cancer therapy and hormonal therapy. Further, drugs are being used indiscriminately in some parts of the world due to self-medication and over the counter availability. Some of these drug treatments may induce oral candidiasis in susceptible individuals. This review focuses on oral candidiasis associated with some frequently used drug treatments with a brief overview of clinical manifestations and mechanisms of pathogenicity. Procedures, which could be used to minimize the onset of such iatrogenic oral candidal infections, are also discussed.

Key words: Oral candidiasis, antibiotics, antacids, corticosteroids, cytotoxics, anticholinergic drugs, contraceptives, therapeutics

Introduction

Oral candidiasis is one of the common infections among medically compromised patients due to the commensal yeast *Candida* species. It appears in different clinical guises namely; pseudomembranous, hyperplastic, erythematous, linear gingival erythema, angular cheilitis and median rhomboid glossitis.[1] Strikingly, the transition of *Candida*- commensalism to parasitism in many instances is associated with the factors other than the pathogen, which is rather unique when compared to many other infectious diseases. Some of the host factors that predispose oral candidiasis include compromised states such as diabetis mellitus, HIV infection, malnutrition and neonatal or elderly age.[2] Furthermore, convalescence after major surgeries and radiotherapy for malignancies are also known to induce oral candidiasis.

Oral candidal infections could also appear as an adverse effect of various drug therapies including antimicrobials, corticosteroids, and cytotoxics.[2]

Dr. J.A.M.S. Jayatilake, (Correspondance) Senior Lecturer, Division of Microbiology, Faculty of Dental Sciences, University of Peradeniya, Peradeniya, Sri Lanka. Tel : 94 8122 397246 Fax : 94 8122 388948 E-mail : sumedhaj@pdn.ac.lk

Dr. N.S. Soysa Senior Lecturer, Division of Pharmacology, Faculty of Dental Sciences, University of Peradeniya, Peradeniya, Sri Lanka. Fax : 94 8122 388948

Dr. A.N.B. Ellepola Department fo Biochemical Sciences, Faculty of Dentistry, University of Kuwait.

Furthermore, it is important to note that the palliative and therapeutic use of drugs have become extremely common with the growth of the elderly population, change of life patterns and modern treatment procedures such as cancer therapy, organ transplantation therapy and hormonal therapy. For example, a survey using 24192 drug histories obtained from 4509 elderly individuals has shown that the average number of drugs used per person has increased by 22% over a decade.[3] Another survey of an elderly community has indicated that the number of medications used by a person increased by 22.5% over an eight year duration.[4] In addition, misuse of drugs in particular, broad-spectrum antibiotics[5,6] is also on the increase especially in the developing world where many drugs are sold over the counter without proper prescription. Self-medication practice among some populations also could lead to unwanted drug use.[7] Such an increase in the usage of pharmaco-therapeutics may be associated with numerous adverse effects including oral candidiasis among susceptible individuals.

Most significantly, it is important to understand that oral candidiasis following various therapeutic drug treatments is induced by that particular drug in association with other host factors such as immune status, age, sex and nutrition etc. Using safer substitutes, antifungals or perhaps discontinuing the therapy may avert this adverse effect. However, there are instances such as cancer therapy organ transplantation therapy and hormone replacement therapy where therapeutics have to be continued despite the adverse effects. In this context, identification, management and the proper monitoring of drug associated oral candidiasis is an important responsibility assigned to the contemporary health care practice. As such, this review discusses how various drug therapies contribute to oral candidiasis, its clinical importance and means of management and prevention of such iatrogenic oral candidal infections.

Different drug groups associated with oral candidiasis

Oral candidiasis is associated with a variety of drug therapies and some of the leading therapeutics are antimicrobials, corticosteroids, and cytotoxics. In addition, use of other medications including anticholinergic drugs, contraceptives, and antacids is reported to induce oral candidiasis in some patients.[8] Latter drugs have been assigned to a miscellaneous group of drugs contributing to oral candidiasis on behalf of descriptive purposes in this review.

Antimicrobials

Antimicrobials constitute one of the important drug groups contributing to iatrogenic oral candidiasis. In particular, broad-spectrum antibiotics are found to increase the occurrence of oral candidal infections by disrupting the balance within the normal flora.[8] In early reports, oral candidiasis following antibiotics has been called antibiotic sensitive tongue or antibiotic sore tongue.[9] Later, studies by Lehner and Ward (1970) have demonstrated that patients having topical application of corticosteroids and tetracycline can develop erythematous or pseudomembranous candidiasis of the oral mucosa.[10] Currently, modern antimicrobial agents with improved broad-spectrum activity remain one of the important causes for the iatrogenic oral candidiasis.[11,12] Clinically, antibiotic associated oral candidiasis is characterized by a raw erythematous appearance over the dorsum of the tongue with or without pain and usually shows an acute onset. This variant of oral candidiasis has now been classified as erythematous candidiasis.[13]

Many antimicrobials including tetracycline, metronidazole, ampicillin and aminoglycosides are found to induce oral candidiasis in different patients.[11] However, the commonest antimicrobial agent associated with oral candididasis is tetracycline and particularly minocyclin and derivatives of former have led to an increase in the incidence of oral candidiasis.[14] Occasionally,

metronidazole used in the treatment of anaerobic and parasitic infections has also induced oral candidiasis in some patients.[15] Studies on hospitalized patients undergoing antibiotic therapy have revealed that the effect of aminoglycosides was higher than that of penicillin and tetracycline in causing oral candidiasis.[16,17] Minocycline, a long active derivative of tetracycline used by the patients having acne and immunobullous disorders has increased those patients' likelihood of getting oral candidiasis significantly.[18,14] Trimethoprim and sulphamethoxazole (cotrimoxazole) is a widely used combination which has been reported to induce oral candidiasis even after short duration of therapy.[19] In addition, various animal studies have contributed substantially to investigate the oral candidiasis following numerous antimicrobial agents.[20,21] Many of these studies have identified that oral candidiasis is a common adverse effect consequential to many antibiotics particularly with broad-spectrum activity.

Though the cause and effect relationship between antimicrobials and the oral candidiasis is yet to be elucidated, it is evident that suppression of normal microbial flora by the antimicrobial therapy leads to overgrowth of the commensal *Candida* in the oral cavity. Normal flora of the oral cavity exerts control over the yeast adhesion and therefore colonization is controlled.[2] At the same time, there is competition for nutrition among the oral microbes including *Candida*. This phenomenon is supported by the findings of Wu and Samaranayake (1990) who have demonstrated that bacteria present in the mixed saliva successfully compete for nutrients controlling the candidal growth and the adhesion.[22] Interestingly, it has been reported that some oral bacteria such as *Streptococcus mutans, Streptococcus salivarious* and *Lactobacillus casei* can inhibit the growth of *Candida* in vitro.[23] Antimicrobials are molecules that interfere with the microbial metabolism by selective disruption of the cell wall or the cell membrane, blocking the synthesis of protein or nucleic acids etc. of the microorganisms. These effects on the homeostasis of the useful oral microbial flora may interrupt the balance within the microbial community and that may lead to opportunistic candidal infections. Moreover, certain antibiotics such as erythromycin, co-trimoxazole and aminoglycosides can alter the *Candida*-cidal activity of neutrophils by which oral colonization of yeast is promoted.[24,25] As such, it is conceivable that the effects of the antimicrobials on the oral microbial community including *Candida* and the host immunity are complex.

However, the most likely mechanism is that the inhibition of normal flora by the action of antimicrobial drugs brings about candidal overgrowth and acute oral candidiasis.[11] In addition, the effects of antimicrobial drugs on the systemic immunity also may affect the candidal super-infection of the oral cavity. All together, these observations suggest that antimicrobial drug therapy that results in the breach of the fine balance within the oral microbial community is a key factor that increase candidal adhesion and colonization leading to super-infection.

Corticosteroids

Adrenal cortex produces corticosteroids naturally. These hormones are important in the regulation of the inflammatory and immune response due to their potent anti-inflammatory and immunosuppressive effects. Thus, synthetic steroids are therapeutically used for their activity in suppressing the inflammation and immunity in clinical conditions such as asthma, rheumatoid arthritis, organ transplantation or autoimmune disorders. Corticosteroid preparations appear to lower host resistance to *Candida* by suppressing immunity, both the non-specific inflammatory response and specific cellular and humoral immunity.

As early as 1964, it has been documented that steroid users in the form of inhalers were susceptible for oral candidiasis.[26] Since then, many studies using patients who were on steroids inhalation have suggested that the steroid

inhalation promotes the oral carriage of *Candida* or candidal infection that appear as erythematous or psudomembranous variants.[27] For instance, steroid inhalers are accountable for about 10-30% of oral candidiasis.[28] In addition, Ellepola and Samaranayake (2001) based on a number of clinical studies suggested that patients on continued steroid inhalation for six months are at a risk of approximately 4-16% comparable to healthy population.[29] Further, due to the anti-inflammatory effect steroids are used as topical applications in the oral cavity in conditions such as lichen planus, pemphigoid, and erythema multiforme and such situations too can incite clinical oral candidiasis.[30]

In an early experiment using Macaque monkeys, Budtz-Jorgensen (1975) has shown that the mechanism of onset of oral candidiasis after steroid therapy entails both the suppression of nonspecific local immunity and the cellular immunity.[31] A recent study by Hu *et al.* (2007) using beclomethasone diproprionate inhalation showed that mice were susceptible to candidal infection in a dose dependent manner; higher the steroid dose greater the risk of getting oral candidiasis.[32] Thus, animal experiments and human clinical studies collectively support the fact that the suppression of the mucosal immunity facilitates *Candida* to become an opportunistic pathogen in the mouth.

Furthermore, it has been identified that certain growth characteristics and virulence features of *Candida* could be modified by the steroids leading to opportunism.[33,34] Apart from the above, steroid therapy may increase the salivary glucose levels that facilitate candidal virulence including adherence and protease production which promote colonization and invasion.[35,36] On the other hand, systemically used steroids in patients having organ transplants and rheumatoid arthritis etc., also suppress the general immune and inflammatory responses increasing the risk of systemic candidiasis and oral infections. Supporting these observations, a study carried out in Mexico has shown that 18.7% patients who were on multi-drug regimen of cyclosporine A, azathioprine and predinisolone were having oral candidais. In those patients, erythematous candidiasis was the predominant (13.3%) clinical diagnosis followed by psudomembraneous (4.4%) and hyperplastic (1.1%) variants.[37] Furthermore, this study indicates that multiple drug usage facilitates parasitic transition of *Candida* in the oral cavity.

