Malaysian Dental Journal (2005) 26(2) 67-68

MALAYSIAN DENTAL JOURNAL

Editor: **Associate Professor Dr. Ngeow Wei Cheong**
BDS (Mal), FFDRCSIre (Oral Surgery), FDSRCS (Eng), AM (Mal)
Department of Oral & Maxillofacial Surgery,
Faculty of Dentistry, University of Malaya,
50603 Kuala Lumpur, Malaysia.
E-mail: ngeowy@yahoo.com

Malaysian Dental Association Council 2005-2006

President:	Dr Shubon Sinha Roy
President-elect:	Dr Wong Foot Meow
Immediate Past President:	Datuk Dr Teo Choo Kum
Hon. General Secretary:	Datuk Dr N. Lakshmanan
Asst. Hon. Gen. Secretary:	Dr R. Vijendran
Hon. Financial Secretary:	Dr How Kim Chuan
Asst. Hon. Finan Secretary:	Dr Lee Chiong Weng
Hon. Publication Secretary:	Dr Thomas Abraham
Chairman, Northern Zone:	Dr Koh Chou Huat
Secretary, Northern Zone:	Dr Neoh Gim Bok
Chairman, Southern Zone:	Dr Steven Phun Tzy Chieh
Secretary, Southern Zone:	Dr Leong Chee San
Elected Council Member:	Dr Sivanesan Sivalingam
Elected Council Member:	Dr Haja Badrudeen
Nominated Council Member:	Dr Nedunchelian
Nominated Council Member:	Dr Adibah Bt Nadar @ Nadarrudin
Nominated Council Member:	Dr Lee Soon Boon

The Publisher

The Malaysian Dental Association is the official Publication of the Malaysian Dental Association. Please address all correspondence to:

Editor,
Malaysian Dental Journal
Malaysian Dental Association
54-2, (2nd Floor), Medan Setia 2,
Plaza Damansara, Bukit Damansara,
50490 Kuala Lumpur
Tel: 603-20951532, 20947606, Fax: 603-20944670
Website address: http://mda.org.my
E-mail: mda@po.jaring.my
mda@streamyx.com

Malaysian Dental Journal (2005) 26(2) 69

MALAYSIAN DENTAL JOURNAL

Aim And Scope

The Malaysian Dental Journal covers all aspects of work in Dentistry and supporting aspects of Medicine. Interaction with other disciplines is encouraged. The contents of the journal will include invited editorials, original scientific articles, case reports, technical innovations. A section on back to the basics which will contain articles covering basic sciences, book reviews, product review from time to time, letter to the editors and calendar of events. The mission is to promote and elevate the quality of patient care and to promote the advancement of practice, education and scientific research in Malaysia.

Publication

The Malaysian Dental Journal is an official publication of the Malaysian Dental Association and is published half yearly (KDN PP4069/12/98)

Subscription

Members are reminded that if their subscription are out of date, then unfortunately the journal cannot be supplied. Send notice of change of address to the publishers and allow at least 6 - 8 weeks for the new address to take effect. Kindly use the change of address form provided and include both old and new address. Subscription rate: Ringgit Malaysia 60/- for each issue, postage included. Payment in the form of Crossed Cheques, Bank drafts / Postal orders, payable to Malaysian Dental Association. For further information please contact :

The Publication Secretary
Malaysian Dental Association
54-2, (2nd Floor), Medan Setia 2, Plaza Damansara, Bukit Damansara,
50490 Kuala Lumpur

Back issues

Back issues of the journal can be obtained by putting in a written request and by paying the appropriate fee. Kindly send Ringgit Malaysia 50/- for each issue, postage included. Payment in the form of Crossed Cheques, Bank drafts / Postal orders, payable to Malaysian Dental Association. For further information please contact:

The Publication Secretary
Malaysian Dental Association
54-2, (2nd Floor), Medan Setia 2, Plaza Damansara, Bukit Damansara,
50490 Kuala Lumpur

Copyright

Membership and change of address

All matters relating to the membership of the Malaysian Dental Association including application for new membership and notification for change of address to and queries regarding subscription to the Association should be sent to Hon General Secretary, Malaysian Dental Association, 54-2 (2nd Floor) Medan Setia 2, Plaza Damansara, Bukit Damansara, 50490 Kuala Lumpur. Tel: 603-20951532, 20951495, 20947606, Fax: 603-20944670, Website Address: http://www.mda.org.my. Email: mda@po.jaring.my or mda@streamyx.com

Disclaimer

Statements and opinions expressed in the articles and communications herein are those of the author(s) and not necessarily those of the editor(s), publishers or the Malaysian Dental Association. The editor(s), publisher and the Malaysian Dental Association disclaim any responsibility or liability for such material and do not guarantee, warrant or endorse any product or service advertised in this publication nor do they guarantee any claim made by the manufacturer of such product or service.

