Sri Lanka Dental Journal

Volume : 45
Number : 01
April : 2015

SRI LANKA DENTAL ASSOCIATION

The Official Publication of the Sri Lanka Dental Association

ISSN 1391-07280

Protect your teeth to enjoy your food

Most of us like to indulge in high starch food like kottu, egg rotti and pizza "for a change". The sugars left around teeth by starchy food enable bacteria to form sugar acids, causing tooth decay.

Clove oil enriched Super Active Power Foam in New Clogard

fights 99% of germs*

helping to prevent sugar acid formation.

Kills bacteria **Fights sugar acid formation**

The No.1 Sri Lankan cavity protector*

SLDA ACCEPTED SRI LANKA DENTAL ASSOCIATION

SLS 275

*Tested under lab conditions. Contains fluoride that helps prevent the demineralization of teeth caused by sugar acids.
*LMRB Household Panel 2014
*Original Clogard is endorsed by the Sri Lanka Dental Association.

HEMAS

Rs. 20/- upwards

Sri Lanka Dental Journal

Volume 45 **Number 01** **April 2015**

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. Ruwan D Jayasinghe, Senior Lecturer, Dept. of Oral Medicine and Periodontology, Faculty of Dental Sciences, University of Peradeniya, ruwanduminda@yahoo.com, Tel: 0777 373689 and Dr. Induwara Goonaratne, Senior Lecturer, Dept. of Forensic Medicine, Faculty of Medicine, University of Peradeniya, induwarag@yahoo.com

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 - Udahamulla, Nugegoda.
Tel/Fax : 011 - 2836190

CONTENTS

Sri Lanka Dental Journal 2015; 45(01) 1-3

Sri Lanka Dental Journal

Volume 45 **Number 01** **April 2015**

EDITORIAL

Ethical and Legal Consideration on Plagiarism in Scientific Publications

Introduction

This article focuses on an imperative issue in the arena of scientific publication known as 'plagiarism'. The aspects of 'plagiarism' and its role in academic and scientific writing has been a concern for many years. The preeminent aim of the article is to raise key legal and ethical issues that are akin to plagiarism in scientific writing.

Scientific publications or university education in Sri Lanka in particular does not seem to embrace or demonstrate a significant impetus on the issue of plagiarism. Although referencing is advocated in Sri Lankan universities in scholarly work, it neither seems that Sri Lankan universities or local scientific publications take adequate steps to teach/train students and researchers on this all important aspect, nor any attempt is seen to be made by thesis examiners or journal editors to purchase or employ a plagiarism detecting objective mechanism. None of the journals published in Sri Lanka seem to have their own plagiarism detecting technology ,nor any university in Sri Lanka subject their local publications or academic writings or thesis to such plagiarism detection. Invariably, Sri Lanka , then, depends on traditional ways in identifying plagiarised events. Such traditional approaches include but not limited to the reviewer or the examiner himself detecting such plagiarism on the basis that the reviewer or the examiner is considered to be an expert in the field and is expected to have read "all" literature in the

field of interest. However, this method will, if at all,arguably detect "bulk" or total copying of some other's work. This does not allow detection of subtle or 'idea plagiarising' as it is not practical for any expert or reviewer to keep all ideas and their origins in mind. Therefore, an acceptable mechanism to detect plagiarism in scientific publications is required[i].

What is Plagiarism ?

Plagiarism is a form of academic, research or publication misconduct; an unethical practice. In some institutions, plagiarism is prohibited by rules, making such instances illegal. Plagiarism is a form of cheating; therefore, it can be considered an illegal action that is punishable. More than it is illegal, plagiarism is a misconduct, a malpractice and a form of unethical practice by a student, academic or a researcher. In plagiarism one would use another person's ideas or work to obtain undue or undeserved advantage without due credit to the original authors. When original authors are not acknowledged in a publications, such ideas or work plagiarised reach the audience as if they are of the new author. Therefore, in scientific writing it is a convention that authors give due credit and acknowledgement to the original authors. This includes any material or idea from any source including texts, internet or visuals[ii].

In the scientific realm, when an article or a book is published it enters in to the public domain. Just because some ideas are in the public domain, one cannot use them directly for a publication without providing cross reference to the source. Coping the text as it is from the original source even with due referencing constitutes the offence of plagiarism. Either one can use direct quotations, paraphrase or summarise the idea with due credit to the original authors for it to be acceptable. If these conventions are not met, the article will be found to be plagiarised. Sometimes some ideas or processes are patented or copyrighted, in such instances, researchers may have to contact the owners directly for permission before using such copy right or patented contents[iii].

Detecting Plagiarism

By conventional methods reviewers, examiners or editors of journals had to manually look for plagiarism. It is not only a tedious task but is an incomplete and a futile task . Reviewers are not capable of keeping all relevant ideas in original forms in their minds. Expecting or claiming such a skill is beyond imagination. The declaration, the author makes to the journal concerned in regard to a scientific paper or the university concerned in regard to a thesis constitutes ample evidence and proof that the paper does not contain materials that are plagiarised. Every researcher and academic is expected to secure this generic skill of scientific writing and professionalism. Therefore traditionally scientific journals assume reliance and trust on the researchers and authors. However with increasing publications, research frauds, commercialization of science warranted journals and universities to be vigilant on issues such as plagiarism[iv].

Now, there are very advanced plagiarism detecting softwares available. One such program is Turnitin and the other is CrossCheck which uses iThenticate software. They are two that can be recommended. Although these software are expensive, the investment is worthwhile as invariably they improve the quality of the journal or the article. What these software does is, it access all possible online data bases and other internet sources and checks for similarities within the article submitted. If the program identifies similarities of writing or ideas elsewhere in the internet or online data base, then such similar texts will be highlighted and provided with the respective source in the internet or data base. It also provides a similarity index or an originality index which shows the percentage of similarities or plagiarised portions. There is no consensus on an acceptable percentages. The editor or the reviewer has to carefully go though the contents and see if the similarities are genuine,unavoidable or deliberate. It is a

tedious task for the editor or the reviewer. Due to the lack of knowledge and skills on the aspect of both scientific writing and plagiarism issues, local articles tend to get higher percentages of plagiarism. However a careful evaluation of the similarities by a content expert and editor is needed to opine in regard to a case of plagiarism[v].

These software only detects from internet sources or internet accessible data bases or repositories. They do not have capacity to cross check unpublished sources or sources that are published in hard copies but not available in the internet. Therefore, even if a paper goes through a plagiarism checker does not mean that the piece of work is authentic or original.

There are many plagiarism checkers available for low prices, however, they are not recommended as their access to data bases is limited. One may use such a cheap checker and get an acceptable result yet if they use a recommended program such as one described above, then, it may reveal a different or rather higher similarity index.

Consequences

As all reputed journals are very concerned about issues on plagiarism, it is inevitable that the paper is rejected from the journal in the event it detects plagiarism. The author can be blacklisted and the reputation of the author and the institute will be at risk. The editor may inform the situation to the institute where the researcher or author is affiliated and the institute concerned may take disciplinary action against the author as plagiarism is considered a serious academic misconduct. Legal issues may also arise when some of the patented or copy righted materials such as pictures figures are used without due permission of the owners of such material. Original authors have the legal right to sue the plagiarist and claim compensation which engender an embarrassing situation and a risk of expulsion from the job on top of paying damages to original authors. Neither ignorance nor the calibre of the person shields an author from ethical and legal ramifications attached to plagiarism. If it is a student, then the studentship will also be at jeopardy.

The author is primarily responsible to submit an article with no plagiarism. Where the author has used ideas or statements of others, due reference and citations ought to be given. However, there is arguably a responsibility with the reviewer and the editor to consider the issue of plagiarism and inform authorities when a case is doubted.

Conclusion

In scientific writing plagiarism is considered a serious academic misconduct. The bench mark in scientific or academic writing is that one gives due credit and reference to other authors and sources when ideas and materials are mobilised and utilised from others. Originality of one's work and the reflection of honesty within the work published are pivotal quality markers of scientific publications. There are advanced commercially available softwares and programs that detect plagiarism. All scientists, academics and researchers should be aware of acceptable approaches to scientific writing and about various methodologies of plagiarism detections. There are serious academic, ethical and legal ramifications connected to plagiarism and should be totally avoided in scientific writing/ publishing.

Induwara Gooneratne & Ruwan Jayasinghe
Editors
Sri Lanka Dental Journal

i. Caroll, A J. (2007) Handbook for detecting Plagiarism in Higher education Oxford Centre for Staff and Learning Development. Oxford: 1-10
ii. Vishniac, E T. (2012). What is Plagiarism. American Astrological Society Proceedings. 13
iii. Melville, B N. Copyright Publications. Colombia Law review. 56 (2)185-202
iv. Declan, B (2010).Journals Step up Plagiarism Policing. Nature 466:167
vi. Bazdaric,K (2012) Plagiarism Detection - Quality Management Tool for all scientific Journals. Croat. Med. J:53:1-3

Research Article I

Sri Lanka Dental Journal 2015; 45(01) 4-13

Hepatic and Hematological Effects of Antiepileptic Treatment in Patients with Trigeminal Neuralgia - A Retrospective Study

D.K.V.P. Senarathna, R.D. Jayasinghe
M.A.M. Sitheeque, R.W. Pallegama, S. Nanayakkara and S.B. Athauda

Abstract

Main management option in trigeminal neuralgia (TN) is medical and antiepileptics are the drugs of choice. The aims of the present retrospective study were to evaluate the hepatic and hematological adverse reactions of carbamazepine in a sample of patients with primary TN and to assess the influence of the starting dose of carbamazepine on the time taken to cause adverse reactions. Adult patients who were treated at the Oral Medicine clinic of the University Dental Hospital, Peradeniya with a minimum follow up period of 6 months and had carbamazepine as the starting drug were included in the study. Data were retrieved from the clinical records and the reports on hepatic and hematological investigations were reassessed. Log- Rank test was used to compare the survival distributions where occurrence of adverse reaction was considered as the event. 142 patients (with 54.2% of females) with a mean age of 57.2 ±12.7 years were included in the study. Majority (52.1%) were between 41-60 years. Right side of the face (69.7%) and the maxillary branch (42%) were commonly affected. Only 59 (41.5%) and 32 (22.5%) patients exhibited hepatic and hematological changes respectively. The mean follow-up time taken to observe hepatic changes was significantly higher (mean 137.09 months with 95% CI 100.1, 174.0) in patients whose starting dose was 100 mg. Only 2 patients had a total white cell count of less than $2x10^9$. The results suggest that monitoring the liver function in patients treated with carbamazepine regularly throughout the treatment period is justified as the mean time taken to induce adverse reactions can be considerably high.

Keywords: Trigeminal Neuralgia, Hepatic Effects, Hematological Effects, Antiepileptic Treatment.

Introduction

Several medical and surgical treatment options are available for trigeminal neuralgia (TN). Even though surgical treatment options including microvascular decompression and ablative

Dr. D.K.V.P. Senarathna Registrar in OMF Surgery, Dental Hospital, Peradeniya.

Dr. R.D. Jayasinghe (Correspondence) Senior Lecturer and Consultant, Department of Oral Medicine and Periodontology, Faculty of Dental Sciences, University of Peradeniya, Peradeniya. Tel: 0777 373 689 E-mail: ruwanja@pdn.ac.lk

Prof. M.A.M. Sitheeque Professor, Department of Oral Medicine and Periodontology, Faculty of Dental Sciences, University of Peradeniya, Peradeniya.

Dr. R.W. Pallegama Senior Lecturer, Department of Basic Sciences, Faculty of Dental Sciences, University of Peradeniya, Peradeniya.

Dr. S. Nanayakkara Senior Lecturer and Consultant, Department of Oral and Maxillofacial Surgery, Faculty of Dental Sciences, University of Peradeniya, Peradeniya.