Although there is ample evidence clinically and experimentally to show that oral candidiasis is associated with steroid therapy, the exact relationship between the cause and effect is not clearly defined so far. Yet, many investigators have suggested that the overall immune and inflammatory suppression lead to candidal infections.[30,38] Most significant features of steroid associated oral candidiasis are that the clinical manifestations were related to the site into which the drug is directly applied and the dosage regimen.[39,40] Intriguingly, the likelihood of having oral candidiasis is higher in topically applied steroids than the inhaled steroids since the former leads to prolonged effect of the drug at the site of the application.[41] On the other hand, simultaneous usage of antimicrobials and steroids also may predispose oral candidiasis due to candidal super-infection facilitated by the suppression of normal flora and the local immune mechanisms.

At the same time, Enwonvu *et al.* (1996) have postulated that the presence of steroid hormones in the oral environment stimulates the growth of *Candida* leading to infections.[42] It has also been reported that steroidal drugs such as triamcenolone and dexamethasone can alter the cellular metabolism of yeast and their susceptibility to antifungals.[43,44] One of the important side effects of steroids inhalation is that it causes local suppression of the immunity. For instance, Fukushima *et al.* (2005) have found that total salivary IgA levels in patients are significantly reduced by inhaled steroids and it has been hypothesized that the local immune suppression

is one of the factors that increase the vulnerability of steroid inhaling patients for oral candidiasis.[45] Ultimately, the suppression of the immunoinflammatory response of the host together with the altered virulence of the yeast is likely to play a combined role in the pathogenesis of oral candidiasis following corticosteroid therapy.

Cytotoxic drugs

In the management of malignancies, cytotoxic drugs play a major role. Suppression of the immune system both locally and systemically in particular, the cell mediated defenses is one of the important adverse effects of cytotoxic drug therapy.[2] As a result, oral candidiasis is frequently observed in patients on anticancer chemotherapy with cytotoxic drugs.[46] Strikingly, one major factor for the morbidity among cancer patients is oral candidiasis. For example, McCarthy and Skillings (1992) showed that 12% of women on anticancer drug therapy for the breast cancer suffer from oral candidiasis.[47]

Radiotherapy often given to cancer patients also partly contributes to oral candidiasis. On the other hand, patients having malignancies carry more yeast in their oral environment when compared with the healthy controls.[48] Interestingly, oral yeast carriage rates have inclined during the process of cancer chemotherapy or the radiotherapy. In addition, the concurrent use of broad-spectrum antibiotics on these patients further can worsen the opportunistic fungal attacks.[49] The problem tends to be more complex as muti-species colonization of *Candida* is already reported in cancer patients.[50] A study done using renal transplant recipients on cyclosporine which is a cytotoxic immunosuppressant showed almost four fold higher candidal carriage and oral candidiasis compared to control subjects.[51] Animal studies using cytotoxic drugs such as cyclophosphamides have revealed that the cancer therapy leads to oral candidiasis due to the suppression of the systemic immune system.[52] Several investigators have demonstrated that immunosuppression produced in animals by cytotoxic drugs gives rise to oral candidal infections.[53] Taken together, both human and animal studies suggest that the cancer therapy that carries a greater adverse effect on immune system brings about increased candidal carriage, colonization and infection in the oral cavity.

Oral candidiasis in patients who are having oral or other malignant conditions seem to have complex etiology assisted by the combined effect of host factors related to the morbidity resulted by the malignancy itself and the immunosuppression caused by the cancer therapy. Cancer therapy suppresses the neutrophils, lymphocyte monocyte axis which is important in preventing candidal opportunism.[54] Thus, normal function of the lymphopoietic system is likely an important factor in prevention of oral candidiasis as many of the cancer patients who are having granulocytopaenia are victims of this mycosis. Reduction of salivary gland function is also a major adverse effect of cancer therapy. This condition may be severe with about 64% reduction of salivary flow rate that brings about the oral microbial population into two fold level.[49] Furthermore, Ueta *et al.* (1994) have reported that the activity of salivary polymorphoneuclear leukocytes is reduced consequential to the cancer chemotherapy.[55] This may even facilitate the onset of oral infections most commonly the oral candidiasis. Several other antimicrobials used for the control of infections in cancer patients too can lead to over colonization of yeast in the oral cavity leading to super-infections. Radiotherapy in particular given to the orofacial neoplasia reduces salivary gland function and induces mucositis allowing candidal infestation.[48] Furthermore, the antineoplastic drugs can affect the proliferation of epithelial cells due to their effect on highly dividing basal cells. This effect may facilitate candidal infections in the atrophic oral epithelium.

Other miscellaneous drugs

Some other drugs have also shown to predispose oral candidiasis though any cause and effect has

not been clearly elucidated. It has been reported that certain psychotropic drugs including chlorpromazine, benztropine, lithium, risperidone having anticholinergic effects can induce oral candidiasis in patients through inducing dry mouth.[56] Tricyclic antidepressants commonly used in the management of psychiatric patients are associated with reduced salivary flow, which can lead to oral candidiasis.[8] Mostly oral anticholinergic drugs would be associated with erythematous candidiasis because xerostomia induced oral candidiasis often manifests as erythematous mucosa with burning sensation of the mouth, or white lesions.[57] Saliva plays an important role in the maintenance of healthy oral milieu. It contains number of immune mechanisms including secretory antibodies, lysozymes, lactoferrin and mucus secretions that inhibit adhesion, oral colonization and proliferation of different microbes such as *Candida* species. In addition saliva helps in the cleansing of the oral tissues by removing the developing biofilms. In other pathological conditions where there is reduction of salivary flow such as diabetes mellitus, radiation to salivary glands in head and neck cancer radiotherapy and sjorgren's disease, oral candidiasis is a common problem.

Another important group of drugs that often has been associated with oropharyngeal candidiasis is antacids. One of the common antacids related to oropharyngeal candidiasis is omeprazole. Esophageal candidiasis has been detected as a common problem in patients using omeprazole for long durations.[58,59] For instance, Sood *et al.* (1995) have produced two case reports on patients having omeprazole developed esophageal candidiasis and it was postulated that the suppression of the acidity by the drug facilitate overgrowth of *Candida* in the oropharynx.[58] In another report, Larner and Lendrum (1992) discussed two cases of oral candidiasis following omeprazole therapy and it was also speculated that the reduction of gastric acidity below the optimum levels, that control yeast growth lead to super-infections in the oropharynx.[59] However, oral candidiasis following antacids such as omeprazole should be further investigated to unravel exact pathological mechanisms.

Contraceptives (birth control pills) mostly containing estrogen or progesterone derivatives have hormonal effects that can lower a woman's immune defenses to overgrowth of *Candida*. Supplemental estrogen in the form of oral contraceptives commonly produce vulvovaginal candidiasis and has been found to promote the growth of *Candida* in the oral cavity as well leading to oral candidal infections among females having contraceptive pills.[60] In an experiment to assess the effect of estrogen on the candidal morphogenesis, White and Larsen (1997) found that beta estradiol strongly induces the filamentation of *C. albicans*.[61] These findings suggest that estrogen therapy may increase the virulence of *Candida* by inducing filamentation that facilitates tissue invasion. Mice treated with estrogen have shown more susceptibility to *C. albicans* vaginitis suggesting that estrogen is an important factor in hormone-associated candidal infections.[62] Yet, these investigators pointed out that progesterone had no effect on the induction and persistence of vaginitis in mice. Finally, oral contraceptives may act as another group of drugs associated with oral candidiasis brought about by host immune suppression and the alteration of fungal growth characteristics. However, further investigations are necessary to confirm these phenomena.

General mechanisms of pathogenesis of drug associated oral candidiasis

Therapeutics such as corticosteroids suppress the host immunity both locally as well as systemically. Some other therapeutics such as antimicrobials, cytotoxics and antacids etc. may affect the immunity in addition to their expected therapeutic effects. On the other hand, antimicrobials facilitate *Candida* colonization by suppressing the normal flora. Certain therapeutics such as antipsycotics, corticosteroids and antacids change the chemical and physical aspects of the oral

milieu to facilitate candidal colonization. At the same time, direct and indirect effects of therapeutics may affect the virulence factors including adhesion to epithelial cells and extracellular enzyme production of *Candida* cells. Oral candidiasis is one of the adverse effects of the above one or more actions of the therapeutics. Moreover, simultaneous use of drugs such as corticosteroids and antibiotics can induce *Candida* opportunism in the oral cavity. For instance, Burton *et al.* (1992) have reported that infants treated for laryngotracheobronchitis with steroid inhalation and antibiotics are more prone to *Candida*-laryngotracheitis.[63] Similarly, cytotoxic or radiotherapy used with antibiotics also may bring about similar effects. If one or more therapeutics breach the host defenses, antimicrobials will potentiate the overgrowth of oral yeasts by suppressing the commensal flora. This emphasizes the necessity of close monitoring of individuals who are having multiple drug treatment in order to avoid opportunistic candidal infections.

Prevention of drug associated oral candidiasis

In the management of drug associated oral candidiasis, patient management protocols such as alternative medication, dose reduction or drug withdrawal, correction of the local effects such as hyposalivation by giving saliva substitutes should be considered. Misuse of antimicrobial drugs may bring about both antibiotic resistance formation and opportunistic candidal infection in the oral cavity. Therefore, improving patient awareness on rational usage of drugs is important in preventing antimicrobial induced oral candidiasis. Incorporation of antifungals into the antibiotic treatment of susceptible patients was reviewed by Soysa *et al.* (2008) and it has been concluded that management of oral candidiasis is highly effective with antifungals alone provided with the control of the underlying contributory agents.[11]

In corticosteroid inhalation, control of the drug deposition and the contact with the oral mucosa could be controlled by using different types of apparatus such as inhalers known as turbuhaler.[64] The latter apparatus will help the patient to control and measure the required dose of the drug needed and therefore it is effective in preventing over dosage also. Using a spacer device attached to the inhalers is known to reduce the drug contact area of the oral epithelium in order to minimize the after effects.[40] Thompson *et al.* (1986) have demonstrated that nystatin a popular antifungal in the form of pastilles and suspensions are equally successful in the management of steroid induced oropharyngeal candidiasis.[65] Washing out of the mucosa with water or mouth wash is also a simple method to minimize the local and systemic adverse effects of steroid inhalation.[38] Finally, the use of prophylactic antifungals during the steroids therapy may control the candidal super-infection and it is appropriate for patients whose immunity is already compromised.[41, 66]

Many studies have shown that cancer patients who are on antineoplastic chemotherapy or radiotherapy are extremely susceptible for oral or even systemic candidiasis. This has led to a high rate of morbidity and mortality among those patients. Therefore, eradication of *Candida* in the oral cavity using antiseptic mouthwashes has been recommended. On the other hand, prophylactic use of antifungal drugs also a popular method to control oral candidal infections in this group of patients. Various antiseptic mouthwashes including chlorhexidine and povidone iodine are proven to reduce the yeast load of the oral cavity and may be helpful in prevention of opportunistic infections. For instance, use of chlorhexidine as an adjunct with the antifungals in the management of oral candidiasis has also been suggested previously.[67] Moreover, provision of artificial saliva may help cleansing of their oral structures preventing colonization of *Candida.* Prevention of smoking and betel chewing is also important in reducing the candidal carriage and infection in the oral cavity.[68]

The prime tool in the management of oral candidiasis is at present is the antifungal drugs and it is important to consider the underlying pathology of the patient. Proper selection and the appropriate dosage regimen will help to control the onset of the disease and adjunctive measures such as maintenance of good oral hygiene and chlorhexidine mouthwash also are beneficial for susceptible individuals. Continuous monitoring of the fungal load in the susceptible patients such as who are on steroid therapy by oropharyngeal swabs may be helpful to predict the risk of developing oral candidiasis. Also, gargle of 1:50 amphotericin B solution is a prophylactic for the prevention of oral candidiasis in steroid inhaling asthmatics.[69] One of the primary approaches in controlling drug associated oral candidiasis is to maintain a good oral hygiene in those patients having different drug therapies. It is advisable to have these patients check their oral health frequently and be vigilant on any adverse effect while they are on treatment as well as afterwards. With reference to xerostomia-induced candidiasis due to drug treatment, it is important to induce salivation or produce saliva substitutes. Prophylaxis antifungals also play a role in such patients.[57] Again, the proper oral hygiene plays a significant role in the management and prevention of oral candidiasis in those susceptible cases.