Malaysian Dental Journal (2005) 26(2) 70

MALAYSIAN DENTAL JOURNAL

CONTENT

MDJ cover page : Cross-section of C - Shaped canal on a Mandibular Second Molar.

The cover page is courtesy of Dr. Chai Wen Lin.

Malaysian Dental Journal (2005) 26(2) 71

MALAYSIAN DENTAL JOURNAL

Editorial
Indexing the Malaysian Dental Journal

One issue of the Malaysian Dental Journal is completed and now this second issue is finally on the way for all to read, as have been promised. What's new? Well, after having taken over the task of editorial work from Dr. M. Thomas Abraham, I find that the hardest thing is to get articles and to get willing referees.

We have 3 public and 2 private dental schools in this country; and the number is growing with more public dental schools on the way. However, the number of dental journal published in this country remains the same; one by the Malaysian Dental Association and another by the Faculty of Dentistry of the University of Malaya. Still, we find shortage of contributions. One reason cited by the academicians for not contributing to the Malaysian Dental Journal is that it is not listed in the INDEX MEDICUS/MEDLINE. This term may be new to some, but for researchers and academicians, having an article published and be included in the INDEX MEDICUS/MEDLINE is very important. INDEX MEDICUS/MEDLINE enables a piece of scientific work to be known to all researchers around the world.

At the moment, not many dentists and researchers in other part of the world know that the Malaysian Dental Journal exists due to its non-listing in INDEX MEDICUS/MEDLINE. Hence, they would not be sending any contributions to this journal. Moreover, even if they know of the Malaysian Dental Journal, they may still not be sending their work to us as it would not be listed in the INDEX MEDICUS/MEDLINE. That practically means no one else would gain access to their work except for the members of the Malaysian Dental Association (as this journal is provided free of charge to all members).

So, what are we going to do? Of course, we are not going to sit back and let the world goes by leaving us behind. We want to catch up. My predecessor, Dr. M. Thomas Abraham has initiated the work to get the Malaysian Dental Journal into the listing. A lot of effort has been done, and till now, we are still in the process of getting it done. With that we hope, more researchers will be able to read our work and achievements; and contribute theirs to share with us. With that too, we hope the Malaysian Dental Journal can gradually rank as high as other prominent dental journals in the world.

Thank you.

Associate Professor Dr. Ngeow Wei Cheong
Editor
Malaysian Dental Journal

Malaysian Dental Journal (2005) 26(2) 72-82

MALAYSIAN DENTAL JOURNAL

Expression of Ki-67 and MDM2 at the Tumour Invasive Front in Oral Squamous Cell Carcinoma: Relationship with Clinicopathologic Factors

Lee CN *Department of Oral Pathology, Oral Medicine & Periodontology, Faculty of Dentistry, UM, Kuala Lumpur.*

Zain RB *Department of Oral Pathology, Oral Medicine & Periodontology, Faculty of Dentistry, UM, Kuala Lumpur.*

Abidin NZ *Institute of Biological Sciences, UM, Kuala Lumpur.*

Khoo SP *Department of Oral Pathology, Oral Medicine & Periodontology, Faculty of Dentistry, UM, Kuala Lumpur.*

Kumar SKS *Department of Oral Pathology, Oral Medicine & Periodontology, Faculty of Dentistry, UM.*

ABSTRACT

Background: The tumour invasive front of oral squamous cell carcinoma (OSCC) has been recognized to harbour better prognostic information. The general aim of this study was to evaluate the biological activity using proliferative marker, Ki-67 and an apoptotic marker, MDM2 at the invasive front of oral squamous cell carcinoma. Methods: A total of 31 OSCC cases from the buccal mucosa were evaluated with specific antibodies to Ki-67 and MDM2 using immunohistochemistry (IHC). The sociodemographic and clinicopathological parameters were recorded and analysed against the IHC findings. Results: The expression of Ki-67 and MDM2 were detected in 90.3% (28) and 64.5% (20) of the cases respectively. The present study found significant association between MDM2 expression with gender (P=0.026) and Broder's tumour grading (P=0.041). Significant association was also observed between combined Ki-67/MDM2 expression with both gender (P=0.011) and Broders' tumour grading (P=0.031). The present study found no significant association between Ki-67 and MDM2 at the tumour invasive front. No association was also found between Ki-67 expression as a proliferative marker (using labelling index) and MDM2 as an apoptotic markers. Conclusions: The tumour invasive front may be suitable for evaluating the expression of Ki-67 as their localization is seen at the basal and parabasal areas. The value of evaluating MDM2 at the tumour invasive front is still controversial as this study showed that the localization of MDM2 is at the suprabasal and the upper region. Such MDM2 distribution is in accordance to some reports but contrary to another report in the literature.