Dr. S.B. Athauda Consultant haematologist, General Hospital, Kandy, Sri Lanka.

treatments have been used to manage trigeminal neuralgia, their use is limited to patients who are not responding to drugs or when the drugs cause severe adverse reactions[1]. Medical management with drugs is the treatment of choice. First line of drugs include carbamazepine and oxcarbazepine, and the second line includes gabapentine, baclofen and lamotrigine, whereas other drugs such as clonazepam, phenytoin, valproate or topiramate are used but with less effectiveness[1,2]. Although the standard treatment is with antiepileptic drugs[3], non-antiepileptic drugs have also been attempted[4]. However, data is scarce on the long term effects of the use of antiepileptic drugs in managing patients with trigeminal neuralgia.

Several systematic reviews support pharmacological management for trigeminal neuralgia. But, the outcome of studies carried out in this respect is inconsistent owing to the fact that the disease is rare[5], natural history of TN is variable, diagnosis is mainly on clinical features than investigative findings and that difficulties exist in conducting controlled trials[1]. The available evidence (level A) suggests carbamazepine to be the drug of choice. But a significant number of patients develop adverse effects with its use including vertigo, sedation, ataxia, diplopia and hematological and liver changes[6,7]. Most patients tolerate these side effects without having the need to discontinue medication. Oxcarbazepine is having level B evidence, whereas the other drugs that are used in the management of TN including gabapentine, baclofen, lamotrigine clonazepam, phenytoin, valproate or topiramate have only level C evidence for their effectiveness[2]. For the patients who are not getting effective pain control with carbamazepine or for the patients who cannot tolerate carbamazepine, a combination of drugs is observed to be beneficial[2,8].

Liver plays a pivotal role in metabolizing antiepileptic drugs. Diseases of the liver can affect the metabolism of antiepileptic drugs hence dose adjustments may be necessary in patients with liver pathology[9]. Antiepileptic drugs also induce changes in liver function[9,10]. Liver enzymes such as aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) are used as reliable markers of hepatocellular injury[9]. Liver injury with carbamazepine may occur due to direct drug injury or as a hypersensitive reaction[9]. Hepatotoxicity is reported to be highest with valproate followed by carbamazepine, whereas it's low in phenytoin[10].

A reduction in mean white cell count, hemoglobin level and red blood cell count and increase in the size of a red blood cell are observed with carbamazepine and oxcarbazepine treatment[11]. Other antiepileptic drugs are also likely to induce similar changes. The rate of development of hematological changes is observed to be independent of the type of antiepileptic drug[12]. Reports of serious side effects like aplastic anemia are rare with antiepileptic drug treatment[11,12]. Starting dose of 100mg carbamazepine two or three times a day is sufficient for pain management in some patients with TN but for the remaining TN patients, the daily dose of carbamazepine should be increased by 100 mg every other day until adequate pain relief is achieved[13].

The present study aimed to evaluate the hematological and hepatic effects of carbamazepine in a sample of patients with trigeminal neuralgia and to identify the relationships between hepatic and hematological changes and age, sex, duration of treatment and combination of treatment. The hypothesis that the starting dose of carbamazepine has an influence on the time taken to cause hepatic and hematological changes was tested.

Materials and Methods

This retrospective study was carried out at the Oral Medicine clinic of the Dental Hospital (Teaching), Peradeniya. This is the only

specialized oral medicine clinic in the country, and patients are referred to this clinic from many primary care hospitals and clinics from all over the country.

Adult patients diagnosed with primary trigeminal neuralgia (TN) were selected by perusal of all clinical records of patients diagnosed with TN at the Oral Medicine clinic. Diagnostic criteria adopted by the International Headache Society[14] were used to diagnose classical trigeminal neuralgia cases which include the following features. 1). At least 3 attacks of unilateral facial pain fulfilling criteria 2 and 3; 2). Occurring in one or more divisions of trigeminal nerve, with no radiation beyond the trigeminal distribution; 3). Pain has at least 3 of the following four characteristics: recurring paroxysmal attacks lasting from a fraction of a second to 2 minutes, severe intensity, electric shock-like, shooting, stabbing or sharp in quality, precipitated by innocuous stimuli to the affected side of the face; 4). No neurological deficit is clinically evident; and 5). Not accounted for by another diagnosis. (IHS Classification[14])

The inclusion criteria were as follows: 1) patients over 20 years of age; 2) having a minimum follow up period of 6 months; 3) treated only with antiepileptic drugs at the outset; and 4) the availability of the complete clinical records including results of hematological and liver function investigations. The exclusion criteria were: 1) patients with secondary TN; 2) patients treated with antiepileptic drugs for more than one month by primary care doctors before being referred to this clinic; 3) patients treated with surgical modalities, Botox or any other alternative treatments; 4) patients who were under other medications for long term; and 4) presence of any ambiguity in records or notes.

Clinical records of all the selected patients were re-examined and the following data were retrieved. 1) gender, 2) age at the diagnosis, 3) any other medical issues, 3) alcohol consumption,4) any other current medication (other than for TN), 5) trigeminal nerve branch, involved 6) follow up period, 7) the type of drug/s, 8) the starting dosage of the drug, and 9) the duration on the drug 10) The hematological investigations (Full blood count/ FBC) and liver function test (LFT) reports were reassessed and findings were recorded. All selected patients had been initially treated with different doses of carbamazepine alone.

The following standared investigations as specified below are routhined carried out on all TN patients treated with antiepileptic drugs at our center. Tests are performed using standard protocols by qualified technical officers. Hematological investigations (hemoglobin concentrations, mean corpuscular volume, packed cell volume, mean corpuscular hemoglobin concentration, white blood cell count, differential count, and platelet count) are carried out at fortnightly intervals in the first 3 months of treatment and once every 3 months thereafter. Liver function tests (alkaline phosphatase [ALP], gamma glutamyl transpeptidase [GGT], aspartate transaminase [SGOT/AST], alanine transaminase [SGPT/ ALT]) are carried out once in 2-3 months. The results of the investigations are interpreted using the standard reference values used by the laboratory. If any patient demonstrates any changes either in any one of the liver function tests, the time taken to observe the change (the event) is recorded in months. For those who have not shown liver changes the total duration of the follow-up was noted. If a liver change or hematological change is noticed, the dosage of carbamazepine is adjusted according to the occurrence and severity of side-effects. Any other drug related complications was also recorded. And the patients was reviewed fortnightly for the first three months and then at one month intervals.

All these information were retrieved from the clinical records by the principal investigator

using a specifically designed data collection sheet. The decisions to include or exclude patients in the study sample were ratified by the second co-investigator. Ethical Clearance for the study and for the use of archival data was obtained from the Research and Ethics Review committee of the Faculty of Dental Sciences, University of Peradeniya. The study conformed to the 2008 amended guidelines of the Declaration of Helsinki on Ethical Principles for Medical Research Involving Human Subjects[15].

Statistical Analysis

Data were managed using Microsoft Excel and statistical analyses were performed using SPSS for Windows version 12.5. Data were examined using explorative statistics and descriptive statistics were calculated. Measurements made on ratio scales were compared using independent sample t test. Chi-square tests were used to compare proportions.

The observation of liver changes (i.e., observing a higher level in one or more of the liver function tests performed compared to the standard reference values) and hematological changes (i.e., lower levels in one or more of the hematological investigations) were considered as the events. The null hypothesis that the starting dosage of carbamazepine used to treat trigeminal neuralgia had no influence on the time taken for hepatic and hematological changes was tested by comparing the survival curves using the Log-Rank test in two separate analyses. The statistical significance was accepted at $\alpha=0.05$.

Results

The study included 142 patients with classic TN treated at the clinic from 1998 to 2014 (a 17-year retrospective study period). There were 77 (54.2%) women with a female-to-male ratio of 1.18:1. The age range was 29 to 86 years with an average age of 57.2±12.0 years. The mean ages of male (57.7±12.7 years) and female (56.7±11.5 years) patients were not statistically different. The age distribution of the entire sample is illustrated in figure 1. In the total sample, 11 (7.8%), 74 (52.1%), 52 (36.6%) and 5 (3.5%) patients were within 29-40, 41-60, 61-80 and 81-86 age groups respectively.

Right side of the face was commonly affected (69.7%) than the left side. Maxillary branch involvement was observed in 60 patients followed by mandibular branch involvement (41 patients), whereas 41 patients had more than one branch involvement. In the overall sample, the involvement of the divisions of the trigeminal nerve is illustrated in Table 1. The data revealed a clear predilection of occurrence on the right side (70%).

There were 33 patients who were treated with 100mg of carbamazepine, 82 patients treated with 200mg, 14 patients treated with 300mg and 12 patients treated with 400 mg, three times a day as the starting dosage regimens. Only 59 patients (41.5%) included in the study exhibited hepatic changes as reflected in one or more liver function tests. Among them 37 (62.7%) patients' had increased levels of all ALP, GGT, SGOT and SGPT, and only 11 patients had increased levels of only one marker. Only three patients (out of 33) with 100mg, 37 patients (out of 82) with 200mg, only 9 patients (out of 14) with 300mg and only 9 patients (out of 12) with 400mg

Table 01. Involvement of different divisions of the trigeminal nerve in the sample of patients

Division of the Trigeminal nerve	Right side (70%)		Left side(30%)	
	Single branch	Both	Single branch	Both
Maxillary	28%	20%	14%	9%
Mandibular	22%		7%	

exhibited hematological changes. The Kaplan-Meier estimates for the mean survival times (in terms of time taken to hepatic changes) with 95% confidence intervals are shown in table 2.

(Chi-Square=16.6, P=0.001). The Kaplan-Meier survival curves for this comparison are illustrated in figure 1.

Table 2. Kaplan-Meier estimates for the mean survival times in months (for time to hepatic and hematological changes)

Dosage Group	Number of participants	Mean time for hepatic changes			Mean time for hematological changes		
		Estimates	95% CI		Estimates	95% CI	
			Lower Bound	Upper Bound		Lower Bound	Upper Bound
100mg	33	137.09	100.15	174.03	104.36	85.64	123.08
200mg	82	70.62	50.07	91.16	137.14	118.68	155.59
300mg	14	26.69	9.21	44.19	24.41	9.33	39.49
400mg	12	56.11	23.42	88.79	126.86	58.11	100.27

95%CI, 95% confidence intervals

The log-rank test revealed a statistically significant difference between survival rates(for time to hepatic changes) over time for different starting doses of carbamazepine

Thirty two patients (22.5%) had exhibited lower levels on one or more of the hematological indices, although this number is relatively less compared to the number of patients who

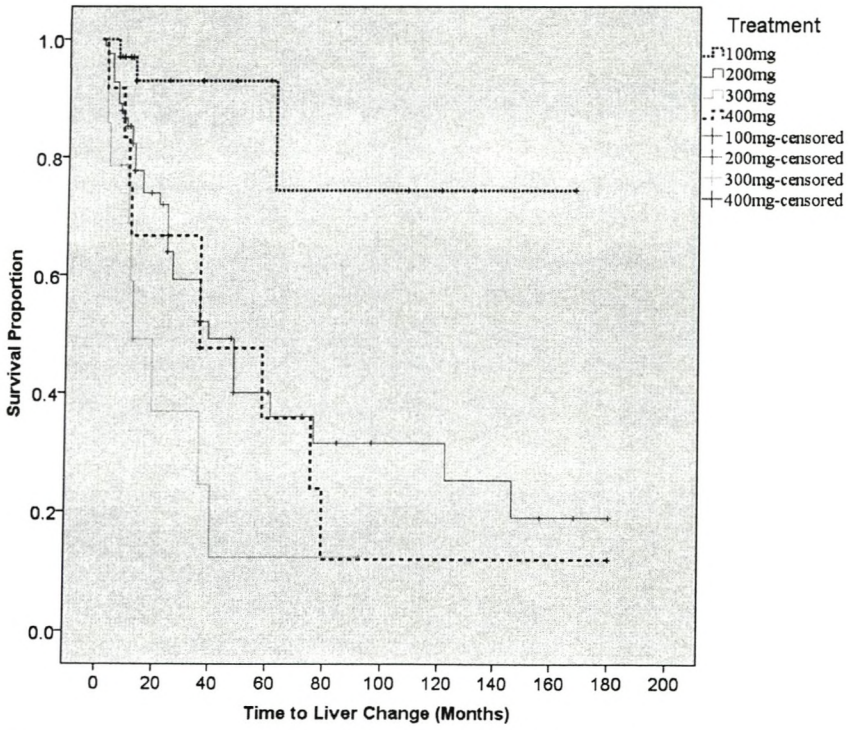


Figure 1. Kaplan-Meier survival curves for the comparison of time to liver change with different starting doses carbamazepine.

exhibited hepatic changes. Only three patients (out of 33) with 100mg, 17 patients (out of 82) with 200mg, only 10 patients (out of 14) with 300mg and only 2 patients (out of 12) with 400mg had hematological changes. A higher percentage of patients treated with carbamazepine 200 mg or 300mg three times a day exhibited higher occurrence of hematological changes (Chi-square = 9.8, P=0.02). But, only 2 patients had a total white cell count of less than 2x109. The Kaplan-Meier estimates for the mean survival times (in terms to time to hematological changes) with 95% confidence intervals are shown in Table 2.