Conclusion

Exact mechanisms by which commensal yeast *Candida* is converted to opportunistic pathogen due to the usage of drugs is yet to be uncovered. However, prime mechanisms that involve oral candidiasis following drug therapy definitely involve the changes in the host local and systemic immunity, interruption to the commensal flora of the oral cavity, together with alteration of candidal growth and the virulence mechanisms. Prudent use of any kind of drug, which can compromise the local or systemic immune mechanisms, is of utmost importance to control this unwanted effect of drug administration and usage. Some of the drug effects such as salivary gland suppression that can pave way for opportunistic infections should be managed using appropriate precautions and proper monitoring of the salivary functions. Eventually, it is of utmost importance to maintain and monitor the oral hygiene of patients on numerous drug treatments to avoid oral candidiasis, one common factor of morbidity among the debilitated community.

Table 1. Drugs that have reported to induce oral candidiasis.

Group of Drugs	Examples	Possible mechanism for inducing oral candidiasis	References
Antimicrobials	tetracycline minocyclin penicillin ampicillin metronidazole aminoglycosides trimethoprim and sulphamethoxazole	Disruption of the balance within the normal microbial flora. Alteration of *Candida*-cidal activity of neutrophils	2, 8, 22-25
Corticosteroids	beclomethasone predinisolone triamcenolone dexamethasone	Suppression of the general immune and inflammatory responses Suppression of the local immunity at the mucosal level. Modification of growth characteristics and virulence features of *Candida.* Increase of salivary glucose levels that facilitate candidal adherence and protease production.	27, 28, 30-36, 38, 42-45
Cytotoxics	cyclosporine cyclophosphamide	Suppression of the immune system both locally and systemically. Reduction of salivary gland function (xerostomia) and polymorphoneuclear leukocytes activity	2, 46-55
Anticholinergics	chlorpromazine, benztropine, lithium, risperidone	Reduction of salivary gland function - xerostomia	8, 56, 57
Antacids	Omeprazole	Suppression of the acidity in the oropharynx	58, 59
Contraceptives	estrogen progesterone	Suppression of the immune system Growth facilitation of *Candida* in the oral cavity	60-62

References

1. Ellepola AN, Samaranayake LP. Antimycotic agents in oral candidosis: an overview: 1. Clinical variants. Dent Update 2000; 27:111-112, 114-116.

2. Samaranayake L. Oral candidosis. Samaranayake LP and MacFarlane TW. 1990. London, Wright.

3. Stewart RB, Moore MT, May FE, *et al.* Changing patterns of therapeutic agents in the elderly: a ten-year overview. Age Ageing 1991; 20:182-188.

4. Linjakumpu T, Hartikainen S, Klaukka T, *et al.* Use of medications and polypharmacy are increasing among the elderly. J Clin Epidemiol 2002; 55:809-817.

5. Al-Azzam SI, Al-Husein BA, Alzoubi F, *et al.* Self-medication with antibiotics in Jordanian population. Int J Occup Med Environ Health 2007; 20:373-380.

6. Okumura J, Wakai S, Umenai T. Drug utilisation and self-medication in rural communities in Vietnam. Soc Sci Med 2002; 54:1875-1886.

7. Greenhalgh T. Drug prescription and self-medication in India: an exploratory survey. Soc Sci Med. 1987; 25: 307-18

8. Gottlieb SL. Oral yeast infections. NOHA NEWS. 1995; 20: 4-5.

9. Lehner T. Classification and clinico-pathological features of Candida infections in the mouth. In: Winner H, Hurley R, eds. Symposium on Candida infections. Livingstone: Edinburgh, pp. 119–136. 1966.

10. Lehner T, Ward RG. Iatrogenic oral candidosis. Br J Dermatol 1970; 83:161-166.

11. Soysa NS, Samaranayake LP, Ellepola AN. Antimicrobials as a contributory factor in oral candidosis—a brief overview. Oral Dis 2008; 14:138-143.

12. Epstein JB, Polsky B. Oropharyngeal candidiasis: a review of its clinical spectrum and current therapies. Clin Ther 1998; 20: 40-57.

13. Samaranayake L. Essential microbiology for dentistry. Churchill Livingstone London. 2007.

14. Ozog DM, Gogstetter DS, Scott G, et al. Minocycline-induced hyperpigmentation in patients with pemphigus and pemphigoid. Arch Dermatol 2000; 136:1133-1138.

15. Simjee AE, Gathiram V, Jackson TF, et al. A comparative trial of metronidazole vs. tinidazole in the treatment of amoebic liver abscess. S Afr Med J 1985; 68:923-924.

16. Walker AM, Jick H, Porter J. Drug-related superinfection in hospitalized patients. JAMA 1979; 242:1273-1275.

17. Caldwell JR, Cluff LE. Adverse reactions to antimicrobial agents. JAMA 1974; 230:77-80.

18. Goulden V, Glass D, Cunliffe WJ. Safety of long-term high-dose minocycline in the treatment of acne. Br J Dermatol 1996;134:693-695.

19. Kocak Z, Hatipoglu CA, Ertem G, et al. Trimethoprim-sulfamethoxazole induced rash and fatal hematologic disorders. J Infect 2006; 52:49-52.

20. Jones JH, Russell C, Young C, et al. Tetracycline and the colonization and infection of the mouths of germ-free and conventionalized rats with Candida albicans. J Antimicrob Chemother 1976;2:247-253.

21. Hassan OE, Jones JH, Russell C. Experimental oral candidal infection and carriage of oral bacteria in rats subjected to a carbohydrate-rich diet and tetracycline treatment. J Med Microbiol 1985; 20:291-298.

22. Wu T, Samaranayake LP. The expression of secreted aspartyl proteinases of Candida species in human whole saliva. J Med Microbiol 1999; 48: 711-720.

23. Krasner RI, Young G, Yudkofsky PL Interactions of oral strains of Candida albicans and lactobacilli. J Bacteriol. 1956; 72:525-9.

24. Bridges CG, Dasilva GL, Yamamura M, *et al.* A radiometric assay for the combined measurement of phagocytosis and intracellular killing of Candida albicans. Clin Exp Immunol 1980;42:226-233.

25. Ferrari FA, Pagani A, Marconi M, *et al.* Inhibition of candidacidal activity of human neutrophil leukocytes by aminoglycoside antibiotics. Antimicrob Agents Chemother 1980; 17:87-88.

26. Dennis M, Itkin IH. Effectiveness and complications of aerosol dexamethasone phosphate in severe asthma. J Allergy Clin Immunol 1964; 35:70-76.

27. Ellepola AN, Samaranayake LP. Oral candidal infections and antimycotics. Crit Rev Oral Biol Med 2000; 11:172-198.

28. Kuna P. Longterm effects of steroid therapy. Wiad Lek 1998; 51:12-18.

29. Ellepola AN, Samaranayake LP. Inhalational and topical steroids and oral candidosis: a mini review. Oral Dis 2001; 7: 211-216.

30. Seymour HR, Meechan JG, Yates MS. Pharmacology and dental therapeutics Oxford University Press, Oxford. 1999.

31. Budtz-Jorgensen E. Effects of triamcinolone acetonide on experimental oral candidiasis in monkeys. Scand J Dent Res 1975; 83:171-178.

32. Hu W, Ninomiya K, Ishibashi H, et al. A novel murine model of pharyngeal candidiasis with local symptoms characteristic of pharyngeal thrush produced by using an inhaled corticosteroid. Med Mycol 2007;45:143-148.

33. Braun PC. Surface hydrophobicity enhances corticosterone incorporation in Candida albicans. Infect Immun 1994; 62:4087-4090.

34. O'Day DM, Ray WA, Head WS, et al. Influence of corticosteroid on experimentally induced keratomycosis. Arch Ophthalmol 1991; 109:1601-1604.

35. Pizzo G, Giuliana G, Milici ME, et al. Effect of dietary carbohydrates on the in vitro epithelial adhesion of Candida albicans, Candida tropicalis, and Candida krusei. New Microbiol 2000; 23:63-71.

36. Samaranayake LP, Hughes A, MacFarlane TW. The proteolytic potential of Candida albicans in human saliva supplemented with glucose. J Med Microbiol 1984; 17:13-22.

37. de la Rosa-Garcia E, Mondragon-Padilla A, Irigoyen-Camacho ME, et al. Oral lesions in a group of kidney transplant patients. Med Oral Patol Oral Cir Bucal 2005;10:196-204.

38. Lipworth BJ. Clinical pharmacology of corticosteroids in bronchial asthma. Pharmacol Ther 1993;58:173-209.

39. Sahay JN, Chatterjee SS, Stanbridge TN. Inhaled corticosteroid aerosols and candidiasis. Br J Dis Chest 1979;73:164-168.

40. Toogood JH, Khan RH, Baskerville JC, *et al.* Effect of corticosteroid on lymphocyte responsiveness in pigeon breeder's lung. Clin Allergy 1980; 10: 547-553.

41. Epstein JB, Komiyama K, Duncan D. Oral topical steroids and secondary oral candidiasis. J Oral Med 1986; 41:223-227, 273.

42. Enwonwu CO, Meeks VI, Sawiris PG. Elevated cortisol levels in whole saliva in HIV infected individuals. Eur J Oral Sci 1996; 104:322-324.