Key words:
tumor markers, oral squamous cell carcinoma, tumour invasive front.

INTRODUCTION

Despite tremendous progress achieved in cancer research over the last few decades, the mortality of oral cancer, in particular oral squamous cell carcinoma (OSCC) patients has remained high and it was observed that half of the patients afflicted would die within 2 to 3 years of diagnosis.[1] Patients with squamous cell carcinoma of the upper aerodigestive tract often have diverse clinical courses and show different growth rates even though given the same clinical stage.[2] This is partly because it consisted of heterogenous cell populations with different biologic characteristics.[3] Currently, prognosis is mainly based on the clinical TNM (tumour, node, and metastasis) classification supplemented by the conventional histopathological tumour grading which is neither definitive prognostic indicator nor factors upon which treatment can be based.[4] Thus, there is a great need for new prognostic tools that can aid the clinician in better defining the most appropriate treatment for individual patient.[1]

One of the most promising recent findings in this respect has been the recognition of the significance of structural and functional features of the most advanced parts of carcinoma, namely the tumour invasive front.[5] The tumour cells in this area are known to have the most aggressive behaviour.[6] Various molecular events of importance in tumour spread occur at this tumour-host interface.[5] Therefore, the molecular and morphological characteristics at this area are believed to reflect tumour prognosis better than other parts of the tumour.[1,5]

Invasive front grading (IFG) is a malignancy grading system used to assess the tumour activities at the tumour invasive front and this system has been proven to have high prognostic value.[7,8] The tumour cell characteristics are solely graded within the lowest differentiated parts of the most invasive 3-6 cell layers at the advancing front of tumours.[5] Among four individual morphological parameters that were evaluated in this grading system, the pattern of invasion seems to be the single most important prognostic parameter.[9,10] The pattern of invasion has been shown to be

associated with nodal and distant metastasis[11] as well as survival.[10,11] The general aim of this study is to evaluate the biological activity using proliferative marker, Ki-67 and apoptotic marker, MDM2 at the invasive front of oral squamous cell carcinoma. These findings may throw some light in predicting the behaviour of the tumour. The proliferation cell marker (Ki-67) and the marker associated with apoptosis (MDM2) have shown some potential towards being diagnostic and prognostic indicators of tumour progression.[12,13] However, their expression at the tumour invasive front is still unclear.

Thus, the specific objectives of this study are to describe the immunohistochemical expression of Ki-67 and MDM2 at the tumour invasive front of OSCC and to relate these findings with certain sociodemographic (Age, gender, ethnic group and habits) and clinicopathological (Tumour size, nodal status, TNM clinical staging, Broders' tumour grading and 'pattern of invasion') parameters.

MATERIALS AND METHODS

Tumour Samples and Criteria of Selection

A total of 31 cases of histopathologically diagnosed oral squamous cell carcinoma (OSCC) of the buccal mucosa were selected from the archives of the Department of Oral Pathology, Oral Medicine and Periodontology, Faculty of Dentistry, University of Malaya. Specimens lacking underlying connective tissue or the superficial layers of OSCC and those that did not show continuity at the invasive front layers were excluded. Cases of early invasive OSCC and cases with incomplete clinical records (description of tumour size, nodal status and distant metastasis) were also excluded.

Sociodemographic and Clinicopathologic Characteristics of Samples

The following data were collected from the biopsy report forms and folders of each patient: Age, gender, ethnic group, habits (if stated), TNM grading. Table 1 summarises the sociodemographic and clinicopathological characteristics of the subjects. The distribution of tumours according to tumour size and nodal status is shown in Table 2.

Criteria for Tumour Grading

The histopathological tumour grading namely, modified Broders' grading[14] and pattern of invasion[5] was evaluated by an oral pathologist (RBZ). The results of immunohistochemistry were determined through individual assessment by the first author and the oral pathologist using specific criteria. In the modified Broders' malignancy grading, tumours were graded as grade 1 (well-differentiated), 2 (moderately-differentiated) and 3 (poorly-differentiated). The criteria for scoring of the pattern of invasion were followed using previous report by Bryne et al.[5] which is a modified version of scoring by Anneroth et al.[15] as given below: score 1 (Pushing, well delineated infiltrating borders), score 2 (Infiltrating, solid cords, bands and/or strands), score 3 (Small groups or cords of infiltrating cells, n<15) and score 4 (Marked and widespread cellular dissociation in small groups and/or in single cell, n<15).