The log-rank test revealed a statistically significant difference between survival rates (time to hematological changes) over time for different starting doses of carbamazepine (Chi-Square=22.8, P=0.001). The Kaplan-Meier survival curves for this comparison are illustrated in figure 2. It reveals that a dose of 300mg carbamazepine takes a significantly shorter time to induce hematological changes compared to other starting doses, although these observations should be interpreted with caution owing to the smaller number of participants and lesser number of events in the 400 mg group.

Chi-square tests revealed no significant associations of age group, gender, side of face, presence of other medical problems, and other drugs or consumption of alcohol with the observed changes in liver function tests.

Among the entire sample, (Table 3) there were only 74 (52.1%) who did not have either hepatic or hematological changes. There were 9 (6.3%) patients who showed only hematological changes and 36 patients (25.4%) who had only hepatic changes. The sample had only 23 (16.2%) patients who showed both hepatic and hematological changes.

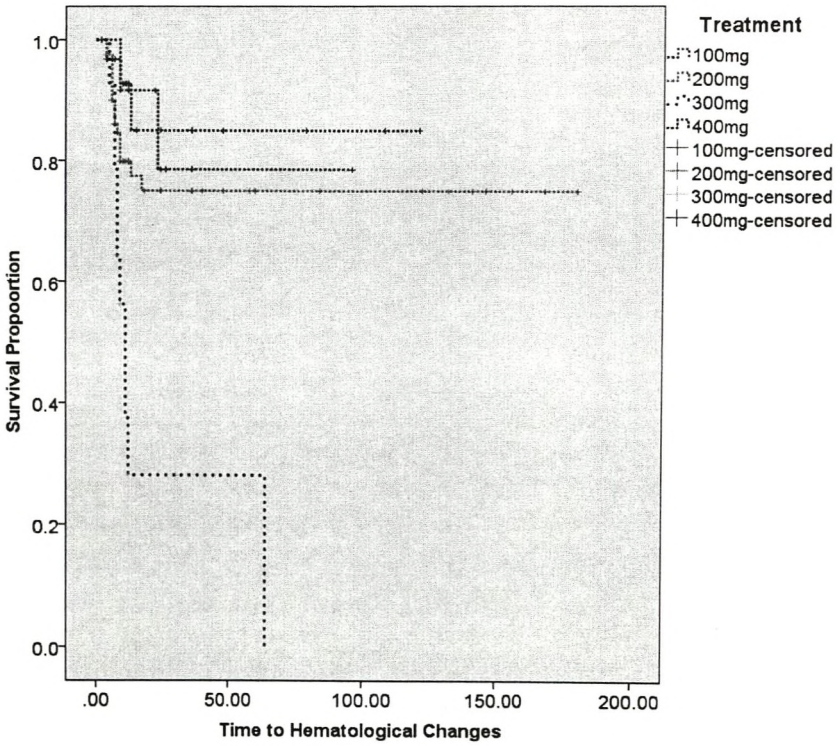


Figure 2. Kaplan-Meier survival curves for the comparison of time to hematological change with different starting doses carbamazepine.

Table 3. Distribution of patients in the total sample who had hepatic and hematological adverse reactions

		Hematological reactions		Total for hepatic reactions
		No	Yes	
Hepatic reactions	No	74 (52.1%)	9 (6.3%)	
	Yes	36 (25.4%)	23 (16.2%)	59 (41.5%)
Total for hematological reactions			32 (22.5%)	

The percentages of the total sample is given within brackets

Discussion

A joint task force of the American Academy of Neurology and the European Federation of Neurological Societies has recommended the use of carbamazepine as the drug of choice for trigeminal neuralgia owing to stronger evidence. They recommend oxcarbazepine as the next alternative because of its high tolerability[2]. Metabolism of carbamazepine occurs as its own mechanism (auto induction) which appears to be completed in the 1st week of starting therapy. Dose-dependent auto-induction of carbamazepine metabolism is the main cause in the relationship between carbamazepine doses, plasma concentration of carbamazepine and concentration of its intermediary metabolite, carbamazepine-10,11-epoxide[16]. The main cause in developing hepatic toxicity in patients receiving antiepileptic treatment is the hypersensitivity reaction or idiosyncrasy[9]. In the present study we document the effects of carbamazepine dosage on the liver and bone marrow as reflected by changes in the levels of liver enzymes and abnormal hematological parameters in a considerably larger sample of TN patients treated in a single institution in Sri Lanka. It reveals that the patients treated with a starting dose of 100mg of carbamazepine took a significantly longer time compared to higher doses to exhibit liver changes. The occurrence of hematological changes was less frequent.

On average, the patients with a 200mg of starting dose of carbamazepine showed liver changes within half the time taken by the patients whose starting dose was 100mg. In contrast, Hussein et al (2013)[10] didn't find a statistically significant influence of the daily dose of carbamazepine on changes in liver enzymes. High dose of carbamazepine alone induced early liver changes, but it was not statistically significant because the number of patients followed up was not enough and further studies would be needed[10]. At the initiation, enzyme inducing property of antiepileptic drugs can cause the liver enzymes to be elevated which can be seen in the first 6 months of the administration of antiepileptic drug[10]. The current study as well as the study of Hussein et al had included only patients with follow up period of 6 months or more. Liver function tests could have been normalized even though the therapy was continued, which may be due to adaptability of the liver. The late development of liver changes observed in this study contradicts the observations made in some studies[9] and highlights the necessity of long term monitoring of the liver function.

The occurrence of liver changes was frequent compared to the occurrence of hematological changes. Several authors have reported hematological abnormalities with antiepileptic drugs. Isojiirvi et al in 1997[11] observed a reduction in mean white cell count, hemoglobin

level and red blood cell count and increase in the size of a red blood cell with carbamazepine and ox-carbamazepine treatment. Hematological changes in this study were mainly seen among the patients receiving either 200mg or 300mg of carbamazepine. Although any change from the reference levels was taken as a positive change, none of the changes seen in the study were significant enough to change the drugs. This again supports the previous observation that hematological effects are infrequent[12] although rare, serious effects like apalstic anemia may occur. After analyzing almost 30,000 patients treated with anti-epileptic drugs, Blackburn and others[12] reported that the chance of getting a serious hematological abnormality is around 3.1/ 100,000 prescriptions or 7.2/ 10,000 treated patients.

The present study reveals that TN was more common in females than in males, with a female- to-male ratio of 1.18:1. Similar findings were reported in a study done by Rothman and Monson (1973)[17] as well. Similar to the findings in the present study, several other studies also found a rise in the incidence rates with increasing age[5,18]. Janetta (1980)[18] suggests that the aging process, specifically with vascular deterioration, causes mechanical, morphological and pathophysiologic effects on the control of trigeminal and other cranial nerves as the possible reason for this pattern. Katusic and colleagues in 1990[5] identified a marginal elevation in the risk of getting TN in patients with hypertension which supports the above theory. The right side of the face was affected to a greater frequency than the left. Several other studies have also reported of a right side prevalence[5] although a plausible theory behind this observation is not yet available.

This study revealed that the age, gender, presence of other medical problems, or consumption of alcohol, side of the face affected etc., had no influence or association on the hepatic and hematological reaction to the treatment for TN. But higher rates of occurrence of serious hepatic impairments with antiepileptic treatment have been reported in elderly patients in previous studies[19].

The present study sample had a relatively less number of patients whose starting dose was either 300mg or 400mg of carbamazepine. Therefore, a reanalysis of data with a larger sample of patients that will have considerably higher number of patients treated with higher starting doses is recommended for more conclusive evidence. Broadening the sample to include patients treated with other antiepileptic drugs especially with a multicenter setup can be expected to provide a complete understanding. Further, this study highlights the importance of monitoring liver function in patients with antiepileptic drug treatment for TN for longer terms as changes are observed after a considerable period. It also demonstrates the rarity of significant hematological reactions to treatment with carbamazepine.

Conclusion

Abnormal liver changes were seen in a significant number of TN patients who were treated with carbamazepine. The occurrence of hematological changes was less frequent. The results suggest that testing the liver function regularly throughout the treatment period is justified. As the average duration to develop adverse reactions with100mg of carbamazepine was 137.1 months, long term monitoring of liver function is mandatory. Only a small fraction of patients on carbamazepine develop abnormalities of bone marrow that could be detected in hematological investigations. Hence frequent full blood count monitoring is not essential during long term use of these drugs. Full blood count once every six months would be adequate.

References

1. Bennetto L, Patel NK and Fuller G. Trigeminal neuralgia and its management. BMJ 2007;334;201-205.

2. Cruccu G, Gronseth G, J. Alksne J, C. Argoff C, M. Brainin M, K. Burchiel K,. NurmikkoT and Zakrzewska JM. American Academy of Neurology Society; European Federation of Neurological Society. AAN-EFNS guidelines on trigeminal neuralgia management. European Journal of Neurology 2008, 15: 1013-1028.

3. Mc Quay H, Carroll D, Jadad AR, Wiffen P,and Moore A. Anticonvulsant drugs for management of pain: a systematic review. BMJ 1995;311:1047-52.

4. Zhang J, Yang M, Zhou M, He L, Chen N, Zakrzewska JM. Non-antiepileptic drugs for trigeminal neuralgia. Cochrane Database Syst Rev. 2013 Dec 3;12:1-40.

5. Katusic S, Beard CM, Bergstralh E, and Kurland LT. Incidence and Clinical Features of Trigeminal Neuralgia, Rochester, Minnesota, 1945-1984. Ann Neurol 1990;27:89-95.

6. Sindrup SH, Jensen TS. Pharmaco-therapy of trigeminal neuralgia. Clin J Pain 2002; 18: 22-7.

7. Taylor JC, Brauer S, Espir ML. Long term treatment of trigeminal neuralgia with carbamazepine. Postgrad Med J 1981; 5: 16-8.

8. Ariyawardana A, Pallegama R, Sitheeque M, Ranasinghe A. Use of single-and multi-drug regimens in the management of classic (idiopathic) trigeminal neuralgia: an 11-year experience at a single Sri Lankan institution. J Investig Clin Dent. 2012 May;3(2):98-102

9. Ahmed SN and Siddiqi ZA. Antiepileptic drugs and liver disease. Seizure (2006) 15, 156-164.

10. Hussein RRS., Soliman RH, Ahmed M. Abdelhaleem Ali AM, Tawfeik MH, Abdelrahim MEA. Effect of antiepileptic drugs on liver enzymes. Beni-Suef University Journal of Basic and Applied Sciences 2 (2013):14-19.