43. Berdicevsky I, Silbermann M. Effect of glucocorticoid hormones on calcium uptake and the morphology of Candida albicans. Cell Biol Int Rep 1982;6:783-790.

44. Khosla PK, Chawla KS, Prakash P, *et al.* The effect of dexamethason and oxytetracyclin on the growth of Candida albicans. Mykosen 1978; 21:342-348.

45. Fukushima C, Matsuse H, Saeki S, *et al.* Salivary IgA and oral candidiasis in asthmatic patients treated with inhaled corticosteroid. J Asthma 2005; 42:601-604.

46. Peterson D, Sonis TS. Oral complications of cancer chemotherapy The Hague: Martinus Nijhoff Publishers. 1983.

47. McCarthy GM, Skillings JR. Orofacial complications of chemotherapy for breast cancer. Oral Surg Oral Med Oral Pathol 1992;74:172-178.

48. Soysa NS, Samaranayake LP, Ellepola AN. Cytotoxic drugs, radiotherapy and oral candidiasis. Oral Oncol 2004;40:971-978.

49. Bergmann OJ. Alterations in oral microflora and pathogenesis of acute oral infections during remission-induction therapy in patients with acute myeloid leukaemia. Scand J Infect Dis 1991;23:355-366.

50. Davies AN, Brailsford S, Broadley K, *et al.* Oral yeast carriage in patients with advanced cancer. Oral Microbiol Immunol 2002; 17:79-84.

51. King GN, Healy CM, Glover MT, *et al.* Prevalence and risk factors associated with leukoplakia, hairy leukoplakia, erythematous candidiasis, and gingival hyperplasia in renal transplant recipients. Oral Surg Oral Med Oral Pathol 1994;78:718-726.

52. Samaranayake YH, Samaranayake LP. Experimental oral candidiasis in animal models. Clin Microbiol Rev 2001;14:398-429.

53. Samaranayake YH, Wu PC, Samaranayake LP, *et al.* The relative pathogenicity of Candida krusei and C. albicans in the rat oral mucosa. J Med Microbiol 1998; 47:1047-1057.

54. Bergmann OJ, Andersen PL. Acute oral candidiasis during febrile episodes in immunocompromised patients with haematologic malignancies. Scand J Infect Dis 1990;22:353-358.

55. Ueta E, Osaki T, Yoneda K, *et al.* Influence of inductive chemoradiotherapy on salivary polymorphonuclear leukocyte (SPMN) functions in oral cancer. J Oral Pathol Med 1994;23:418-422.

56. Stevens HE. Oral candidiasis secondary to adverse anticholinergic effects of psychotropic medications. J Child Adolesc Psychopharmacol 2007;17:145-146.

57. Jensen JL, Barkvoll P. Clinical implications of the dry mouth. Oral mucosal diseases. Ann N Y Acad Sci 1998;842:156-162.

58. Sood A, Sharma M, Jain NP, *et al.* Esophageal candidiasis following omeprazole therapy: a report of two cases. Indian J Gastroenterol 1995;14:71-72.

59. Larner AJ, Lendrum R. Oesophageal candidiasis after omeprazole therapy. Gut 1992; 33:860-861.

60. Krekeler G, Ochs G, Haderthauer W. Oral contraceptives and oral candidiasis. Fortschritte der Medizin, 1981; 99: 230-2.

61. White S, Larsen B. Candida albicans morphogenesis is influenced by estrogen. CMSL, Cell mol life sci. 1997; 53: 744-749.

62. Fidel Jr. PL, Cutright J, Steele C. Effects of reproductive hormones on experimental vaginal candidiasis. Infect Immun 2000; 68: 651-657.

63. Burton DM, Seid AB, Kearns DB, et al. Candida laryngotracheitis: a complication of combined steroid and antibiotic usage in croup. Int J Pediatr Otorhinolaryngol 1992;23 :171-175.

64. Jackson LD, Polygenis D, McIvor RA, *et al.* Comparative efficacy and safety of inhaled corticosteroids in asthma. Can J Clin Pharmacol 1999; 6:26-37.

65. Thompson PJ, Wingfield HJ, Cosgrove RF, *et al.* Assessment of oral candidiasis in patients with respiratory disease and efficacy of a new nystatin formulation. Br Med J (Clin Res Ed) 1986; 292: 1699-1700.

66. Prentice AG, Bradford GR. Prophylaxis of fungal infections with itraconazole during remission-induction therapy. Mycoses 1989;32 Suppl 1:96-102.

67. Ellepola AN, Samaranayake LP. Adjunctive use of chlorhexidine in oral candidoses: a review. Oral Dis 2001; 7:11-17.

68. Soysa NS, Ellepola AN. The impact of cigarette/tobacco smoking on oral candidosis: an overview. Oral Dis 2005; 11:268-273.

69. Fukushima C, Matsuse H, Tomari S, *et al.* Oral candidiasis associated with inhaled corticosteroid use: comparison of fluticasone and beclomethasone. Ann Allergy Asthma Immunol 2003; 90:646-651.

Research Article I

Sri Lanka Dental Journal 2008; 38(03) 115-123

Oral health status and treatment needs of pregnant women in the Divulapitiya Medical Officer of Health area

Nisansala Karunachandra and Lilani Ekanayake

Abstract

Objective: To assess the oral health status and treatment needs in pregnant women from the Divulapitiya Medical Officer of Health area.

Material and methods: The sample consisted of 459 pregnant women in the second trimester attending antenatal clinics of the area. The data were collected by means of an interviewer administered questionnaire and an oral examination.

Results: The mean DMFT of the sample was 5.4 ± 3.0. Caries experience as well as decayed, missing and filled teeth differed significantly according to levels of education and family income. Nearly 59% had bleeding gums. According to the normatively assessed needs, 53% needed restorations while 59% required only oral hygiene instructions. Of the sample, only 16% considered their oral health status to be very good/ good while 63% thought that they had an oral health problem. The most common perceived oral health problem was decayed teeth (43%). Of those who perceived that they had an oral health problem, 50% felt that they do not need care for that problem. The main reason for this being that they felt dental treatment during pregnancy could harm the foetus.

Conclusion: The present study showed that the burden of caries and periodontal diseases as well as both normatively assessed and perceived treatment needs were high in this group of pregnant women. Moreover, the women had certain misconceptions about obtaining dental care during pregnancy.

Introduction

It is well known that various physiological, hormonal and anatomical changes that occur during pregnancy could have an impact on oral health and that the most significant effect of pregnancy is observed in relation to the gingival tissues. Studies have shown that the severity of gingival inflammation increases during pregnancy and decrease post-partum.[1] This is attributed to the elevated levels of sex hormones during pregnancy which could accentuate clinical changes associated with gingivitis as a result of mechanisms such as partial immune suppression, increased fluid exudation and stimulation of fibroblast synthesis.[2] Moreover current research indicates that maternal periodontal disease is associated with adverse pregnancy outcomes such as preterm birth and low birth weight.[3,4] Also oral health related quality of life has shown to be poorer in pregnant women than in non-pregnant women.[5] In addition, maternal oral health can have a significant effect on the infant's oral health. Infants acquire cariogenic bacteria from their mothers.[6] Hence high titres of cariogenic flora in the mother could increase the risk of the infant developing early childhood caries.

Dr. Nisansala Karunachandra Dental Surgeon, Teaching Hospital, Anuradhpura.

Prof. Lilani Ekanayake (Correspondence) Professor in Community Dentistry, Department of Community Dental Health, Faculty of Dental Sciences, University of Peradeniya, Peradeniya. Tel: 081-2397321 Fax: 2388948 E-mail: lilanie@pdn.ac.lk

Oral health in pregnancy has been a topic of interest among researchers from all over the world with the recent focus being on the effect of periodontal diseases in pregnancy on birth outcomes. However, there are only a few published studies on oral health in pregnancy among Sri Lankan women and they have been confined to assessing periodontal disease in pregnancy.[1] Moreover, the Ministry of Health is currently in the process of formulating guidelines for provision of oral health care to pregnant women. Hence information on oral health status and oral health care needs of this group of women will be useful for this purpose. Considering these factors the present study aimed at assessing the oral health status and treatment needs of pregnant women.

Material and methods

This descriptive study was conducted in the Divulapitiya Medical Officer of Health (MOH) area between August and October 2008. The target group consisted of pregnant women in their second trimester. Those pregnant women who had completed the 12th week of period of amenorrhoea (POA) but not entered the 28th week of POA were considered to be within the second trimester. As it has been stated that dental treatment is best performed during the second trimester, it was decided to include only those women in the second trimester in the study.[7]

The sample size was calculated using the formula for estimating a population proportion with specified absolute precision. As data on prevalence of dental caries are not available for pregnant women in Sri Lanka, to calculate the sample size, the prevalence was considered as 50%. When the expected population proportion is unknown, the safest choice for population proportion is considered as 50%.[8] Accordingly the minimum sample size required for the present study using a prevalence estimate of 50% at the 95% confidence level and accepting a sampling error of 5% was 384. This was increased by 10% to compensate for non responses and thus the minimum sample required was 422.

According to the data available at the MOH office, around 3000 women become pregnant per year in the Divulapitiya area. The total number of pregnant at any given point of time is usually assumed to be 50% of this number which is 1500. Of the 1500, around one third is assumed to be in the second trimester (500). As the difference between the number of women assumed to be in the second trimester and the calculated sample size was 78 (500-422), it was decided to include all available pregnant women who were in the second trimester in the study.

Data were collected by means of an oral examination and pre-tested interviewer administered questionnaire. The questionnaire was used to obtain information about socio-demographic data and subjective measures of oral health status. The oral examination included the assessment of dental caries and treatment needs for caries using the World Health Organization Basic methods and CPITN to assess periodontal treatment needs. The oral examination was conducted by the first author who was calibrated with a consultant in dental public health on the use of indices. Every 10th woman was re-examined to test for intra-examiner variability.

The first author visited all ante-natal clinics in the MOH area and based on the pregnant mother's register maintained by the public health midwives selected the eligible pregnant women for the study. The questionnaire was administered by the first author at the ante-natal clinic. This was followed by the oral examination which was also carried out by the first author while the participant was seated on a straight back chair under natural light.

SPSS 13.0 statistical package was used for data analysis. As caries levels in the sample were not normally distributed non-parametric tests (Mann-

Whitney and Kruskal Wallis) were used to determine the differences between two groups and more than two groups respectively. The associations between categorical variables were determined by chi square test.

Ethical clearance for the present study was obtained from the Ethical Review Committee of the Faculty of Medicine, University of Colombo. Written informed consent was obtained from all participants. Any woman who was identified as having oral health problems was referred to the nearest government dental clinic for appropriate management.