Immunohistochemistry (IHC)

IHC staining was done separately for the expression of Ki-67 and MDM2. The immunostaining was carried out using the avidin biotin technique. Heat mediated antigen retrieval was carried out by incubating sections (4 µ thick) in 0.01M citrate buffer at pH 6.0 in a microwave oven heat at 100°C. The primary antibodies used were polyclonal rabbit anti-human Ki-67 antigen (DAKO, code N1574) and monoclonal mouse anti-human MDM2 protein (DAKO, code M7146). Procedural positive controls were from known cases of OSCC that reacted positively to Ki-67 and MDM2. Negative controls were obtained by replacing the primary antibody with TBS.

Evaluation of staining

A qualitative and semi-quantitative evaluation of immunohistochemical staining was done. The immunoreactivity scoring for Ki-67 and MDM2 was evaluated at the tumour invasive fronts which consist of 3-6 cell layers at the advancing front of tumours. Cells were counted as positive when an unequivocal diffuse or localised brown nuclear staining could be identified, regardless of staining intensity. Cytoplasmic staining alone was not considered whereas cells were counted as positive if both nuclear and cytoplasmic staining were visible.

A uniform scoring system for immunopositive cells of Ki-67 and MDM2 was followed: < 20% of positively stained cells = 1+; 20-50% = 2+ and >50%= 3+.[16] All immunostained slides were evaluated by light microscopy with the aid of an image analyzer. Using a 40¥ objective, a field at the invasive front was randomly selected; the numbers of positive cells were counted followed by the counting of the total number of cells. The labelling index (LI) for Ki-67 was calculated by taking an average of the percentage of positively stained cells in the 3 fields at the invasive front for each marker.

Statistical analysis

Categorical data was statistically analyzed by chi-square and Fisher's exact tests. The continuous data were summarized as mean ± SD (standard deviation). Significance was assessed by Mann-Whitney U test and Kruskal-Wallis test. P values of <0.05 were considered to be significant.

Table 1: Clinical and histopathological parameters of 31 subjects studied

Patient No.	Age/ Ethnicity	Gender	TNM Grading	TNM Stage	Grade 1,2,3	Pattern of Invasion	Habits
1	52/I	M	T2N0M0	II	1	1	A
2	74/I	F	T4N1M0	IV	1	3	B
3	65/I	F	T1N0M0	I	1	1	B
4	58/I	F	T2N3M0	IV	1	3	B
5	71/I	F	T2N0M0	II	1	1	B
6	61/I	F	T4N2M0	IV	1	3	B
7	73/I	F	T2N2M0	IV	2	1	B
8	50/I	F	T1N1M0	II	1	1	B
9	72/I	F	T1N0M0	I	1	2	B
10	46/M	F	T2N0M0	II	2	3	-
11	55/I	F	T2N0M0	II	1	1	B
12	73/I	F	T2N1M0	III	1	1	B
13	48/I	F	T2N2M0	IV	1	3	-
14	55/I	F	T4N1M0	IV	2	3	B
15	55/M	F	T4N2M0	IV	1	2	B
16	50/I	F	T1N0M0	I	1	2	B
17	66/I	F	T4N2M0	IV	1	3	B
18	70/C	M	T2N1M0	III	2	1	S
19	70/C	M	T4N1M0	IV	2	1	S
20	59/I	F	T4N2M0	IV	1	3	B
21	45/I	F	T2N1M0	III	2	2	B
22	53/I	F	T2N1M0	III	1	2	B
23	70/I	F	T4N1M1	IV	2	3	B
24	54/C	F	T2N1M0	III	1	3	-
25	66/I	M	T3N2M0	IV	1	1	B,A
26	71/I	M	T2N2M0	IV	2	3	B
27	47/I	M	T4N2M0	IV	2	2	A
28	75/I	F	T1N2M0	IV	1	3	-
29	69/I	M	T3N2M0	IV	2	2	B,S
30	62/I	F	T3N2M0	IV	1	2	-
31	59/I	F	T4N1M0	IV	1	1	-

Ethnicity: I: Indian; M: Malay; C : Chinese
Grade: 1: Well-differentiated; 2: Moderately-differentiated; 3: Poorly-differentiated
Habits: B: Betel quid chewing; S : Smoking; A : Alcohol consumption
- : Not stated