11. Isojiirvi JI, Pakarinen AJ and Myllyla VV. Basic haematological parameters, serum gamma-glutamyl-transferase activity, and erythrocyte folate and serum vitamin B12 levels during carbamazepine and oxcarbazepine therapy. Seizure 1997: 6:207-211.

12. Blackburn SC, Oliart AD, Garcia Rodriguez LA, and Perez Gutthann S. Antiepileptics and Blood Dyscrasias: A Cohort Study. Pharmacotherapy 1998; 18(6): 1277-128.

13. Obermann M. Treatment options in trigeminal neuralgia. Ther Adv Neurol Disord (2010) 3(2) 107-115.

14. Headache Classification Committee of the International Headache Society (IHS) The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia33(9) 629-808.

15. World Medical Association Declaration of Helsinki Ethical Principles for Medical Research Involving Human Subjects. Bulletin of the World Health Organization, 2001,79 (4); 373-4.

16. Kudriakova TB, Sirota LA, Rozova GI & Gorkov VA. Autoinduction and steady-state pharmacokinetics of carbamazepine and its major metabolites. Br. J. clin. Pharmac. (1992), 33, 611-615.

17. Rothman KJ, Monson RR. Epidemiology of trigeminal neuralgia. J Chronic Dis 1973; 26: 2-12.

18. Janetta PJ. Neurovascular compression in cranial nerve and systemic disease. Ann Surg 1980; 192: 518-25.

19. Dreifuss FE andLanger DH. Hepatic Considerations in the Use of Antiepileptic Drugs. Epilepsia, 1987: 28: S23-S29.

Research Article II

Sri Lanka Dental Journal 2015; 45(01) 14-26

Prevalence and socio-behavioural correlates of early childhood caries in preschoolers from the Kegalle district

Vajira Nanayakkara and Lilani Ekanayake

Abstract

Objective: to determine the prevalence and socio-behavioural correlates of early childhood caries (ECC) and severe early childhood caries (S-ECC) in preschoolers residing in the Kegalle district.

Materials and methods: a descriptive cross-sectional study was conducted among 784 children aged between 48 to 72 months attending preschools in the Kegalle district. Data were collected by means of a self-administered, semi-structured questionnaire to mothers/care givers and an oral examination of the children.

Results: The prevalence of ECC was 71.9% and 38.8% had S-ECC. The presence of both ECC and S-ECC were associated with history of dental visits, prolonged breast feeding, brushing once a day and presence of plaque on teeth. In addition the use of non-fluoride toothpaste and low family income were associated with ECC while S-ECC was inversely associated with the level of education of the father.

Conclusions: The prevalence of ECC and S-ECC was high among this group of preschoolers and several socio-behavioural factors were associated with both conditions.

Key words: early childhood caries, prevalence, risk indicators, severe early childhood caries.

Introduction

Early childhood caries (ECC) is defined as the presence of one or more decayed (non cavitated or cavitated lesions), missing due to caries or filled tooth surfaces in any primary tooth of a child under the age of 6 years. The term severe ECC (S-ECC) is used to refer to children with atypical, progressive, acute or rampant patterns of dental caries[1]. ECC is a public health problem worldwide. Analyses of data from the US National Health and Nutrition Examination surveys indicate that caries in the permanent teeth of children have decreased but the prevalence of caries in 2-5-year-olds has increased from 24% in 1988-1994 to 28% in 1999-2004[2]. In contrast, a study conducted among 3-5-year-old Indian children has reported that the prevalence of ECC is as high as 63%[3]. In both developed and developing countries, ECC rates are particularly high among the disadvantaged groups[2,4]. Moreover ECC has a negative impact on the quality of life of children and their parents[5].

ECC is a multi-factorial disease and evidence indicates that risk factors are population specific. Socio-demographic factors such as low socio-economic status, low parental education, dietary and feeding habits such as consumption of sweets and carbonated drinks, in between meal snacking, on demand breast and bottle feeding, prolonged breast feeding, oral health behaviours such as delay in starting oral hygiene practices,

Prof. Lilani Ekanayake (Correspondence) Faculty of Dental Sciences, University of Peradeniya, Peradeniya. E-mail: lilanie@pdn.ac.lk

Dr. Vajira Nanayakkara Office of the Provincial Director of Health Services, Colombo.

not using fluoridated toothpaste as well as microbial composition of plaque have been found to be associated with ECC in different populations[3,4,6-9].

Findings from two recent studies indicate that the prevalence of ECC ranges from 30-69% in Sri Lanka[10,11]. However there is a paucity of information related to the risk indicators associated with ECC. Wellapulli and Amarasena[10] in their study had assessed only the influence of family structure on caries experience of 3-5-year-olds. Kumarihamy *et al*[11] found that long term use of medications, intake of sugar with milk and sweet consumption were associated with early childhood caries in 1-2 year olds in Sri Lanka but the authors have failed to control for confounding in the analysis. As previous studies conducted in Sri Lanka have certain limitations, it was felt that there is a need for a further study to determine the factors associated with ECC in Sri Lankan children. Moreover the prevalence of S-ECC is yet to be determined in Sri Lankan children. Therefore the present study was carried out with the aim of determining the prevalence and socio-behavioural correlates of ECC and S-ECC in 48-<72-month-old preschoolers residing in the Kegalle district.

Materials and methods

The data for the present paper were obtained from a broader study that was carried out to assess the association between diet, nutritional status and oral health among preschool children in the Kegalle district. Only the methodology related to the present paper is described here.

Ethical clearance for the study was obtained from the Ethical Review Committee of the Faculty of Medicine, University of Colombo. Permission to conduct the study was also obtained from the Regional Director of Health Services and the Zonal Director of Education of the Kegalle district. Written informed consent was obtained from parents/caregivers of children. Children who were physically and mentally challenged were excluded.

This study was conducted among 48-<72month-old children attending preschools in the Kegalle district. The formula for estimating a population proportion with absolute precision was used to calculate the sample size. Using the prevalence of dental caries (65%) in 5-year-olds reported in the third National Oral Health survey[12] at 95% level of confidence and accepting a sampling error of 5%, the sample size required was 350. As it was decided to use the cluster sampling method to select the sample, it was necessary to make allowance for the design effect which was considered as 2. Therefore the minimum sample size needed to satisfy these requirements was 700. However the sample was increased by 15% to compensate for non-responses and therefore totaled 805. Eight hundred and thirty eight children were included in the main study which exceeded the calculated sample size (805) for this part of the study. Therefore that sample of 838 was adopted for the present study as well.

When the cluster sampling technique is used, it is necessary to include at least 30 clusters to obtain valid data and a large number of small clusters are preferable to a small number of large clusters[13]. A cluster consisted of 10 children and therefore 84 clusters had to be included in the study ($838/10 \approx 84$). A preschool was considered as a cluster. In Sri Lanka, a district is divided into Divisional Secretariat (DS) divisions for administrative purposes. Based on the number of preschool children in each DS division, the 84 clusters were allocated to the 10 DS divisions of the district using the probability proportionate to size technique. Use of the probability proportionate to size technique and selecting equal number of children from each cluster, gives each child in the population the same probability of being selected for the sample[14]. The following method was adopted to select the clusters from a chosen DS division; the main street of the DS division was located and the preschool

closest was selected as the first cluster from that division. Then the preschool closest to the first was selected as the second cluster and this method was followed until the required number of clusters was selected from each DS division. Ten children aged between 48-<72 months were selected randomly from each preschool.

Data collection took place at the school premises. A 25-item semi-structured, self-administered questionnaire to mothers/caregivers was used to obtain information on socio-demographic data, feeding habits, oral health and dietary behaviours. The questionnaire was developed and validated by the first author for the purpose of the present study. The questionnaire was administered in the presence of the first author, which enabled the respondents to clarify any doubts. Information from those respondents who were unable to read or write was obtained through an interview conducted by the preschool teacher. The questionnaire was pretested among 20 mothers of preschool children from the same district and based on the findings certain questions were rephrased for better clarity. These 20 mothers were excluded from the main study.

Dental caries and plaque levels were assessed by the first author under natural day light while the child was seated on a chair. The examiner was trained and calibrated against a paedodontist for recording dental caries and plaque levels. Caries was recorded according to the criteria recommended by Drury *et al.*[1] Both non-cavitated and cavitated lesions were considered as caries but they were not recorded separately. Intra–examiner agreement associated with caries detection was determined by re-examining 10% of the children examined on a particular day and the Kappa statistic associated with caries diagnosis was 0.89. Plaque was determined by running the periodontal probe along the cervical margins and adjacent areas of teeth. Presence of plaque was recorded on 4 surfaces of 6 teeth; distal, mesial, buccal/labial and lingual/palatal surfaces of 55, 61, 63, 75, 81 and 83 and the percentage of surfaces with plaque were determined.

STATA 12.0 software (Stata Corp., College Station, TX, USA) was used for data analysis. ECC was defined as the presence of one or more decayed (cavitated and non-cavitated lesions), missing due to caries or filled tooth surface in a child (dmfs≥1). The case definition for S-ECC was a dmfs score ≥ 5 at 48-59 months of age or a dmfs score of ≥6 at 60-<72 months of age (Drury et al, 1999). Chi Square test was used to determine the associations between categorical variables. Having excluded for the presence of multi-collinearity, those variables that were associated with ECC and S-ECC at P<0.06 level in the bivariate analyses were used in two Poisson regression models with robust variance to determine the independent associations between the dependent and explanatory variables.

Results

Eight hundred and thirty eight pairs of children and mother/caregivers were recruited for the study. All 838 mothers/caregivers responded to the questionnaire but only 784 children participated in the oral examination which gave an overall response rate of 94%. The mean age of the sample was 57.3 ±6.2 months. The overall prevalence of ECC in the sample was 71.9% and 38.8% had S-ECC. The mean dmfs in the group with ECC and S-ECC were 2.32 ± 1.1 and 14.56 ±8.6 respectively.