Results

The mean age of the sample was 27 ± 4.6 years with a range of 18-44 years. Nearly 72% of the women were housewives and 45% were in their second pregnancy. The majority (68%) was educated up to GCE Ordinary Level.

Table 1 shows the caries status according to socio-demographic variables. The mean DMFT of the sample was 5.4 ± 3.0. The average numbers of decayed, missing and filled teeth were 2.2± 2.3, 1.3±2.0 and 1.9±1.9 respectively. Caries experience as well as decayed, missing and filled teeth differed significantly according to levels of education and family income. Caries experience, decayed and missing teeth were significantly lower where as filled teeth were significantly higher in the better educated and those with a higher income. Periodontal status based on the CPITN index is given in Table 2. Nearly 59% of the sample had bleeding gums while those with shallow and deep pockets were minimal. Periodontal status also differed significantly according to levels of education and family income. Table 3 shows the normatively assessed treatment needs for caries and periodontal disease. The majority needed restorations (53%) while 37% required extractions. With respect to periodontal treatment needs 59% required only oral hygiene instructions. Of the sample, only 16% considered their oral health status to be very good/ good while 63% thought that they had an oral health problem. Nearly 43% and 21% perceived that they had decayed teeth and bleeding gums respectively. Of those who perceived that they had an oral health problem, 50% felt that they do not need care for that problem. When questioned why they did not perceive a need for their problem, 50% had stated that dental treatment during pregnancy could harm the foetus.

Discussion

As pregnancy can affect oral health and oral health status in pregnancy can have adverse pregnancy outcomes, pregnant women are considered as an important group in the population with special oral health care needs. The present study assessed the oral health status and both normative and perceived treatment needs in pregnant women in the Divulapitiya MOH area. The results indicate that the prevalence of dental caries was very high (92%) in this group of women. Confirming with the findings of a study on pregnant low-income Brazilian women, a high prevalence of untreated caries was also found.[9] Moreover, the mean caries experience was found to be 5.38 and caries levels significantly differed according to education and income levels of the women. Jago *et al.* in their study conducted as far back as 1984 have reported that the mean DMFT of pregnant women attending a maternity hospital in Brisbane, Australia was 15.8 while the mean caries experience of pregnant women attending an antenatal clinic in Lagos Nigeria was found to be as low as 1.54.[10,11] To date there is no evidence to suggest that pregnancy increases the risk of dental caries. Hence the level of caries observed in the participants of the present study as well as those reported in other studies on pregnant women may reflect the level of caries in the general population of women in the respective countries at that time. Similar to dental caries, the prevalence of periodontal diseases was also very high (93%). However, two African studies that had assessed the periodontal status in pregnant women using the CPITN had reported that the prevalence was 68%.[11,12] But

confirming with the above studies those with shallow and deep pockets were minimal. Moreover periodontal status was associated with the education and income levels of the women and supports the findings of Taani *et al* (2003).[13]

Due to the high prevalence of oral disease normatively assessed treatment needs were also high in this group of pregnant women. The majority required restorations followed by extractions as treatment for dental caries. A similar pattern was reported by Agbelusi *et al* (2000).[11] With respect to treatment needs for periodontal diseases, a majority needed only oral hygiene instructions where as Agbelusi *et al.* found that a majority of pregnant women in their study needed scale and polish.

Oral health status and treatment needs are traditionally measured using normative assessments. However, demand for care will depend on the patient's subjective assessments of his/her own oral health status and treatment needs.[14] Hence perceived outcomes of oral health are also important when assessing treatment needs. In addition to the normatively assessed treatment needs the present study also assessed perceived oral health status and perceived needs in the sample. Nearly 34% of women considered their oral health to be poor or very poor while Honkala and Ansari in their study on Kuwaiti pregnant women reported that 20% had felt that their oral health was poor.[15] Sixty three percent perceived that they had some oral condition with decayed teeth being the most common problem. However, in a study conducted on American pregnant women from three states, it has been mentioned that only 12-25% had perceived that they had an oral health problem.[16] Confirming with a previous study, of those who reported having an oral health problem, one half had stated that they would not seek dental care during pregnancy.[17] The most common reason cited for not seeking dental care during pregnancy is the belief that the foetus could be harmed by dental treatment. A similar finding has been reported by Dinas *et al* (2007).[18] This indicates that certain misconception that pregnant women have about oral health care can lead to avoidance of dental treatment during pregnancy thus having detrimental effects on both the woman and her child.

Confining this study to women in their second trimester of pregnancy can be considered a limitation. Nevertheless the study was able to provide insight into an important problem/issue that hitherto has not been investigated in Sri Lanka.

Conclusion

In conclusion, the present study revealed that the burden of caries and periodontal diseases as well as both normatively assessed and perceived treatment needs were high in this group of pregnant women. Moreover the women had certain misconceptions about obtaining dental care during pregnancy. Thus there is a need to educate women on the importance of maintaining good oral health in pregnancy.

Table 1. Caries status in the sample according to socio-demographic variables

Variable	Decayed teeth		Missing teeth		Filled teeth		DMFT	
	mean (sd)	mean rank	mean (sd)	mean rank	mean (sd)	mean rank	mean (sd)	mean rank
Age (years)#								
≤ 27 (227)	2.27 (2.3)	232.0	0.82 (1.5)	206.3*	2.02 (1.9)	237.9	5.11 (2.7)	218.7
> 27 (232)	2.21 (2.3)	228.1	1.67 (2.3)	253.2	1.78 (1.8)	222.3	5.66 (3.2)	241.0
Education #	2.54 (2.3)	251.6*	1.51 (2.1)	247.5*	1.66 (1.8)	214.8*	5.71 (2.8)	245.0*
≤ GCE O'L(310)								
> GCE O'L(149)	1.69 (2.2)	185.2	0.71 (1.5)	193.5	2.39 (2.0)	261.6	4.70 (3.2)	198.8
Income ## (rupees)								
<10,000 (172)	2.61 (2.2)	258.7**	1.60 (2.1)	252.9*	1.50 (1.8)	200.6*	5.71 (2.8)	246.8*
10,001-20,000(204)	2.06 (2.1)	221.3	1.21 (2.0)	227.1	2.10 (1.9)	246.6	5.37 (2.9)	229.4
>20,000 (83)	1.89 (2.8)	192.0	0.63 (1.4)	189.8	2.22 (2.0)	250.1	4.74 (3.5)	196.6
Total sample (459)	2.24 (2.3)		1.25 (2.0)		1.90 (1.9)		5.39 (3.0)	

Differences between groups determined by #Mann-Whitney and ## Kruskal-Wallis tests
Differences between groups significant at * P<0.01; ** P<0.05

Table 2. Periodontal status based on the CPITN according to socio-demographic variables

	CPITN 0		CPITN 1		CPITN 2		CPITN 3		CPITN 4	
Age (years)										
≤ 27 (227)	18	7.9	143	63.0	56	24.7	10	4.4	0	0.0
>27 (232)	14	6.0	127	54.7	83	35.8	6	2.6	2	0.9
					P=0.07					
Education										
≤ GCE O'L (310)	16	5.2	176	56.8	106	34.2	10	3.2	2	0.7
>GCE O'L (149)	16	10.7	94	63.1	33	22.2	6	4.0	0	0.0
					P=0.019					
Income (Rs)										
<10,000 (172)	8	4.7	90	52.3	66	38.4	7	4.1	1	0.6
10,001-20,000 (204)	12	5.9	133	65.2	51	25.0	7	3.4	1	0.5
>20,000 (83)	12	14.5	47	56.6	22	26.5	2	2.4	0	0.0
					P=0.007					
Total sample (459)	32	7.0	270	58.8	139	30.3	16	3.5	2	0.4

For statistical analysis CPITN scores 3 and 4 were combined

Table 3. Distribution of sample according to normative treatment needs for oral conditions

Treatment needs		
Caries	N	%
Restorations	245	53.4
Dentures	74	16.1
Extractions	170	37.0
Pulp care	13	2.8
Periodontal condition		
Oral hygiene instructions only	270	58.8
Scaling	155	33.8
Periodontal surgery	2	0.4

Table 4. Distribution of measures of perceived oral health status

Variable	N	%
Perceived oral health status (n=459)		
Very good/good	74	16.1
Fair	216	47.1
Very poor/poor	155	33.8
Cannot comment	14	3.1
Perceived awareness about presence of oral disease (n=459)		
Present	290	63.2
Absent	133	28.0
Don't know	36	7.8
**Perceived oral condition (n=290)*		
Decayed teeth	124	42.8
Tooth ache	40	13.8
Retained roots	33	11.4
Bleeding gums	60	20.7
Pain in gums	34	11.7
Stained teeth	36	12.4
Sensitivity	56	19.3
others	8	2.7
Perceived need for dental care for oral condition (n=290)		
Care needed	129	44.5
Care not needed	133	49.9
Don't know	28	9.6
Reasons for not perceiving a need for care (n=133)		
Could harm foetus	67	50.4
Fear of dental treatment	10	7.5
Not urgent and could postpone	56	42.1

*percentages do not add up to 100 as multiple responses were allowed.

References

1. Tilakaratne A, Soory M, Ranasinghe AW, Corea SMX, Ekanayake SL, De Silva M. Periodontal disease status during pregnancy and 3 months postpartum in a rural population of Sri Lankan women. J Clin Periodontal 2000; 27: 787-792.

2. Sooriyamoorthy M, Gower DB. Hormonal influence on gingival tissue: Relationship to periodontal disease. J Clin Periodontal 1989; 16: 201-208.

3. Pitiphat W, Joshipura KJ, Gillman MW, Williams PL, Douglass CW, Rich-Edwards JW. Maternal periodontitis and adverse pregnancy outcomes. Community Dent Oral Epidemiol 2008; 36: 3-11.

4. Saddki N, Bachok N, Hussain NH, Zainudeen SC, Sosrosena W. The association between maternal periodontitis and low birth weight among Malay women. Community Dent Oral Epidemiol 2008; 36: 294-304.

5. Acharya S, Bhat PV. Oral health related quality of life during pregnancy. J Public Health Dent 2009; 69: 74-77.

6. Caufield PW, Cutter GR, Dasanayake AP. Initial acquisition of mutans streptococci by infants: evidence of a discrete window of infectivity. J Dent Res 1993; 72: 37-45.

7. Boggess KA, Edelstein BL. Oral health in women during preconception and pregnancy: Implications for birth outcomes and infant oral health. Matern Child Health J 2006; 10: S169-S174.

8. Lwanga SK, Lemeshow S. Sample size determination in health studies. A practical manual. Geneva: World Health Organization, 1991.

9. De Oliveria BH, Nandanovsky P. The impact of oral pain on quality of life during pregnancy in low-income Brazilain women. J Orofac Pain 2006; 20: 297-305.