Table 2: Distribution of tumours according to tumour size and nodal status

Tumour size	Nodal status				Total
	N0 n (%)	N1 n (%)	N2 n (%)	N3 n (%)	n (%)
T1 n (%)	3 (9.7)	1 (3.2)	1 (3.2)	0 (0)	5 (16.1)
T2 n (%)	4 (12.9)	5 (16.1)	3 (9.7)	1 (3.2)	13 (41.9)
T3 n (%)	0 (0)	0 (0)	3 (9.7)	0 (0)	3 (9.7)
T4 n (%)	0 (0)	5 (16.1)	5 (16.1)	0 (0)	10 (32.3)
Total n (%)	7 (22.6)	11 (35.5)	12 (38.7)	1 (3.2)	31 (100)

RESULTS

Expression of Ki-67 and MDM2 at the tumour invasive front of OSCC in buccal mucosa

The results of Ki-67 and MDM2 are shown in Table 3. Twenty eight (90.3%) out of 31 specimens showed immunopositivity for Ki-67, with staining scale of 1+ in 8 specimens (25.8%), 2+ in 19 specimens (61.3%) and 3+ in 1 specimen (3.2%). Three specimens (9.7%) did not show any expression of Ki-67. Positive staining for Ki-67 was located mainly in cells in the basal and suprabasal regions (Figure 1a). Diminishing expression was noted towards the upper region or the centre of the tumour islands. The staining was almost entirely confined to the nuclei and consistent throughout the invasive front layer. The intensity of the staining was variable and ranged from faint to dark brown reaction.

There were 20 (64.5%) out of 31 specimens that showed immunopositivity of MDM2 with staining scale of 1+ in 9 specimens (29.0%), 2+ in 9 specimens (29.0%) and 3+ in 2 specimens (6.5%). Eleven specimens (35.5%) did not show any expression of MDM2 (Table 3). The positive staining was mainly in cells at the suprabasal and at the superficial regions (Figure 1b). Immunoreactivity was predominantly a combination of nuclear and cytoplasm in all subjects with some subjects showing intense cytoplasmic staining. In a few cases, staining was observed predominantly in the cytoplasm. The nuclear staining showed accentuation at the nuclear membrane and the nucleoli.

Relationship between the Ki-67 labelling index and the immunohistochemical expression of MDM2 at tumour invasive front of OSCCs in buccal mucosa

There was an increase in Ki-67 LI for the MDM2 immunopositive cases (24.6%) as compared to the MDM2 immunonegative cases (21.5%). However, no significant difference was observed in Ki-67 LI for MDM2 immunopositive and immunonegative cases (Table 4).

Table 3: Immunostaining results of tumour markers Ki-67 and MDM2

Immunohistochemical Staining Scale	Tumour Markers	
	Ki-67	**MDM-2**
1+	25.6%	29%
2+	61.3%	29%
3+	3.2%	6.5%
Total Positive Cases	90.3%	64.5%
Total Negative Cases	9.7%	35.5%

Immunostaining scale of Ki-67 and MDM2:

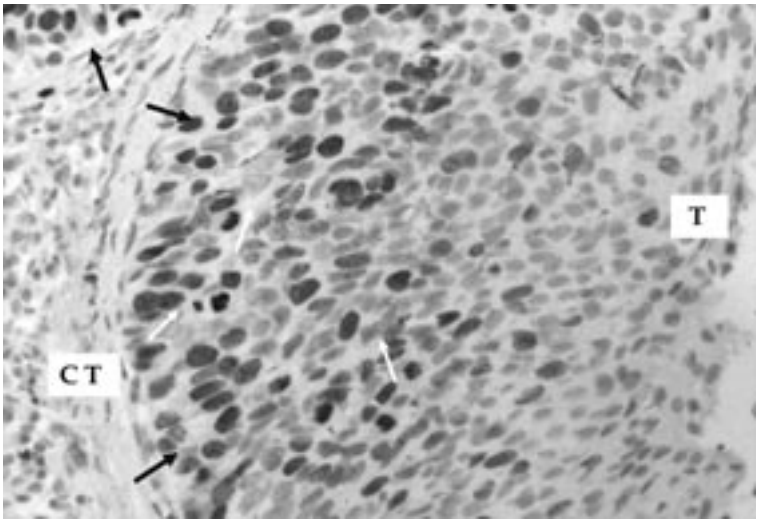
- 1+ : indicates <20% of positive-stained cells
- 2+ : indicates 20-50% of positive-stained cells
- 3+ : > 50% of positive-stained cells
- 0 : No immunoreactivity

Table 4: Relationship between MDM2 immunohistochemical phenotype and Ki-67 labelling index (LI).