Table 1 shows the bivariate associations between ECC and various socio-demographic and behavioural variables. Presence of ECC was higher in the 60-<72-month olds, those from low income families and from families with 3 or more children, consumed sugary snacks more than twice a day, who were still being breast fed at the time of participating in the study, brushed their teeth once a day, used non-fluoride toothpaste, with a history of dental visits and had plaque on teeth. S-ECC was higher in boys

Table 01. Associations between ECC and selected socio-demographic and behavioural variables

Variable	with ECC		no ECC		
	n	%	n	%	P. value
Age group (months)					
48- <60 (466)	316	67.8	150	32.2	.002
60-<72 (318)	248	78.0	70	22.0	
Gender					
Boys (383)	282	73.6	101	26.4	0.30
Girls (401)	282	70.3	119	29.7	
Ethnicity					
Sinhala (704)	500	71.0	204	29.0	0.15
Tamil (18)	16	88.9	2	11.1	
Moor (62)	48	77.4	14	22.6	
Father's education (years)					
0-5 (52)	40	76.9	12	23.1	0.09
6-12 (656)	476	72.6	180	27.4	
>12 (72)	44	61.1	28	38.9	
Mother's education (years)					
0-5 (35)	29	82.9	6	17.1	0.10
6-12 (636)	460	72.3	176	27.7	
>12 (109)	71	65.1	38	34.9	
Employment status of mother					
Not employed (680)	495	72.8	185	27.2	0.23
Employed (100)	67	67.0	33	33.0	
Monthly family income					
Rupees ≤13,000 (371)	285	76.8	86	23.2	0.008
Rupees >13,000 (383)	247	68.0	116	32.0	
No of children in family					
Up to 2 (617)	430	69.7	187	30.3	0.009
3 or more (165)	132	80.0	33	20.0	
Frequency of sugary snack consumption					
Up to twice/day (616)	428	69.5	188	30.5	0.008
>twice/day (161)	129	80.1	32	19.9	

Carbonated drink consumption					
Never (107)	79	73.8	28	26.2	0.36
2-3 times/week (559)	389	69.6	170	30.4	
Once or more/day (61)	47	77.0	14	23.0	
Breast fed at the time of participating in study					
No (651)	453	69.6	198	30.4	0.003
Yes (126)	104	82.5	22	17.5	
Time of breast feeding (126)					
Only at night when going to bed (88)	72	81.8	16	18.2	
On demand (34)	28	82.4	6	17.6	0.97
Tooth brushing frequency/day					
Once (87)	77	88.5	10	11.5	<0.001
More than once (695)	485	69.8	210	30.2	
Type of toothpaste used					
Fluoride (729)	517	70.9	212	29.1	0.03
Non fluoride (43)	37	86.0	6	14.0	
Brushing teeth done by					
Child (190)	144	75.8	46	24.2	0.11
Parent/caregiver (592)	418	70.6	174	29.4	
History of dental visits					
No (493)	325	65.9	168	34.1	<0.001
Yes (285)	233	81.8	52	18.2	
Dental plaque on teeth					
Absent (238)	129	54.2	109	45.8	
Present (546)	435	79.7	111	20.3	<0.001
Total (784)	564	71.9	220	28.1	

ECC group includes children with dmfs ≥1

Table 02. Associations between S-ECC and selected socio-demographic and behavioural variables

Variable	with S-ECC		No caries		
	n	%	n	%	P. value
Age group(months)					
48- <60 (314)	164	52.2	150	47.8	0.001
60-<72 (210)	140	66.7	70	33.3	
Gender					
Boys (271)	170	62.7	101	37.3	0.02
Girls (253)	134	53.0	119	47.0	
Ethnicity					
Sinhala (468)	264	56.4	204	43.6	0.06
Tamil (12)	10	83.3	2	16.7	
Moor (44)	30	68.2	14	31.8	
Father's education (years)					
0-5 (41)	29	70.7	12	29.3	<0.001
6-12 (443)	263	59.4	180	40.6	
>12 (38)	10	26.3	28	73.7	
Mother's education (years)					
0-5 (20)	14	70.0	6	30.0	0.06
6-12 (432)	256	59.3	176	40.7	
>12 (70)	32	45.7	38	54.3	
Employment status of mother					
Not employed (449)	264	58.8	185	41.2	0.52
Employed (73)	40	54.8	33	45.2	
Monthly family income					
Rupees ≤13,000 (249)	163	65.5	86	34.5	0.003
Rupees >13,000 (243)	127	52.3	116	47.7	
No of children in family					
Up to 2 (414)	227	54.8	187	45.2	0.006
3 or more (108)	75	69.4	33	30.6	
Frequency of sugary snack consumption					
Up to twice/day (399)	211	52.9	188	47.1	0.001
>twice/day (122)	90	73.8	32	26.2	

Carbonated drink consumption					
Never (63)	35	55.6	28	44.4	0.19
2-3 times / week (384)	214	55.7	170	44.3	
Once or more / day (46)	32	69.6	14	30.4	
Breast fed at the time of participating in study					
No (442)	244	55.2	198	44.8	0.005
Yes (79)	57	72.2	22	27.8	
Time of breast feeding (79)					
Only at night when going to bed (51)	35	68.6	16	31.6	
Any time (24)	18	75.0	6	25.0	057
Tooth brushing frequency/day					
Once (59)	49	83.1	10	16.9	<0.001
More than once (463)	253	54.6	210	45.4	
Type of toothpaste used					
Fluoride (498)	286	57.4	212	42.6	0.43
Non fluoride (18)	12	66.7	6	33.3	
Brushing teeth done by					
Child (128)	82	64.1	46	35.9	0.11
Parent / caregiver (396)	222	56.1	174	43.9	
History of dental visits					
No (315)	147	46.7	168	53.3	<0.001
Yes (207)	155	74.9	52	25.1	
Dental plaque on teeth					
Absent (138)	29	21.0	109	79.0	
Present (386)	275	71.2	111	28.8	<0.001

S-ECC group includes 48-59-month-olds with ≥ 5dmfs and 60-<72-month-olds with ≥6 dmfs

Table 03. Poisson regression model for presence of ECC

Variable	Adjusted PR	95% CI	P value
Age group(months)			
48 - <60	1.00		
60 - 71	1.07	0.98-1.67	0.16
Monthly family income (Rs)			
≤13,000	1.00		
>13,000	0.91	0.83-0.99	0.04
No of children in family			
Up to 2	1.00		
3 or more	1.10	0.99-1.21	0.055
Frequency of sugary snack consumption			
Up to twice/day	1.00		
>twice/day	1.08	0.98-1.18	0.12
Breast fed at the time of participating in study			
No	1.00		
Yes	1.16	1.05-1.28	0.003
Tooth brushing frequency/day			
Once	1.00		
More than once	0.84	0.76-0.92	< 0.001
Type of toothpaste used			
Fluoride	1.00		
Non fluoride	1.32	1.14-1.52	<0.001
History of dental visits			
No	1.00		
Yes	1.24	1.13-1.35	<0.001
Dental plaque on teeth			
Absent	1.00		
Present	1.33	1.18-1.51	<0.001

PR=prevalence ratio. ECC dichotomized as 0= if absent; 1= if present

Table 04. Poisson regression model for presence of S-ECC

Variable	Adjusted PR	95% CI	P value
Age group(months)			
48 - <60	1.00		
60 - <72	1.05	0.91-1.21	0.50
Gender			
Boys	1.00		
Girls	1.00	0.87-1.15	0.99
Father's education (years)			
0-5	1.00		
6-12	0.99	0.79-1.26	0.97
>12	0.49	0.31-0.85	0.01
Mother's education (years)			
0-5	1.00		
6-12	0.93	0.61-1.44	0.76
>12	0.93	0.57-1.52	0.78
Monthly family income			
Rupees ≤13,000	1.00		
Rupees >13,000	0.93	0.81-1.07	0.33
No of children in family			
Up to 2	1.00		
3 or more	1.11	0.96-1.31	0.15
Frequency of sugary snack consumption			
Up to twice/day	1.00		
>twice/day	1.13	0.97-1.30	0.10
Breast fed at time of participating in study			
No	1.00		
Yes	1.19	1.01-1.40	0.03
Tooth brushing frequency/day			
Once	1.00		
More than once	0.77	0.66-0.89	0.01
History of dental visits			
No	1.00		
Yes	1.53	1.33-1.77	<0.001
Dental plaque on teeth			
Absent	1.00		
Present	2.69	1.93-3.74	<0.001

PR=prevalence ratio S-ECC dichotomized as 0= no caries; 1= with S-ECC

and the 60-<72-month olds, children whose father had a low level of education, those from low income families and from families with 3 or more children, consumed sugary snacks more than twice a day, who were still being breast fed at the time of participating in the study, brushed their teeth only once a day, with a history of dental visits and had plaque on the teeth (Table 2).

The results of the Poisson regression analysis for the presence of ECC are shown in Table 3. The prevalence of ECC was significantly higher in those with a history of dental visits, had plaque on teeth, who were still being breast fed at the time of participating in the study, used non-fluoride toothpaste but significantly lower in those who brushed their teeth more than once a day and were from high income families. Table 4 gives the Poisson regression analysis for the presence of S-ECC. S-ECC was significantly higher in those with a history of dental visits, had plaque on teeth, who were still being breast fed at the time of participating in the study but significantly lower in children whose fathers had >12 years of education and brushed their teeth more than once a day.

Discussion

The burden of ECC in 4-5-year-old Asian children has been assessed in many studies. However the findings of the present study cannot be directly compared with most of them due to an important methodological issue; i.e. the difference in the case definition used for diagnosing and reporting ECC. Conforming to the recommendations of Drury *et al.*[1], both cavitated and noncavitated carious lesions on tooth surfaces were considered as caries in the present study whereas some studies have recorded caries at the level of the dentine only[4,11,15].

The overall prevalence of ECC in the sample and the prevalence of ECC in children aged 48-59 and 60-<72 months are almost similar to those of a Chinese study which had used the same criteria as the present study for recording ECC[6]. On the other hand Jin *et al.*[16] found that the prevalence of ECC in 4-5-year-old South Korean children was as high as 91% while the prevalence of S-ECC was 70% when both non-cavitated and cavitated lesions were included. According to Tsai *et al.*[9] 75% of 4-year-olds and 89% of 5-year-olds from Taiwan were affected by ECC and the researchers have reported that the prevalence rates may have been much higher had they considered the non-cavitated lesions as well. However compared to the above studies, relatively lower rates of ECC have been reported for 4-(37%) and 5-year-olds (49%) from Singapore[17].

Several socio-behavioural factors were associated with both ECC and S-ECC. Children who gave a history of a dental visit had higher levels of ECC and S-ECC than those who did not. Similar findings have been reported from other developing countries as well[7,18]. Sri Lankans of all age groups mainly visit dental clinics when they have a problm[12]. This suggests that the perceived need is an important factor that influences dental visits among Sri Lankans. Therefore it is very likely that dental visits in children with ECC and S-ECC may have been initiated due to the presence of symptoms associated with dental caries. In fact Vargas *et al.*[19] found that 2-5-year-olds without a perceived need were more likely to have never visited a dentist than those with a perceived need. In contrast children who had a regular dental visits had lower ECC rates than those who did not visit a dentist regularly[20].

Many studies have assessed the impact of breast feeding on ECC and the duration of breast feeding has been considered as the explanatory variable in most studies. In contrast the explanatory variable in the present study was whether or not the child was breast fed at the time of participating in the study. The use of this variable may have minimized constraints arising from failure to recall past events. Accordingly

16% of the children were still being breast fed at the time of participating in the study and the mean age of this group was 55.5 months. These children had more ECC and S-ECC than those who were not breast fed at the time of participation in the study. However the time of breast feeding when categorized into "only at night when going to bed" and "on demand" was neither associated with ECC nor S-ECC. Evidence related to the effect of prolonged breast feeding on early childhood caries is inconsistent. Nunes *et al.*[15] compared ECC levels in Brazilian children who were breast fed for <12 months with those who were still breast fed at the time of their study and found that prolonged breast feeding was not a risk factor for ECC. The mean duration of breast feeding in that study was 34 months. On the other hand, a study on 41-50-month-old Japanese children has shown that breast feeding for 18 months or longer was positively associated with ECC[21]. Sankeshwari *et al.*[3] found that prolonged breast feeding was a significant determinant of ECC in Indian children with ECC present in 75% of those who were breast fed beyond the age of 24 months. But it is important to note that the duration of breast feeding in those studies was much lower than the duration of breast feeding in the breast fed group of the present study. Valaitis *et al.*[22] following a systematic review on the relationship between breast feeding and early childhood caries, have concluded that valid conclusions could not be drawn due to methodological inconsistencies and that evidence does not indicate a consistent and strong association between breast feeding and the development of ECC.

Sugar is the main aetiological agent for dental caries. Several studies have shown that consumption of sugary snacks more than twice a day is associated with both ECC and S-ECC[7,20,27]. Although consumption of sugary snacks was significantly was associated with both ECC and S-ECC in the bivariate analyses, it lost its significance in the Poisson regression analyses indicating possible confounding effects of other variables.

Conforming to previous studies[18,23], the presence of plaque on teeth was associated with both ECC and S-ECC. Also tooth brushing frequency was inversely associated with ECC as well as S-ECC and supports the findings of other workers[4,24]. As expected children who used fluoride toothpaste had lower levels of ECC compared to those who used non-fluoride toothpaste. Similar results have been reported elsewhere[4]. These findings highlight the importance of plaque control and good oral hygiene practices in preventing ECC.