10. Jago JD, Chapman PA, Aitken JF, McEniery TM. Dental status of pregnant women attending a Brisbane maternity hospital. Community Dent Oral Epidemiol 1984; 12: 398-401.

11. Agbelusi GA, Akinwande JA, Shutti YO. Oral health status and treatment needs of pregnant women in Lagos state. Niger Post Grad Med J 2000; 7: 96-100.

12. Wandera S, Engebresten IMS, Okullo I, Tumwine JK, Astrom AN. Socio-demographic factors related to periodontal status and tooth loss of pregnant women in Mbale district, Uganda. BMC Oral Health 2009; 9:18 doi 10.1186/1472-6831-9-18.

13. Taani DQ, Habasheneh R, Hammad MM, Batieha A. The periodontal status of pregnant women and its relationship with socio-demographic and clinical variables. J Oral Rehabil 2003; 30: 440-445

14. Kressin NR. Symposium on self-rated assessment of oral health outcomes. J Dent Edu 1996; 60: 485-487.

15. Honkala S, Ansari J. Self-reported oral health, oral hygiene habits and dental attendance of pregnant women in Kuwait. J Clin Periodontol. 2005; 32: 809-814.

16. Gaffield ML, Gilbert BJ, Malvitz DM, Romaguera R. Oral health during pregnancy: an analysis of information collected by the pregnancy risk assessment monitoring system. J Am Dent Assoc. 2001 ;132: 1009-1016.

17. Reseeler-Maerlender J, Krishna R, Robinson V. Oral health during pregnancy: current research. J Womens Health (Larchmt) 2005; 14: 880-882.

18. Dinas K, Achyropoulos V, Hatzipantelis E, Mavromatidis G, Zepiridis L, Theodoridis T, Dovos D, Tantanasis T, Goutzioulis F, Bontis J. Pregnancy and oral health: utilization of dental services during pregnancy in northern Greece. Acta Obstet Gynecol Scand. 2007; 86: 938-944.

Research Article II

Sri Lanka Dental Journal 2008; 38(03) 124-129

Malocclusion pattern in orthodontic pateints: A hospital based study

M.A.G.P. Wimaladharma, D.P. Wettasinghe, W.M.G.I.K Wimaladharma

Abstract

Dental malocclusion is one of the commonest dental problems which patients seek treatment. Although this is true to the whole world, prevalence, incidence and orthodontic treatment seek vary to a greater extent among different nations. Incidence and prevalence of malocclusion is well documented in developed countries. But these data are very scarce in developing countries. There has been no published data of prevalence or incidence of malocclusion in Sri Lanka.

Objectives: The aim of this retrospective study was to evaluate the malocclusion patterns, and to provide quantitative information on the pattern of dentofacial characteristics among orthodontic population.

Material and methods: A total of 700 patient records were randomly selected from patient data base in the Orthodontic Unit, General Hospital, Kandy, Sri Lanka reported in the years between 2001 to 2007. (100 patients records from each year). Malocclusions were categorized into six classes according to the British Standards Institute's Incisor classification.

Results: The majority of patients presented was females aged between 11 to 15 years from Kandy district.

The highest malocclusion pattern was Class II D I (54%) and it was mainly associated with skeletal base II discrepancy (52%). Next highest was the class I malocclusion (31%). Out of them majority (28%) was associated with class I skeletal base. The rest was associated with class II skeletal base. Class III malocclusion and bimaxillary Proclination were about 5% and 6% respectively. Malocclusions associated with cleft lip and palate and Class II D II were about 2% and 2.6% respectively.

Conclusion: These results gave a detailed pattern of malocclusions and orthodontic treatment seek among orthodontic patients. This may provide base line data for planning orthodontic services. However, there is a great need of epidemiological surveys with large sample sizes to find out the prevalence of malocclusion among Sri Lankan population in order to plan orthodontic manpower and services needed.

Introduction

The prevalence of malocclusion has been the subject to debate for decades and there are little

Dr. M.A.G.P. Wimaladharma (Correspondence) BDS(SL), Registrar in Orthodontics, General Hospital, Kandy.

Dr. (Mrs) D.P. Wettasinghe MS(Col), D Orth RCS(UK), Consultant in Orthodontics, General Hospital, Kandy.

Dr. (Mrs) W.M.G.I.K Wimaladharma MS(Col), MRACDS(Aus), Lecturer, Department of Prosthetic Dentistry, Faculty of Dental Sciences, University of Peradeniya.

available epidemiological data for the incidence or prevalence of malocclusions through out the globe. The conclusions reached by the epidemiological studies largely vary in their results probably due to the lack of general agreement on what constitutes a malocclusion. As a consequence, between 1930 and 1965 the prevalence of malocclusion in USA was variously estimated as 35% to 95%. To eliminate the confusion introduced by individual judgment of malocclusion, several classification systems have been evolved. Edward Angle in 1899 classified malocclusion according to the first permanent molar relationship that had several draw backs. To overcome these drawbacks British Standards Institute's Incisor classification was formulated in 1983. However, Angle's classification with or without several modifications is still the most widely used classification to record malocclusions.[1]

Among the few first recorded studies on the frequency of malocclusion, Angle examined and classified 1,000 school children of St. Louis, Missouri in USA.[2] This sample was apparently not random but restricted to malocclusions. Therefore it was impossible to determine the overall prevalence of malocclusions from this study. In 1970s, a series of studies carried out by public health/university groups in most developed countries provided a reasonably clear picture of the prevalence of various occlusal relationships or malrelationships. In USA two large scale surveys carried out by Division of Health Statistics of the US public health service (USPHS) covered children aged 6-11 between 1963 and 1965 and youths aged between 12-17 in 1969 and 1970.[3,4] Again, as a part of national survey of health care problems and needs in the USA in 1989 – 1994 (National Health and Nutrition Estimates Survey 111, NHANES111), estimates of malocclusion were obtained. These data provided current information for the population of USA. According to this survey 30% had Angle's normal occlusion. Class1 malocclusion was 50-55% and by far the largest single group. Class I1 malocclusion was approximately 15%. Class 111 represented very small proportion of the total which is less than 1%.[5,6] Burgersdijk *et al,* (1991) studied the prevalence of malocclusion and orthodontic treatment needs in adolescents and adults in Netherlands and found crowding in the incisor region in 15%, Angle's Class II malocclusion in 28%, and an open bite greater than 5 mm in 23% of their sample.[7] According to the epidemiological studies of the World Health Organization (WHO) carried out in Hungary, the prevalence of malocclusion in 12-year olds was 40.8% in 1985 and 41.3% in 1991.[8]

Though prevalence of malocclusions in developed countries is well documented, data for developing countries are very scarce. One of the studies done in orthodontic patients in Pakistan showed, the commonest malocclusion presented was Class II DI (64%).[9]

Nonetheless, there has been no published data available for either prevalence or incidence of malocclusions in Sri Lanka.

There for Identifying malocclusions, their incidence and the treatment needs will be helpful to determine the appropriate manpower and resources needed in orthodontics.

This retrospective study was carried out to analyze the malocclusion pattern among patients who presented to the Orthodontic Unit, General Hospital, Kandy, Sri Lanka.

The aim of the study was to provide quantitative information regarding dentofacial characteristics age and gender differences and the malocclusion pattern among orthodontic patients.

Material and methods

A random sample of 700 patients history records registered in the Orthodontic Unit, General Hospital Kandy in the years between 2001 to 2007, (hundred from each year) was selected for the study. Malocclusions of the patients were

classified according to the WHO incisor classification and were confirmed by the same Consultant Orthodontist at the time of consultation. Data with reference to age, sex, district and diagnosis was transcribed from original records to a specially designed sheet for the analysis.

Assessment of the Patterns of malocclusion
According to the British Standards Institute's Incisor classification, presenting malocclusions were categorized into six classes. Class I malocclusion, Class II Division I (D I) malocclusion, Class II Division II (D II) malocclusion, Class III malocclusion, Bimaxillary Proclination (Bimax) and malocclusions associated with cleft lip and palate. All these were subcategorized according to the skeletal base. The classification of the patients was carried out by the same examiner (Consultant Orthodontist) through out the study.

Results

District Distribution

Figure 1 shows the Disribution of the sample according to districts. It shows that the highest number of patients presented to the clinic (67%) was from Kandy district. Matale, Nuwara Eliya and Kegalle were the next highest districts. Overall, there was a vast district distribution and patients represented almost all the districts of SriLanka.

Age presentation

Figure 2 gives the age distribution of the sample. Age range varies from 06 years to 35 years. However, ages were categorized into groups of five year intervals for analytical purposes. Patients aged between 11-15 years were the highest presented age group followed by 6-10 years and 16-20 years. Between 0-5 years and 31 and above were the least presented age groups.

Gender Variation

There were 427 (61%) females and 373 (39%) males in the sample.

The highest malocclusion presented was Class II D I (54%) and it was mainly associated with skeletal base II discrepancy (52%). Next highest was the class I malocclusion (31%). Out of them majority of patients of about 28% was associated with class I skeletal base. The rest was associated with class II skeletal base. Class III malocclusion and bimaxillary Proclination, almost equally presented and were about 5% and 6% respectively. Least presented malocclusion were malocclusions associated with cleft lip and palate and Class II D II and they were about 2% and 2.6% respectively.

Discussion

According to these findings, patients presented to the Orthodontic Unit, Kandy were mainly from the central province. Rest represented almost all districts of the country. Majority of the patients was between 11-15 years, which is the transitional period from deciduous to permanent dentition. This is a common age which patients seek orthodontic advices.[9] According to Mangoury and Mostafa (1990) as late as 1960s, 95% or more of all orthodontic patients were children or adolescent. From 1975 to late 1980s, it changed to adults (age 18 or older). By 1990, 25% of all orthodontic patients were adults and it remains constant since then while the number of younger patients has grown, so in the late 1990s, proportion of adult patients had dropped to 20%.[10]

Majority of the patients presented in this study was females (61%) and this data was compatible with many other studies.[9] This age and gender distribution reflects the reality that youngsters are more concerned with their appearance and also more parents attention at this age.

These data were compatible with most of the studies done in Arabian and Asian region while this is different from other European and American studies.[9,11,12,13,14] Differences in malocclusion characteristics between developed (eg; USA) and developing countries would be expected because of differences in racial, cultural and ethnic composition, and dental awareness.

Conclusion

According to this study, the frequency of Class I, Class II DI, Class II DII, Class III, bimaxillary proclination and cleft lip and palate malocclusions were found to be 31%, 54%, 5%, 6%, 2% and 2.6% respectively. This study represents the incidents of malocclusion among a restricted group of population. Identifying the occlusal problems, their prevalence and incidence and the need for treatment is essential as base line data in planning dental manpower. Hence, there is a strong need, of analyzing the prevalence of Malocclusions in the Sri Lankan population.