Immunohistochemical phenotype	No. of cases	Ki-67 LI; Mean (%)± SD (Mean Rank)	p value T-test (Rank test)
MDM2 immunopositive cases	20	24.6 ± 17.0 (16.67)	0.577*
MDM2 immunonegative cases	11	21.5 ± 12.9 (14.77)	

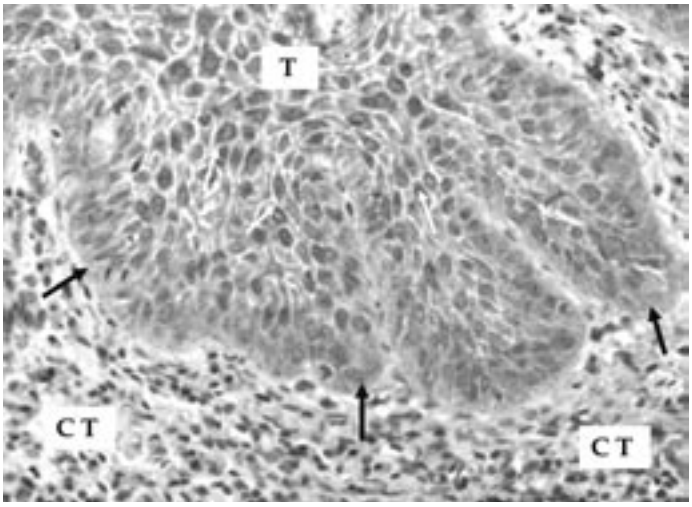
* p value were obtained from Mann-Whitney U test

Figure 1a = Immunoreactivity of Ki-67 at the tumour invasive front of OSCC in buccal mucosa.



Immunoreactivity of Ki-67 at the tumour invasive front of OSCC in buccal mucosa. Positive staining was located mainly in cells in the basal and suprabasal regions. Diminishing expression was noted towards the upper region or the centre of the tumour islands (T). Staining was almost entirely confined to the nuclei and consistent throughout the invasive front layer. The intensity of the staining was variable and ranged from faint to dark brown reaction. Black arrows show tumour invasive front; White arrows show positive Ki-67 staining; T – Tumour area; CT – Connective tissue area (Original Magnification X100)

Figure 1b - Immunoreactivity of MDM2 at the tumour invasive front of OSCC in buccal mucosa



Immunoreactivity of MDM2 at the tumour invasive front of OSCC in buccal mucosa. The positive staining was mainly at the suprabasal and at the upper regions. Immunoreactivity was predominantly a combination of nuclear and cytoplasmic. Nuclear staining showed accentuation at the nuclear membrane and the nucleoli Black arrows show tumour invasive front; White arrows show positive MDM2 staining; T – Tumour area; CT – Connective tissue area (Original Magnification X100)

Relationship between the immunohistochemical expression of Ki-67 and MDM2 at tumour invasive front of OSCCs in buccal mucosa with selected clinicopathologic parameters

The association of Ki-67 and MDM2 single expression and combined expression with sociodemographic and clinical histopathological parameters are shown in Tables 5 and 6 respectively. Only gender and Broders' tumour grading correlated significantly with MDM2 expression (Table 5) and the combined Ki-67/MDM2 expression (Table 6). Male subjects had significantly higher percentage of MDM2 expression than female subjects [male = 100%; female: 54.2%; p=0.026] (Table 5). Similarly, the male subjects had significantly higher combined Ki-67/MDM2 expressions than the female subjects [100% and 45.8% respectively; P = 0.025] (Table 6). Moderately-differentiated tumours had significantly higher percentage of MDM2 expression and the combined Ki-67/MDM2 expression than well-differentiated tumours [MDM2: Grade 2 = 81.8% and Grade 1 = 52.4%, P = 0.041; Ki-67/MDM2: Grade 2 = 90.0% and Grade 1 = 42.9%, P = 0.031 (Table 5 & 6)]. None of the sociodemographic and clinical histopathological factors was correlated with Ki-67 individually (Table 5).