The prevalence of ECC and S-ECC was inversely associated with family income and father's level of education and was in agreement with those of previous studies[25,26]. However the number of children in the family was not associated with the presence of ECC and is in contrast to findings of others[6,11,25].

The two previous studies on ECC reported in the literature were confined to very small urban units of Sri Lanka[10,11] whereas the present study included a representative sample selected from both urban and rural areas of a district which is the largest administrative unit of the country. Also this is the first study to report the prevalence and risk indicators of S-ECC. There are a few limitations in this study. To obtain data, this study relied on a self-administered questionnaire. It is possible that information bias such as not responding accurately to sensitive questions like income and social desirability bias such as selectively under- or over reporting certain oral health and dietary behaviours may have had an effect on the accuracy of the data collected. Also the cross-sectional nature of the study limits causal interpretation of the results.

In conclusion, it is evident from the results that the prevalence of ECC and S-ECC was high in this group of preschoolers. The socio-behavioural risk indicators identified would be useful for educating parents/care givers

about ECC as well as for planning preventive programmes to control early childhood caries in the Kegalle district.

References

1. Drury DF, Horowitz AM, Ismail AI, Maertens MP, Rozier RG, Selwitz RH. Diagnosing and reporting early childhood caries for research purposes. J Public Health Dent 1999; 59: 92-197.

2. Tinanoff N, Reisine S. Update of early childhood caries since the Surgeon General's Report. Acad Pediatr 2009; 9: 96-403.

3. Sankeshwari RM, Ankola AV, Tangade PS, Hebbal MI. Feeding habits and oral hygiene practices as determinants of early childhood caries in 3- to 5-year-old children of Belgaum City, India. Oral Health Prev Dent 2012; 10: 283-290.

4. Prakash P, Subrananiam P, Durgesh BH, Konde S. Prevalence of early childhood caries and associated risk factors in preschool children of urban Bangalore, India: a cross-sectional study. Eur J Dent 2012; 6: 141-152.

5. Martins-Jùnior PA, Vieira-Andrade RG, Corrêa-Faria P, Oliveira-Ferreira F, Marques LS, Ramos-Jorge ML. Impact of early childhood caries on the oral health-related quality of life of preschool children and their parents. Caries Res 2013; 47: 211-218.

6. Li Y, Zhang Y, Yang R, Zhang Q, Zou J, Kang D. Associations of social and behavioural factors with early childhood caries in Xiamen city in China. Int J Paediatr Dent 2011; 6: 121-128.

7. Naidu R, Nunn J, Kelly A. Socio-behavioural factors and early childhood caries: a cross-sectional study of preschool children in central Trinidad. BMC Oral Health 2013; 13: 30.

8. Parisotto T, Steiner-Oliveira C, Duque C, Peres RCR, Rodrigues LKA, Nobre-dos-Santos M. Relationship among microbiological composition and presence of plaque, sugar exposure, social factors and different stages of early childhood caries. J Dent 2010; 55: 365-373.

9. Tsai AI, Chen CY, Hsiang CL, Hsu KH. Risk indicators for early childhood caries in Taiwan. Community Dent Oral Epidemiol 2006; 34: 437-445.

10. Wellappuli N, Amarasena N. Influence of family structure on dental caries experience of preschool children in Sri Lanka. Caries Res 2012; 46: 208-212.

11. Kumarihamy SLM, Subasinghe LD, Jayasekara P, Kularatne SM, Palipana PD. The prevalence of early childhood caries in 1-2 yr olds in a semi-urban area of Sri Lanka. BMC Research Notes 2011; 4:336.

12. Ministry of Health Care and Nutrition. National Oral Health Survey 2002/2003. Colombo: Ministry of Health Care and Nutrition, 2008.

13. Kirkwood, BR, Sterne JAC. Essential Medical Statistics. 2nd ed. London: Blackwell Publishing Company; 2004.

14. Benett S, Woods T, Liyanage WM. Simplified general method for cluster-sample survey in developing countries. Health Stat Q 1991; 44: 98-106.

15. Nunes AMM, Alves CMC, Araujo FB, Ortitrz TML, Silva AAM, Ribeiro CCC.

Association between prolonged breast-feeding and early childhood caries: a hierarchical approach. Community Dent Oral Epidemiol 2012; 40: 542-549.

16. Jin BH, Ma DS, Moon HS, Paik DI, Hahn SH, Horowitz AM. Early childhood caries prevalence and risk factors in Seoul, Korea. J Public Health Dent 2003; 63: 183-188.

17. Gao XL, Hsu CY, Loh T, Koh D, Hwamg HB, Xu Y. Dental caries prevalence and distribution among preschoolers in Singapore. Community Dent Health 2009; 26: 12-17.

18. Kiwanuka SN, Astrøm AN, Trovik TA. Dental caries experience and its relationship to social and behavioural factors among 3-5-year-old children in Uganda. Int J Paediatr Dent 2004; 14: 336-346.

19. Vargas CM, Ronzio CR. Relationship between children's dental needs and dental care utilization: United States, 1988-1994. Am J Public Health 2002; 92: 1816-1821.

20. Han D, Kim D, Kim M, Kim J, Jung-Choi K, Bae K. Regular dental checkup and snack-soda drink consumption of preschool children are associated with early childhood caries in Korean caregiver/preschool children dyads. Community Dent Oral Epidemiol 2014; 42: 70-78.

21. Tanaka K, Miyake Y, Sasaki S, Hirota Y. Infant feeding practices and risk of dental caries in Japan: the Osaka Maternal And Child Health Study. Pediatr Dent 2013; 35: 267-271.

22. Valaitis R, Hesch R, Passarelli C, Sheehan D, Sinton J. A systematic review of the relationship between breast feeding and early childhood caries. Can J Public Health 2000; 91: 411-417.

23. Declerck D, Leroy R, Martens L, Lesaffre E, et al. Factors associated with prevalence and severity of caries experience in preschool children. Community Dent Oral Epidemiol 2008; 36: 168-178.

24. Hsieh H-J, Huang S-T, Tsai C-C, Hsiao S-Y. Toothbrushing habits and risk indicators of severe early childhood caries among aboriginal Taiwanese. Asia Pac J Public Health 2012; DOI: 10.1177/1010539511430721.

25. Congiu G, Campus G, Sale S, Spano G, Cagetti MG, Lugliè PF. Early childhood caries and associated determinants: a cross-sectional study on Italian preschool children. J Public Health Dent 2013, doi: 10.1111/jphd.12038.

26. Psoter WJ, Pendrys DG, Morse DE, Zhang H, Mayne ST. Associations of ethnicity/ race and socio-economic status with early childhood caries patterns. J Public Health Dent 2006; 66: 23-29.

27. Hashim R, Williams S, Thomson WM. Severe early childhood caries and behavioural risk indicators among young children in Ajman, United Arab Emirates. Eur Arch Paediatr Dent 2011; 12: 205-210.

Sri Lanka Dental Journal 2015; 45(01) 27-29

Sri Lankan Declaration on Oral Cancer, Smokeless Tobacco and Arecanut "Towards a society free of oral cancer"

Declaration

In conformity with the scientific evidence forwarded during the first ever International Symposium on Oral Cancer, Smokeless Tobacco and Areca nut, it has been recognized that the urgent need of the hour is to put forward a declaration to combat health hazards of smokeless tobacco and areca nut. This symposium was held in Kandy on 8th and 9th August 2014, with the collaboration of the Centre for Research in Oral Cancer, Faculty of Dental Sciences, University of Peradeniya, National Cancer Control Programme, Ministry of Health and the World Health Organization. The symposium brought together key international organisations and leading world experts in the field of smokeless tobacco/ areca nut control and oral cancer. The objective was to provide strategic leadership to coordinate and synergise policy, strategy and programs within a common stakeholder framework to implement a model of smokeless tobacco / areca nut control / oral health care based on health promotion, disease prevention and disease management. Leading specialists and professionals of the fields related to this multi disciplinary topic from various countries addressed the delegates of this scientific symposium.

Oral cancer is a major killer in Sri Lanka. The country has one of the highest incidences of oral cancer in the world and it is predominantly due to smokeless tobacco and areca nut use.

Introduction and background

- Oral cancer is the commonest cancer among Sri Lankan males. 50% die of the disease within 5 years of diagnosis.
- Major causal factors include tobacco in all forms and betel quid with or without tobacco. Areca nut is the primary ingredient in betel quid and is a proven to be carcinogen in humans.
- Use of Smokeless Tobacco (ST) and areca nut is common in Sri Lanka.
- Commercially prepared areca nut products are a serious emerging health hazard.
- These agents are known to be psychoactive, and have been proven to be addictive. They are the major causes of oral disease, other systemic conditions, disability and death. Studies report up to a five-fold increase in incidence of oral cancer among tobacco and areca nut chewers compared to non-users.
- Worldwide, this issue has not received the attention it deserves from policy makers, researchers and health professionals.
- There is also little data on factors associated with the initiation and maintenance of such products in different populations and groups within populations.

Aim of the Declaration

The aim of this declaration is to identify strategic priorities and to set out a roadmap for action aimed at reducing tobacco and areca nut usage and thereby reduce the incidence of oral cancer

in Sri Lanka.

For health care professionals / institutions

1. Health care professionals must promote oral health among their patients.

2. Health care professionals should conduct a visual examination of the oral cavity whenever feasible.

3. ST/areca nut consumers are considered a potentially high risk group for malignant disorders/oral cancer according to the risk factor model and thus are in need of high risk screening

4. Counselling against ST and areca nut use should be a part of a routine practice of every health care professional.

5. All health institutions (clinics, hospitals, medical colleges, dental colleges, nursing colleges, paramedical colleges etc.) must be declared as free of ST and commercially prepared areca products.

6. Sri Lanka Medical and Dental associations should conduct continuing medical educations (CME's)) on ST, areca nut and oral cancer control regularly.

7. ST/ areca nut cessation skills should be incorporated in all undergraduate curricula.

For the mass media

8. Mass media should inform the health hazards of using all forms of tobacco including chewing tobacco and areca nut.

For Sri Lanka Customs

9. Be alert and vigilant on illegal imports of smokeless tobacco consignments and areca nut from neighbouring countries.

For Religious leaders

10. Advise the general public on the harmful effects of ST and areca nut products and refrain from using such products.

11. Move towards achieving religious premises free of ST and areca nut.

For the Government of Sri Lanka

12. Classify areca nut as a hazardous substance in the same category as use of tobacco products.

13. Institute a comprehensive areca nut control program.

14. Conduct periodic mass awareness programmes for ST and areca nut products.

15. Prohibit import, manufacture and sale of packed tobacco and areca nut products (chewable).

16. Strengthen National Authority on Tobacco & Alcohol Act towards effective control of the use and sale of smokeless tobacco and areca nut products.

17. Raise the taxes on ST and areca nut products.

Signatories

Dr Palitha Mahipala
Director General of Health Services

Dr (Mrs) Lakshmi Somatunga
Deputy Director General (Medical Services 1)

Dr JayasundaraBandara
Deputy Director General (Dental services)

Prof W M Tilakaratne
Director Centre for Research in oral cancer

Sri Lankan Declaration on Oral Cancer, Smokeless Tobacco and Arecanut
"Towards a society free of oral cancer"

Prof SamanWarnakulasuriya
Director, WHO collaborating center for oral cancer and pre-cancer/UK

Prof Newell W Johnson
Emeritus Professor of Dental Research, Griffith Health Institute, Australia

Mr D H Hapuarachchi
Commissioner General of Excise

Prof E A P D Amaratunga
Head, Department of oral pathology, Faculty of Dental Sciences

Prof Narada Warnasuriya
Chairman, Tobacco & Alcohol committee, SLMA

Dr Sajeeva Ranaweera
Tobacco & Alcohol committee, SLMA

Prof Lilani Ekanayake
Head, Division of Community Dentistry, University of Peradeniya

Dr Pankaj Chaturvedi
Head/Voice of tobacco victims/India, cancer surgeon/Tata Memorial Hospital

Mr PubuduSumanasekara
Director/Alcohol &Drug Information Centre

Dr Prakash Gupta
Head of the HealisSekhsaria Institute of Public Health, India.