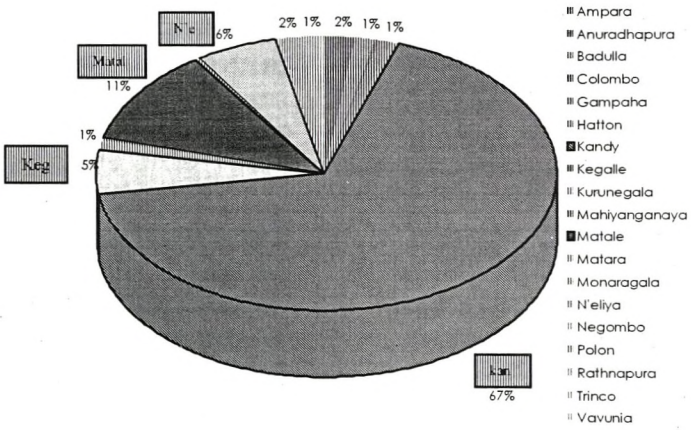

Figure 1. Distribution of the sample according to districts

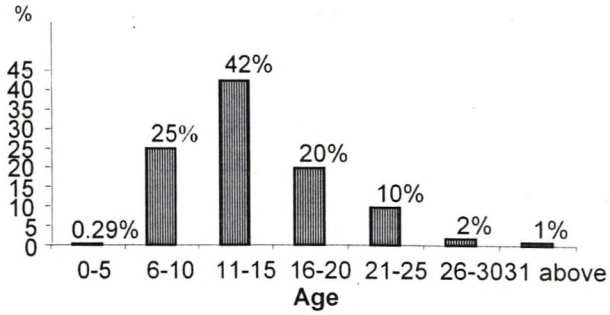

Figure 2. Age distribution of the sample

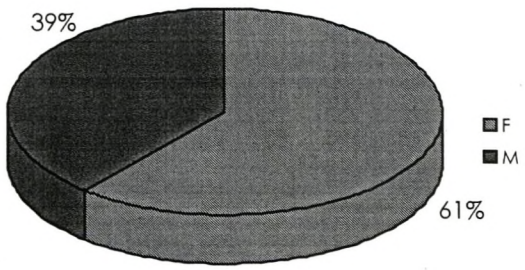

Figure 3. Gender variation of the sample

MALOCCLUSION		SKELETAL BASE 0	1	2	3	Total
CLASS I	Number	0	198	18	1	217
	Percentage	.0%	28.3%	2.6%	1%	31.0%
CLASS II Division I	Number	0	10	365	0	375
	Percentage	.0%	1.4%	52.1%	.0%	53.6%
CLASS II Division II	Number	0	3	13	2	18
	Percentage	.0%	.4%	1.9%	.3%	2.6%
CLASS III	Number	0	5	0%	30%	35%
	Percentage	.0%	.7%	.0%	4.3%	5.3%
BIMAX	Number	0	4	36	0	40
	Percentage	.0%	.6%	5.1%	.0%	5.7%
Cleft lip / palate	Number	15	0	0	0	15
	Percentage	2.1%	.0%	.0%	.0%	2.1%
Total	Number	15	220	432	33	700
	Percentage of Toal	2.1%	31.4%	61.7%	4.7%	100.0%

Table 1. Percentages of different types of malocclusion and associated skeletal patterns of the sample

Reference

1. Tang EL and Wei Sh 1993. Recording and measuring malocclusion: A review of the literature. Am J Orthod Dentofacial Orthop 1993; 103: 341-345

2. Emrich RE, Brodie AG, Blayney JR. Prevalence of Class I, Class II, and Class III Malocclusions (Angle) in an Urban Population. An Epidemiological Study. J. Dent. Res 1965; 44: 947-953.

3. Kelly JE, Sanchez M, Van Kirk LE. An assessment of the occlusion of teeth of children, DHEW 1973. Publication No (HRA) 74-1612, Washington, DC, National Center for Health Statistics.

4. Kelly J, Harvey C. An assessment of teeth of youths 12-17 years, DHEW 1977, Publication No (HRA) 77-1644, Washington, DC, National Center for Health Statistics.

5. Brunelle JA, Bhat M, Lipton JA 1988-91: Prevalence and distribution of selected occlusal characteristics in the US population. J Dent Res1998; 75: 706-713.

6. Proffit WR, Fields HW, Moray LJ. Prevalence of malocclusion and orthodontic treatment need in the United State: estimates from the NHANES-111 survey, Int J Adult Orthod Orthogn Surg 1998; 13: 97-106.

7. Burgersdijk R, Truin GJ, Frankenmolen F, Kalsbeek H, van 't Hof M, Mulder J. Malocclusion and orthodontic treatment need of 15-74-year-old Dutch adults. Community Dentistry and Oral Epidemiology 1991; 19: 64 - 67.

8. Gábris K, Márton S, Madléna M. Prevalence of malocclusions in Hungarian adolescents. European Journal of Orthodontics 2006; 28: 467-470.

9. Guel-e-Rum, Fida M. Pattern of Malocclusion in orthodontic patients: A hospital based study. J Ayub Coll Abbottabad 2008; 20: 43-47.

10. El - Mangoury NH, Mostafa YA. Epidemiologic panorama of malocclution. Angle Orthod 1990; 60: 207-214.

11. Ijas A. A Cephalometric Study to identify classification of Malocclusion in patients attending orthodontic unit of the dental section at Ch & ICH Lahore, J Park Dent Assoc 2004; 13: 130-138.

12. Onyeaso CO, Aderinokun GA, Arowojolu MO. The pattern of malocclusion among orthodontic patients seen in dental centre. University collage Hospital, Ibadan, Nigeria. Afr J of Med Sci 2002; 31: 207-211.

13. Soh j, Sandham A, Chan YH. Malocclusion severity in Asian men in relation to malocclusion type and Orthodontic treatment need. Am J Orthod Dentofacial orthop 2005; 128: 648-652.

14. Patient census survey results. Bulletin Am Assn Orthod 1997; 15: 4 (July- Aug).

Case Report

Sri Lanka Dental Journal 2008; 38(03) 130-135

Endodontic treatment of a three rooted maxillary first premolar: A case report

K.A. Wettasinghe

Introduction

A sound knowledge of normal root canal morphology with possible anatomical variations is an essential pre requisite for successful endodontic treatment of a tooth, as many of the difficulties encountered in root canal treatment are due to variations in root canal morphology. The presence of extra roots is an additional challenge which begins at diagnosis, radiological assessment and involves all stages of treatment including access cavity design, localization and shaping of the root canal system.

The incidence of three rooted maxillary 1st premolars ranges from 0% to 6% and the findings of the available studies are summarized in table.[1,2,3,4,5,6,7,8]

Three separate roots each containing a single root canal, is the most common root canal configuration of three rooted maxillary premolars. This root canal configuration resembles that of a miniature maxillary molar and the canals are classified as the mesiobuccal, distobuccal and palatal canals.[9]

Case report

A 39 year old male was referred to the general dental practice of the author for management by a colleague. The patient had developed a sinus in the palatal side of the left upper premolar region after a number of episodes of acute apical infection. The upper first premolar was diagnosed non vital and endodontic treatment commenced. There has been no improvement of the patient's condition despite two Ca $(OH)_2$ dressings for the ⌊4.The patient was well experienced in dental treatment, with a non contributory medical history.

On examination,⌊4 was not mobile but slightly tender to percussion. It had a distal class II cavity restored with composite resin and the access cavity made was sealed with Zinc Oxide Eugenol. There was a non discharging sinus on the palatal side in relation to ⌊4, without any swelling. The patient had a number of radiographs of the region and one of them clearly showed the three rooted nature of the tooth (Fig 1). The radiological appearance was similar to an upper first molar with two buccal and a palatal root. A small radioluscency in relation to the apex of the palatal root was evident. Endodontic treatment was commenced under local anesthesia with 2% lidocaine with 1:80,000 adrenaline but the patient insisted on not having a rubber dam as he has experienced an extremely unpleasant episode of choking previously.The implications of not working under rubber dam were explained to him and all files used had a length of dental floss tied to the handle as a precaution.

The access cavity was modified with an endo access bur (Dentsply Maillefer, Switzerland) to a T shaped outline. Access to the palatal and distobuccal roots have been achieved by the

Prof. K.A. Wettasinghe (Correspondence) BDS (SL) MS (Col) FDSRCS (Eng and Edin) Associate Professor in Restorative Dentistry, Department of Restorative Dentistry, Faculty of Dental Sciences, University of Peradeniya. E-mail: awettasinghe@yahoo.com

previous operator but the orifice of the mesiobuccal root was hidden under a ledge of dentine. The ledge of dentine was carefully removed and straight line access was obtained. A no.2 Gates drill (Mani, Japan) was used to prepare the coronal 1/3 of the root canals with copious irrigation of canals with 5% Sodium Hypochlorite solution. The mesiobuccal canal was negotiated with a size 10 K file using a Root ZX apex locater (J. Morita, Japan) for guidance. The apical constriction was noted at 19 mm. Apical stops were felt for the other two canals with a size 25 K file at 20 mm for the palatal root and a size 20 K file at 19 mm for the disto buccal root. A periapical radiograph was taken with the files in place and the working lengths were confirmed (Fig 2).

Biomechanical preparation of the root canals was done using Profiles (Dentsply Maillefer, Switzerland), following the manufacture's guidelines. The palatal canal was prepared to F3 whereas the smaller buccal canals were prepared to F2 size.

It was then decided to obturate the canals at the same visit and it was done using corresponding Protaper Gutta-Percha points (Dentsply Maillefer, Switzerland), and Apexit Plus (Ivoclar Vivadent, Germany) as root canal sealer. Another ZOE temporary dressing was placed for the access cavity.

A post operative radiograph confirmed complete and satisfactory obturation of the three root canals (Fig 3). The patient was prescribed Amoxycillin 250mg, Metronidazole 200mg three times for a day for 3 days. Post operative recovery was uneventful and following a review after two weeks, he was referred back to his general dental practitioner for permanent restoration of the tooth.

Discussion

The normal anatomic configuration of the maxillary first premolar is well documented in the literature, except for the small incidence of these teeth with three roots and root canals.