DISCUSSION

The field of immunohistochemical analysis of tumour markers in the present study was limited to the tumour invasive front as this area was believed to be the most anaplastic,[7] containing the most aggressive cells with the ability to invade the surrounding tissues.[6,17]

Expression of Ki-67 and MDM2 at the tumour invasive front of OSCC in buccal mucosa

Ki-67 immunoreactivity was detected in 90.3% of the cases at the tumour invasive front. This high percentage recorded concurred with the results of other Ki-67 studies on OSCC. Ng *et al.*[16] had demonstrated 98.8% of immunoreactivity in their study while Sittel *et al.*[12] recorded complete immunoreactivity (100%) in their studies. It had been suggested that Ki-67 as a proliferating cell antigen should be detected in both normal and neoplastic tissues.[18] However, Ng *et al.*[16] demonstrated that its expression in normal oral mucosa was lower than in tumours. The lack of complete immunoreactivity has also been reported earlier[16] and even at the invasive front of OSSC.[19] Piffko *et al.*[19] had observed that Ki-67 immunonegative cases often showed pronounced maturation and keratinisation. Such observation was evident in central tumour areas where there is maturation and keratininsation in the present study. The presence of high percentage of Ki-67 expression at the basal and parabasal layer of the invasive front in this study supports the conclusion by Piffko *et al.*[19] that the invasive front might be the best field for growth fraction estimations.

Analysis of the pattern of Ki-67 immunoreactivity in the present study revealed that the staining was almost entirely confined to the nucleus with little and no cytoplasmic staining which was also observed earlier.[20] The distribution of nuclear labelling varied from little to none in the central areas and to high reactivity in the actively proliferating peripheral portions. This distribution of nuclear labelling was not consistent with the findings of other studies[16,17,20,21] which studied tumours from different parts of the head and neck. However, such distribution of Ki-67 nuclear labelling was not found in other sites (non-head and neck tumours).[22,23] Such different distribution of Ki-67 immunoreactivity noticed in different tumours from different sites of origin suggested cellular heterogeneity. Tumours originating from different stem cells have different biological properties.[20] Thus, our results were comparable with those studies done on head and neck tumours.

In the present study, the immunoreactivity detected for MDM2 was 64.5%. Significant discrepancies in the prevalence of MDM2 immunoreactivity in OSCC has been noted in different studies. The frequencies of MDM2 immunoreactivity have ranged from 12.2% to 96%. However, the immunoreactivity rate of MDM2 in the present study was only slightly lower than most of the other OSCC studies.[24-28] A much lower rate of immunoreactivity of 12.2% and 25.9% was recorded in two earlier studies.[16,29] The discrepancies observed may be due to the different background (geographic origin/ethnic group) of the subjects studied or the different visualisation techniques where some studies used an enhanced visualisation by En Vision while this study used the avidin biotin technique.

The present observation is comparable with studies from India and Taiwan involving quid chewing habit where similarly high immunoreactivity was observed.[24,26,27] MDM2 overexpression in these patients may reflect a persistent response to DNA-damaging agents present in the quid.[27]

Immunoreactivity of MDM2 demonstrated in the present study (64.5%) compared to those studies in USA (12.2%), UK (95.6%) and Hong Kong (25.9%) may partly be also due to the field of the present study which was restricted to the tumour invasive front.[16,25,29] The other studies looked at whole tumours. Unlike Ki-67, MDM2 staining has been mainly located at the suprabasal and superficial regions leaving only the basal layer negative.[16,28] Similar distribution was also noted in the present study. However, Piffko *et al.*[30] showed that the expression of MDM2 is in the basal and suprabasal area which is in contrast to the findings of this study. Thus, the value of evaluating MDM2 at the tumour invasive front is still controversial as this study showed that the localization of MDM2 is at the suprabasal and the upper region.

Studies by Agarwal et al.[24] and Ralhan *et al.*[26] have reported that the MDM2 positive cases showed nuclear staining ranging from faint to intense. Apart from main nuclear immunoreactivity, there were subset of cases that

Table 5: Association of Ki-67 and MDM2 immunohistochemical expression with sociodemographic and clinical histopathological parameters.

Sociodemographic and clinical histopathologic parameters	Total No. of cases	Immunohistochemical expression			
		Ki-67		*MDM2*	
		n (%)	*p value*	*n (%)*	*p value*
Age:					
<60 years	15	13 (86.7%)	0.505#	9 (60.0%)	0.611#
≥60 years	16	15 (93.8%)		11 (68.8%)	
Gender:					
Male	7	7 (100%)	0.325#	7 (100%)	0.026#
Female	24	21 (87.5)		13 (54.2%)	
Ethnic group:					
Indians	26	23 (88.5%)	1.000*	17 (65.4%)	1.000*
Non-Indians	5	5 (100%)		3 (60.0%)	
Habits:					
Only betel quid chewing	19	16 (84.2%)	0.147#	12 (63.2%)	0.841#
Others, combination or unknown	12	12 (100%)		8 (66.7%)	
TNM staging:					
Stage I, II	8	7 (94.4%)	0.754#	7 (87.5%)	0.115#
Stage III, IV	23	21 (91.3%)		13 (56.5%)	
Tumour size:					
T1, T2	18	17 (94.4%)	0.361#	11 (61.1%)	0.641#
T3, T4	13	11 (84.6%)		9 (69.2%)	
Nodal status:					
Node negative (N0)	7	6 (85.7%)	0.639#	6 (85.7%)	0.183#
Node positive (N1, N2, N3)	24	22 (91.7%)		14 (58.3%)	
Broders' tumour grading:					
Grade 1 (well-differentiated)	21	18 (85.7%)	0.208#	11 (52.4%)	0.041#
Grade 2 (moderately differentiated)	10	10 (100%)		9 (81.8%)	
Pattern of invasion:					
Cohesive (pattern 1&2)	19	18 (94.7%)	0.296#	13 (68.4%0	0.567#
Non-cohesive (pattern 3&4)	12	10 (83.3%)		7 (58.3%)	