Dr Prasad Abeysinghe
Consultant Oncologist/National Cancer Institute/ Maharagama)

Dr Sajeeva Ranaweera
Tobacco and Alcohol subcommittee, SLMA

Dr Palitha Abeykoon
Chairman/National Authority for Tobacco and Alcohol

Dr Neelamani Parangama
Director, National Cancer Control Programme

Dr E.A. Fernando
Deputy Director, National Cancer Control Programme

Dr A M Attygala
Senior Lecturer and Consultant OMF Surgeon, Faculty of Dental Sciences

Dr Vajira Jayasinghe
Senior Lecturer and Consultant in Restorative Dentistry, Faculty of Dental Sciences

Dr Ruwan Jayasinghe
Senior Lecturer and Consultant OMF Surgeon, Faculty of Dental Sciences

Dr Mani l Fonseka
Senior Lecturer and Consultant in Restorative Dentistry, Faculty of Dental Sciences

Dr Hemantha Amarasinghe
Head, Unit for Tobacco and oral cancer control, National Cancer Control Programme

Case Report

Sri Lanka Dental Journal 2015; 45(01) 30-35

Metastatic carcinoma of the gingiva mimicking a denture granuloma: A case report

C. Prasanna, S.P.I. Silva and R.L.P.R. Liyanage

Abstract

Introduction - Metastatic tumours of oral cavity are rare, about 1% of all oral cancers account for metastasis of primary tumours elsewhere in the body, mainly originating from the breast, followed by lung, kidney, thyroid gland, intestine, prostate gland, stomach, testis and bladder. It may occur in the oral soft tissue or hard tissues.

Aim: The aim of this report is to describe an unusual presentation of a metastatic tumour in the oral cavity mimicking a benign lesion.

Clinical history and investigation: A 73 year old denture wearing male patient presented to our clinic with the complaint of recurrent growth in the anterior maxillary region for a period of 3 months duration. It was clinically diagnosed as denture granuloma and an excision biopsy was performed and sent for histopathological evaluation. It was histopathologicaly diagnosed as a metastaticad enocarcinoma. Further investigations were performed including immunohistochemistry in order to detect the site of primary origin.

Diagnosis: Primary tumor was identified in the lungs.

Discussion and conclusion: The diagnosis of metastatic tumour to oral cavity is a challenge to a clinician. Clinical examination including systemic evaluation is essential where site of the primary tumour is unknown. The use of radiological investigations with immuno-histochemistry can facilitate the identification of the primary lesion. Metastasis from a distant site should be considered among differential diagnosis of oral lesions and the importance of sending all tissue sample for histopathological evaluation must be emphasized.

Keywords: metastatic carcinoma, jaw metastasis, metastatic tumour

Introduction

Metastatic tumours (MT) of the oral and maxillofacial region are uncommon, comprising nearly 1% of all oral malignancies[1,2,3]. Such metastasis can occur in the jaw bones or oral soft tissues elsewhere in the body. The common primary sites of origin are the breast and lung followed by the kidney, prostate, thyroid, intestine, stomach, testis and bladder.

These tumours have high clinical significance as their appearance may be the first indication of an undiscovered malignancy at the distant primary site or the first evidence of dissemination of a known tumour from its primary site. MT in the oral cavity is very often missed because it

Dr. C. Prasanna (Correspondence) — Oral and MaxillofacialSurgery Unit-B, Dental Institute, Colombo 07.

Dr. S.P.I. Silva — Oral and MaxillofacialSurgery Unit-B, Dental Institute, Colombo 07.

Dr. R.L.P.R. Liyanage — Dental Institute, Colombo 07.

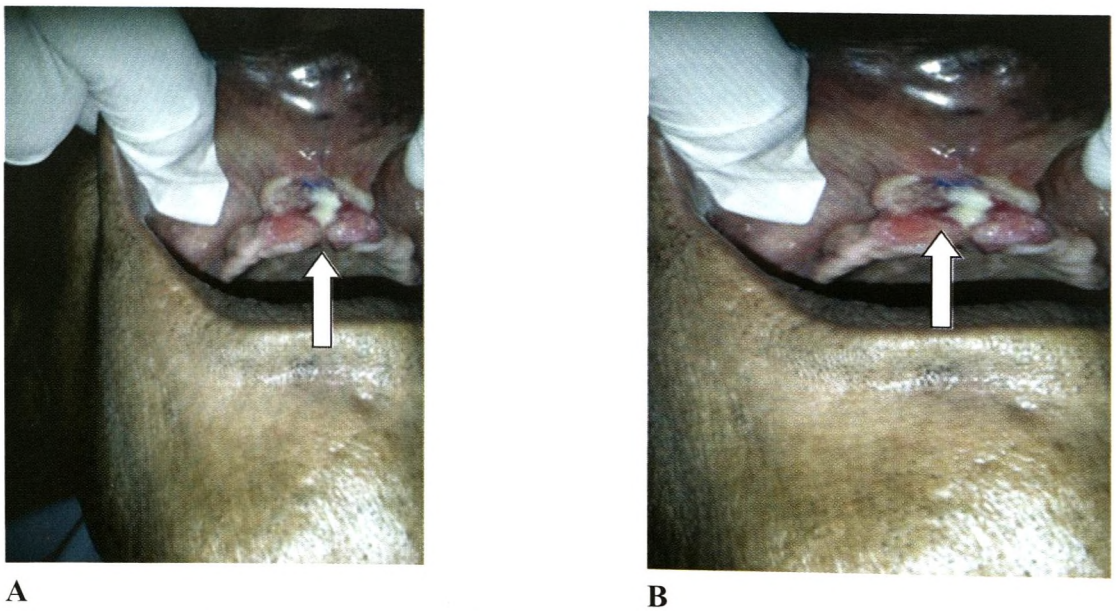
may simulate a benign process. The diagnosis of a MT in oral region is challenging both to the clinician and the pathologist. The clinician must recognize the possibility that the lesion may represent a metastasis and the pathologist can assit to determine the site of tumour origin[4].

Case Report

A 73 years old edentulous male was referred to oral and maxillofacial clinic with a complaint of an ulcerative growth in the upper anterior labial sulcus and alveolar ridge for three months duration. The lesion had been excised previously about three months ago but it had not been sent for histopathological examination and recurred. Past medical history revealed that he had been a known diabetic for the past ten years and was on anti-hyperglycemic drugs and anti-platelet drugs.

On clinical examination patient was ill looking, dyspneic with generalized body wasting. Intra oral examination revealed an ulcerated exophytic growth in relation to the anterior maxillary alveolar mucosa region, which was 1.5 x 1.0 cm in size. On palpation it was firm in consistency and non tender with the tendency to bleed. No cervical lymphadenopathy was present. As the lesion was hyperplastic in nature and associated with upper denture flanges, the provisional diagnosis of denturegranuloma was made. Routine investigations were performed; full blood count, fasting blood sugar, bleeding time, clotting time and the results were unremarkable. Upper standard occlusal and periapical radiograph of 1/1 region were performed that showed slight evidence of bone resorption in relation to the lesion.

An excision biopsy was performed under local anesthesia (fig-01). Initial histopathological report indicated the lesion to be a metastatic adenocarcinoma with the possibility of adenocarcinoma (NOS) of salivary origin. The Mucicarmine stain showed focal positivity. Further, immmunohistochemical staining for CK7,CK20 and TTF1 were done. CK7 showed strong positivity, while TTF1 was focally positive in the tumour cells. CK20 was negative. Based on the above immunohistochemical and histopathological findings, a primary adenocarcinoma arising from lung was suggested as the most possible site of origin,



A **B**

Figure 1. A,B intra oral view following excisional biopsy

Subsequent radiological investigations of the chest, pelvis and mandible were performed. Radiological changes was observed on the right side of the lungs(fig-02). Computerized tomography (CT) of the chest was performed and a primary lesion on right side lung chest field was reported (fig-03). The diagnosis was confirmed with a lung biopsy.

Oncological referral was done for further management. Considering the wide spread nature of the lesion, patient's age and medical condition, the patient was managed palliatively but died 6 months after diagnosis.

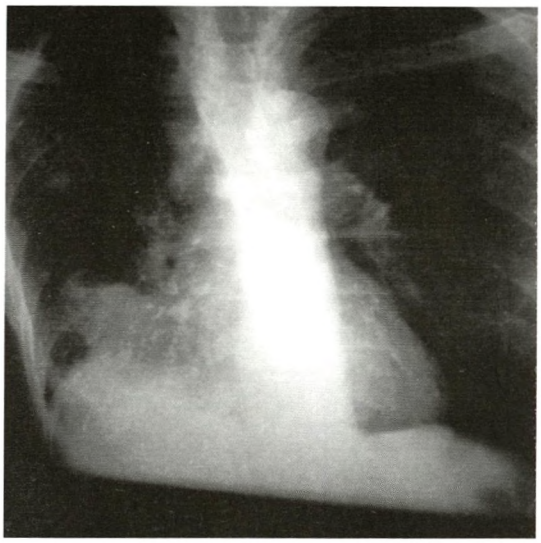


Figure 2. Patient's chest radiograph shows radio opaque mass on the right side of lung field

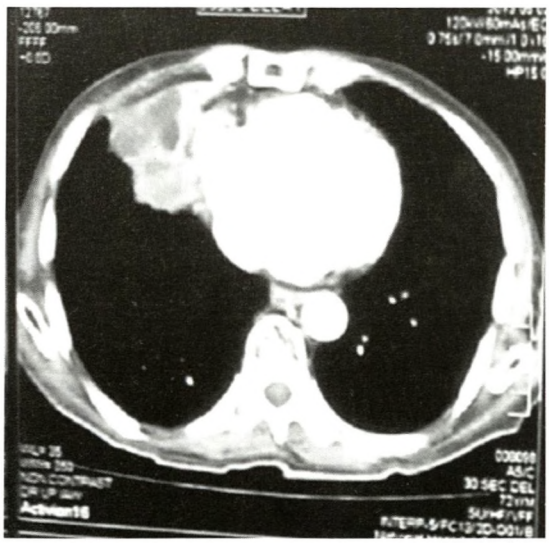


Figure 3. CT Scan of lung shows primary tumour on the right side of lung

Discussion

Oral metastasis is considered as a late complication and is frequently associated with multiple organ metastasis. Almost any malignancy from any site of the body is capable of metastasis in the oral cavity. Most MT of oro-facial region are seen in patients aged between 40-70 years, mean age of 45years, 80% of patient in their 7th decade of life[5]. As reported, metastasis is common in the jaw bones compare to oral soft tissue. Posterior part of the mandible is the most common site (80%). Maxillary involvement is uncommon[6]. The site preference exists despite the fact that mandible and maxilla share common blood supply, the maxillary artery[4].

The most frequent primary sites of oral metastasis are lung, breast and bone. The nature of primary tumour and the site of metastasis within the oral cavity differ between the sexes[7]. The breast is the most common site for tumour metastasis of the jaw bone in the female, where the lung is the most common site in males. The most common site for oral soft tissue metastasis is the gingivae (50%) followed by tongue (25%) of all cases.

Clinically in the oral soft tissue the lesion usually appears as a nodular mass and often resembles hyperplastic or reactive growth, such as pyogenic granuloma. Occasionally the lesion appears as a surface ulceration. The adjacent teeth may become loosened by an underling destruction of the alveolar bone. Paraesthesia of the lower lip and chin was found as ominous sign of MT in the mandible as thus constituted significant deep invasion of tumour in to the bone and involvement of inferior alveolar or mental nerve[6]. In our case patient also presented with recurrent hyperplastic or reactive growth on the gingival with surface ulceration. There wasn't any paresthesia observed.