The precise three-dimensional determination of the internal structure of teeth, their anatomy and form clinically, is a challenge. Whenever there is any indication of different anatomy, additional radiographs should be obtained with different horizontal angles. This should be followed by careful and detailed examination of the radiographs.[10]

The anatomy of a three rooted maxillary premolar is similar to the adjacent maxillary molars and they are therefore some times called small molars or 'radiculous' premolars.[11,12] Although the pre operative radiograph gives a two-dimensional image of the three-dimensional root canal system, its interpretation reveals external and internal anatomic details that suggest the presence of extra canals and/or roots. Whenever there is an abrupt straightening or loss of a radioluscent root canal outline, an extra canal should be suspected; this could be in the same root or in other independent roots.[13] In straight-on radiographs of maxillary premolars Sieraski *et al,* (1989) found that whenever the mesio-distal width of the mid root image was equal to or greater than the mesio-distal width of the crown, the tooth most likely had three roots. Proper viewing facilities and magnification are also essential prerequisites.[9]

Based on this information the 3 rooted nature of this tooth was properly diagnosed. Preparation of a T shaped outline by extending the existing access cavity in the mesiobuccal direction as suggested by Balleri made the access easier to the mesiobuccal root which was hidden by a ledge of dentine.[14]

Conclusion

Many of the challenges and complications of root canal therapy may be directly attributed to an inadequate understanding of the anatomy of the root canal system. It's also likely to fail if extra roots or/ root canals are not assessed and obturated. Clinicians should be aware of both the common and rare anatomical variations and apply

this knowledge in interpreting radiographs which may have to be repeated.

Table 1. Incidence of three rooted maxillary frist premolars : Summary of reported studies

Author (year)	One Root (%)	Two Roots (%)	Three Roots (%)
Ingle (1965)	43.0	55.0	2.0
Carns and Skidmore (1973)	22.0	72.0	6.0
Vertucci and Gegauff (1978)	26.0	70.0	4.0
Pecora *et al.* (1991)	55.8	41.7	2.5
Loh (1996)	49.4	50.6	0.0
Kartal *et al.* (1998)	37.3	61.3	1.3
Chaparro *et al.* (1999)	40.0	56.7	3.3
Atieh (2008)	17.9	80.9	1.2

Figure 1. Periapical radiograph to show the tree rooted nature of the tooth.

Figure 2. Periapical radiograph with files in place for the purpose of confirming the working length.

Figure 3. Post operative radiograph to show the satisfactory obturation of root canals.

References

1. Ingle JI. Endodontics. Philadelphia: Lea & Febiger; 1965.

2. Carns EJ, Skidmore AE. Configurations and deviations of root canals in Maxillary premolars. Oral Surg 1973; 36: 880-886.

3. Vetucci FJ and Gegauff A. Root canal morphology of the maxillary first premolars. J Am Dent Assoc 1979; 99: 194-198.

4. Pecora JD, Saquy PC, Sousa Neto MD, Woelfel JB. Root form and canal anatomy of maxillary first premolar. Braz Dent J 1991; 2: 87-94.

5. Loh HS. Root morphology of maxillary first premolars in Sigaporeans. Aus Dent J 1998; 43: 399-402.

6. Kartal N, Ozelik B, Cimilli H. Root canal morphology of maxillary Premolars. J Endod 1998; 24: 417-419.

7. Chaparro AJ, Segura JJ, *et al.* Number of roots and canals in maxillary first premolars. Endod Dent Traumatol 1999; 2: 65-67.

8. Atieh MA. Root canal morphology of maxillary first premolars in a Saudi population. J Cont Dent Prac 2008;9: 1-7.

9. Sieraski SM, Taylor GT, Kohn RA. Identification and endodontic management of three canalled maxillary pemolars. Journal of endodontics 1989;15: 29-32.

10. Soares JA, Leonardo RT. Root canal treatment of three- rooted maxillary first and second premolars-a case report. International endodontics Journal 2003; 36: 705-710.

11. Maibaum WW. Endodontic treatment of a radiculous maxillary premolar: a case report. General Dentistry 1989; 37: 340-341.

12. Goon WWY. The radiculous maxillary premolar: recognition, diagnosis and case report of surgical intervention. Northwest Dentistry 1993; March/April: 31-33.

13. Jafarzadeh H. Endodontic treatment of bilaterally occurring three-rooted maxillary premolars: A case report. New Zealand Dental Journal 2007; 2: 37-38.

14. Balleri P, Gesi A, Ferrari M. Primer premolar superior com tres raices. Endodontic Practice 1997; 3:13-15.

Instructions to Authors

The Sri Lanka Dental Journal publishes the following categories of articles which have relevance to Dentistry and allied sciences.

1. Leading articles - One article per issue. It may be solicited by the Editor. Authors are welcome to submit leading articles on current topics of interest. One's expertise or commentaries on general practice etc. They should be approximately 1500 words in length. References should be 20 or less.

2.Reviews - Reviews are detailed surveys of published research pertinent to dentistry and associated sciences. They should be critical in nature and should not normally exceed 3000 words and 30 references.

3. Research articles - Articles resulting from research work belong to this group. Results from routine clinical examinations or laboratory investigations will not be considered under this category. Subjects may vary from clinical trials to basic science research, historical analysis to dental economics. They should not exceed 3000 words and 30 references. A reasonable number of tables and illustrations will be accepted.

4. Short reports - These include reports on current topics, modified techniques. new materials, practice management etc. Interesting results from routine, clinical work or laboratory ill vestigations also may be accepted.

5. Case reports - Reports such as of rare diseases or conditions. Modifications to accepted treatment procedures, new management methods etc. may be included in this category.

6. Letters to Editors - Subjects unlimited, but may include short critique of published papers in the SLDJ.

7. Miscellaneous topics - Subjects unlimited and the format are free. These may also include details of scientific meetings, conferences, annual sessions, examinations, news and views, visits and obituaries.

8. Proceedings of annual sessions - Abstracts from annual sessions of the SLDA and other colleges will be published under this category.

Submission of manuscripts

Authors submitting a paper do so on the Understanding that no part has been published before. that it is not being considered for publication elsewhere and that it has been read and approved by all the authors.

Manuscripts including Tables and Figures should be sent in triplicate as the work will be reviewed by two or more referees. While papers are subject to editing. The journal does not hold it responsible for statements made by the contributor. The author alone is responsible for the statements made in his paper.

Submission of a manuscript means that authors automatically agree to assign exclusive copyright to the Sri Lanka Dental Association if and when the manuscript is accepted for publication.

Manuscript on disk

Once an article has been accepted for publication. the author will be asked to supply a copy of the final manuscript on disk together with two copies of the complete manuscript. Every disk must be clearly labelled with the name of the author, title, software and program version number.

Manuscript style

The manuscripts should be typed, double-spaced: on A4 (2 I 2x297 mm) paper and submitted in correct English: both English and American spelling are acceptable, provided this is consistent throughout the manuscript.. Manuscripts not submitted in proper format or in poor English may be returned without review. The format of a manuscript should be as

follows: Title page. Abstract. Introduction, Material and methods. Results. Discussion. Acknowledgements. References. Figure legends. and Tables, arranged in that order.

Title page - The title page should contain the following information in the order given: 1) a concise but informative title; 2) author's full names' (without degrees and titles): 3) author's institutional affiliations: 4) a running title. not exceeding 40 letters and spaces; 5) name, address, telephone, telefax and electronic mail address of the author responsible for correspondence.

Abstract page - Original and review articles must contain an abstract of approximately 250 words with four specified subtitles:

I) Objective: An introductory sentence indicating the objective and purpose of the study.

2) Material and methods: A description of experimental procedure including applicable statistical evaluation.

3) Results: A summary of the new. Previous unpublished data and results.

4) Conclusion: A statement of the study's conclusion 3-5 key words according to Index Medicus should be provided.

Introduction - The introduction should carry sufficient background information on the subject of study.

Material and methods - Procedures should be described in such detail as to make it possible to repeat the work. Subheadings may be used to improve clearness. Correct unit abbreviations should be used (e.g.; "h", "min", "s" and "Fm" rather than "hr", "minutes", "sec" and "Fl". respectively).

The authors should consider the ethical aspects of their research and ensure that the work has been approved by an appropriate Ethical Committee. Where applicable, a copy of the ethical clearance certificate should be attached. In human experimentation. informed consent from individuals should be Obtained and this should preferably be stated.

Statistical analysis - Since many scientific investigations rely on statistical treatment, authors are strongly urged to consult a person with in-depth statistical knowledge. Manuscripts with a clear element of statistics are regularly refereed by the Journal's statistics consultant.

Results - The Results section should clearly and concisely present the findings of the research, as a rule in the past tense without subjective comments and reference to previous literature. The results should be supported by statistical or illustrative validation. For the sake of clarity this section may have subheadings.

Discussion - This section should present the interpretations of the findings and is the only proper section for subjective comments. Authors are strongly urged to avoid undue repetition of what has been reported in Results.

Tables - The tables should be numbered in the order" of appearance in Arabic numerals, Each table should have a brief explanatory title. Each table; should be typed on a separate sheet, with due regard to the proportion of the printed column/page.

Figures - All graphs, drawings, and photographs are considered figures and should be numbered in the order of appearance in Arabic numerals. Each figure should have a brief and specific legend, and all legends should be typed together on a separate sheet of paper. Photographs should be glossy prints and the reverse should give the figure number, title of paper principal author's name and have a mark indicating the top. Colour illustrations may be submitted in instances where their use may contribute significantly to the scientific value of the article. Colour illustrations may be printed free of charge at the Editor's discretion, whereas others may be printed at the author's expense.

References - References are listed double-spaced in a separate reference section immediately following the text. References are numbered consecutively in the order in which they appear in the text; do not, alphabetise. Identify references in texts, tables and legends by Arabic numerals (within parenthesis).

Congress abstracts should not be used as references nor may "unpublished observations" and "personal communications" be placed in the reference list. References cited as "in press" must have been accepted for publication and not merely in preparation or submitted for publication

Examples of correct. forms of references are given below. These are based on the format used in the *Index Medicus*. Abbreviate journal names according to the *List of Journals Indexed*, printed annually in the January issue of *Index Medicus*. List all authors; do not use *et al.* in the reference list.

Journals

Standard journal article

Bartlett IG, O'Keefe P. The bacteriology of the perimandibular space infections. JOral Surg 1979; 37: 407409.

Corporate (collective) author

WHO COLLABORATING CENTRE FOR ORAL PRECANCEROUS LESIONS. Definition of leukoplakia and related lesions: an aid to studies on oral precancer. Oral Surg Oral Med Oral Pathol 1978; 46: 518-539.

Unpublished article

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

Books and other monographs

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

Chapter in book

Boyde A. Amelogenesis and the structure of enamel. In: Cohen B. Kramer KH (eds). Scientific Foundations of Dentistry. William Heinemann Medical Books Ltd. London. 1976: 335-352.

No authror given

International statistical classification of diseases and related health problems, 10th revision, vol 1. Geneva: World Health Organisation, 1992; 550--564

GlaxoSmithKline

Panadeine

Paracetamol 500 mg + Codeine 8 mg

Has the power to relieve pain


Signal
TOOTHBRUSH