The mechanism by which tumours can spread to oral cavity is poorly understood. Primary malignancy from immediate adjacent tissue might spread by the lymphatic route. However such a mechanism cannot explain metastasis of the tumour from the lower part of the body, which are almost certainly blood borne and should be filtered out by the lungs. One possible explanation for blood borne to head and neck especially in the absence of pulmonary metastasis is Batson's plexus[8], a valveless vertebral venous plexus that might allow retrograde spread of tumour cells by passing filtration through the lungs[9].

The presence of teeth may play an important role in the preference of metastases to the gingivae. Once malignant cells reach the oral cavity, the rich vascular network of inflamed gingival tissue may serve as fertile soil for further growth[9].

Radiological changes in the jaw bone depend mainly on mineral loss in the area of tumour. Although most of the MTs to jaw bone are osteolytic, some of the lesions particularly prostatic metastasis is mostly osteosclerotic in nature and may stimulate new bone formation in the metastatic site, resulting in radiopaque or mixed radiolucent and radiopaque lesions. In our case slight evidence of bone rosorption was observed in relation to the lesion.

The histological appearance of MT is highly variable, depending on the tumour type and degree of differentiation. Well differentiated MT provides a strong indication of the primary site; like renal cell or thyroid carcinoma. .However MT is more frequently poorly differentiated and in these cases it is difficult to determine the primary site. In such cases immunohistochemistry can aid in determining the site of origin. .Immunohistochemical stain for cytokeratin will verify the presence of epithelial differentiation confirming the tumour most likely to be a carcinoma, and tissue specific markers may be used to further characterize the tumour. However the final diagnosis will depend on a complete medical history, physical examination, and appropriate investigations[10].

In our case initial histopathology report reviled as metastatic adenocarcinoma. With the judgment of initial findings, a supplementary immuno- histochemical study was performed to identify the primary site. Following immunonhisto chemical staining were used cytokeratin-7(CK7), cytokeratin-20 (CK20) and thyroid transcription factor-1(TTF1) were done. CK7 showed strong positive, (useful to distinguish metastatic adenocarcinoma of pulmonary origin) while TTF1 was focally positive and CK20 was negative in tumour cells. CK7 expression was significantly more frequent in adenocarcinomas of the pulmonary and of breast origin than gastrointestinal origin, CK20 expression was significantly more prevalent in adenocarcinoma that originate in the GI track than that of pulmonary origin and most of adenocarcinoma express TTF1[11]. Depending on these immuno- histochemical findings, the most possible primary site was identified as the lung. Diagnosis was confirmed with further clinical investigations.

Identification of the primary site and determining the extent of metastatic involvement is the first step in the management of MT. Once oral metastasis is identified in a patient, it is revealed that the status disease is widely disseminated and prognosis is poor, by definition distant metastasis automatically places the tumour in stage-IV disease. When the systemic investigations confirm that the jaw represents only the site of metastasis or where primary tumour was treated successfully, the metastatic lesion can be treated aggressively with adequate surgical excision or chemo or ratio therapy, if the patient's medical condition permits. This may improve the prognosis[10,12]. Palliative treatment should be aimed for elimination of pain and preservation of function. It may also involve surgical excision of metastatic deposit, radiation therapy, chemotherapy and/or chemo radiation[13].

Our patient was managed palliativelly by considering patient age, wide spread of lesion and patients medical condition. Overall the prognosis of MT of the jaw is poor with the mean survival rate of of 6 to 7 months.

Conclusion

Metastasis of distant site malignancy should be considered as a differential diagnosis of oral lesions. The use of clinical information, radiological investigations and histopathology with immunohistochemistry can facilitate identification of the primary site of origin. Importance of sending all excised tissue for histopathological evaluation has to be emphasized.

Acknowledgements

Staff at the OMF unit B, dental institute for their support, patient who give me his cooperation, without him this study wouldn't have been possible and also I extend my gratitude to Prof. W.M Tilakaratne Professor of oral pathology and the staff of Department of Oral Pathology FDS University of Peradeniya for providing histopathological and immuno-histochemical investigations.

References

1. Sriwardena B.SM.S ,Tilakaratne W.M, Jayasooriya P.R, PalithaRatnayaka. Metastatic tumours of the oral and maxillofacial region; A retrospective study of 18 cases. Merit research journal of biochemistry and bioinformatics vol.1(1) May 2013 pp. 001-004.

2. Van der Wall RI, Buter J, et al . Oral metastasis. A report of 24 cases; Br J Oral and maxillofacial surg.2003; 41(1):3-6.

3. Moutasim K A, Thomas GJ: Soft tissue and bone tumour. Saman Warnakulasuriya, Tilakaratne W.M . Oral medicine and pathology A guide to diagnosis and management. Jaypee brothers medical publishers (p) LTD 1st ed.2014; 179-204.

4. Bodner l, Sion-vardy N, et al. Metastatic tumour of the jaw. A report of eight new cases: Med oral patol oral bucal, 2006; E 132-5.

5 Singh H , Kumar P, et al .possible pathogenic mechanisms and over view of metastatic tumour to the oral cavity. the internet journal of oncology. 2010 vol-8 number -1.

6. Naville, Damm, Allen, Biuquot. Oral and maxillofacial pathology 2nd ed: Elsevier; 2004: 533-52.

7. Hirshberg A, Leibovich P, Buchner A; metastatic tumour of the jaw bones: analysis of 390 cases. J Oral patholMED . 1994; 23:337-41.

8. Batson OV,; The function vertebral vein and their role in the spread of metastasis. Ann sur1940;112 ;138-149.

9. Neville, Damm, Allen, Bouquot. Oral and maxil!ofacial pathology 2nd ed:Elsevier; 2004: 489-90.

10. Anthonypogrel M, Briah L. eal. clin icalpathology:odontogenicandnonod ontogenictumoure of jaw. Peterward booth, stphan A .maxillofacial surgery chuchilllivingstoneelsevier; vol(1), 2nd : 527-28.

11. Yue- chisu SU, Yu-chang HSU, Chee-yin chei. Role of TTF1, CK20 and CK7 immunohistochemistry for diagnosis of primary and secondary lung adenocarcinoma. Kaohsing J med sci. 2006; 22:14-9

12. Kumar G S, Manjunatha. Metastatic tumour to the jaw and oral cavity. Journal of maxillofacial pathology. 2013 jan-apr; 1791):71-75.

13. Hirshberg A, Leibovich P, Bucher A. Metastatic tumour to jaw bones; Analysis of 390 cases, J of oral pathol Med 23(80);1994:337-341.

Instructions to Authors

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

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 of general practice etc. They should be 1500 words in length. References should be 20 or less.

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

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.

Short Reports

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

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.

Letters to Editor

Subjects unlimited, but may include short critique of published papers in the SLDA.

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.

Proceedings on 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 (2I2x297 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 naming 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:

1. **Objective**: An introductory sentence indicating the objective and purpose of the study.
2. **Materials 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.

Materials 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 arc considered figures and should be numbered in the order of appearance in Arabic numerals. Each figure should have a brief and specific 1egend, 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 Ieukoplakia 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 J. Geneva: World Health Organisation, 1992; 550--564

DENTA®

ADULT TOOTHBRUSH

SLS 276

SLDA ACCEPTED SRI LANKA DENTAL ASSOCIATION

VITA CAD/CAM MATERIALS – Perfect solutions for single tooth restorations

VITABLOCS®
Mark II

Feldspar ceramic/monochromatic
1-step material

Benefits
- proven a million times over simple , fast and reliable processing
- excellent integration of the shade

Recommended indications*
For smaller, esthetic reconstructions:
- Inlay
- Onlay
- Partial crown

Example

VITABLOCS®
TriLuxe / TriLuxe forte / RealLife

Feldspar ceramic/multichromatic
1-step material

Benefits
- proven 2nd generation VITABLOCS
- shade gradient ensures high esthetics
- ideal for highly individual dental esthetics

Recommended indications*
For highly esthetic reconstructions in the visible region:
- Anterior crown
- Veneer

Example

VITA ENAMIC®

Hybrid ceramic/monochromatic
1-step material

Benefits
- hybrid ceramic that absorbs occlusal forces
- ideal for minimally invasive restorations
- enables high - precision resultsand eficient CAD/CAM fabrication

Recommended indications*
For minimally invasive reconstructions and posterior crowns:
- Reconstructions in cases with limited space
- Reconstructions of minor defects
- Non-prep veneer / tabletop
- Posterior tooth/molar crown
- Implant-supported crown

Example

VITA SUPRINITY®

Glass ceramic, precrystallized/monochromatic
2-step material

Benefits
- high reliability thanks to enormous strength
- versatile use thanks to wide indication range
- optimized edge stabilty compared to lithium disilicate

Recommended indications*
Various (esthetic) options and wide indication range are provided:
- Posterior crown
- Anterior crown
- Implant-supported crown
- Veneer

Example

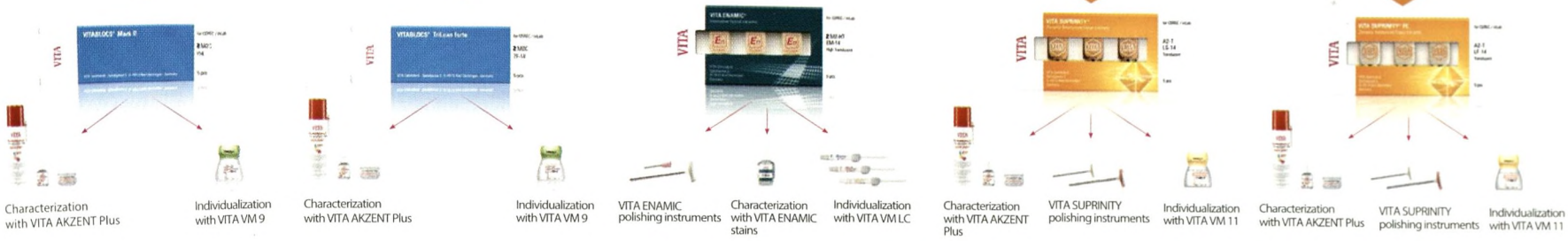
VITA SUPRINITY® FC**

Glass ceramic, fully crystallized/monochromatic
1-step material

Benefits
- simple processing
- fast solutions
- excellent dental esthetics

Recommended indications*
For smaller, esthetic reconstructions
- Inlay
- Onlay
- Partial crown

Example



*) Note: While these are recommede indications; the entire range of single tooth restorations can normally be febricated. For details, please refer to the respective working instruction of the products. **) VITA SUPRINITY® FC is scheduled to be available during the year 2016. This is preliminary product information. Final product details will be published during the market launch.

VITA CAD/CAM MATERIALS – for perfect solutions.
Proven a million times over.

Densu Lab Available CAD / CAM Solutions.

Densu Ceramic Dental Laboratory

No. 08, Norris Canal Road, Colombo - 10, Sri Lanka.
Tel: +94 112 680336/ 337 Fax : +94 112 680338
Hot Lines : +94 777 678116 / 722 238121
E-mail : densudentallab@sltnet.lk Web : www.densu.lk

DENSU Laboratory Service

Signal

SUPERIOR CAVITY PROTECTION*

NON-FLUORIDE TOOTHPASTE

Vs

WITH CAVITY PROTECTION SHIELD

Rs. 40/- upwards

Unilever

*Based on maximum permissible level of fluoride as anticavity agent in chalk based toothpaste formulation.
Creative visualization of protection against cavities offered by fluoridated toothpaste Vs non fluoridated toothpaste without actives.

0112 629 629
Call us