* p values were obtained from the Fisher's exact test

p values were obtained from chi square test

Table 6: Association of Ki-67/MDM2 combined expression with sociodemographic and clinical histopathological parameters.

Sociodemographic and Clinical histopathologic parameters	Total no. of cases	IHC phenotype of combined expression	
		Ki-67/ MDM2 n (%)	*p value*
Age:			
<60 years	15	8 (53.3%)	0.605#
≥60 years	16	10 (62.5%)	
Gender:			
Male	7	7 (100%)	0.011#
Female	24	11 (45.8%)	
Ethnic group:			
Indians	26	15 (57.7%)	1.000*
Non-Indians	5	3 (60.0%)	
Habits:			
Only betel quid chewing	19	10 (52.6%)	0.440#
Others, combination or unknown	12	8 (66.7%)	
TNM staging:			
Stage I, II	8	6 (75.0%)	0.260#
Stage III, IV	23	12 (52.2%)	
Tumour size:			
T1, T2	18	10 (55.6%)	0.184#
T3, T4	13	8 (61.5%)	
Nodal status:			
Node negative (N0)	7	5 (71.4%)	0.667*
Node positive (N1, N2, N3)	24	13 (54.2%)	
Broders' tumour grading:			
Grade 1 (well differentiated)	21	9 (42.9%)	0.031#
Grade 2 (moderately differentiated)	10	9 (90.0%)	
Pattern of invasion:			
Cohesive (pattern 1&2)	19	12 (63.2%)	0.399#
Non-cohesive (pattern 3&4)	12	6 (50.0%)	

* p values were obtained from the Fisher's exact test

p values were obtained from the chi square test

demonstrated both nuclear and cytoplasmic staining and only staining in the cytoplasm and plasma membrane. The staining description was in agreement with the present study.

Relationship between the Ki-67 labelling index and the immunohistochemical expression of MDM2 at tumour invasive front of OSCCs in buccal mucosa

The presence of apoptosis indicated by MDM2 (64.5%) immunoreactivity along side with high Ki-67 (90.3%) immunoreactivity in the present study appeared to support the previous observation by Izawa *et al.*[21] and Macluskey *et al.*[31] that indices of proliferation and apoptosis were all increased with tumour progression but did so at different rates with proliferation starting earlier and proceeding at a higher rate than apoptosis.

Relationship between the immunohistochemical expression of Ki-67 and MDM2 at tumour invasive front of OSCCs in buccal mucosa with selected clinicopathologic parameters

The results in this study appeared to suggest an association between gender and MDM2 expression where higher percentage of MDM2 protein expression were seen in male (100%) subjects compared to females (54.2%). However, as the present study have very few male subjects (22.6%), further investigation is awaited as to whether such association between gender and MDM2 expression has any implication. The composition of sample in this study was very much influenced by the habits practiced, as the chosen oral mucosal site in this study was the buccal mucosa. However, no significant association was seen between habits and any of the markers studied (Ki-67 and MDM2). Huang *et al.*[27] had suggested that MDM2 may participate in the carcinogenesis of quid chewing-associated OSCC. Such observation was not obvious in the present study though the main habit recorded was quid chewing.

Previous studies[19,20,32,33] have preferred to correlate the expression of tumour markers (Ki-67 and MDM2) with tumour size and nodal status compared to the TNM staging. Though the present study correlated the expression of Ki-67 and MDM2 with TNM staging, tumour size as well as nodal status, no association was found between them. The present study was conducted at the tumour invasive front while most of these previous studies were evaluating the whole tumour except for Piffko *et al.'s*[19] study which also evaluated the tumour invasive front. These previous studies had not found any association between Ki-67 and MDM2 expression with the tumour size and nodal status. However, there has been one study on oral cancer by Ralhan et al. that found correlation between MDM2 expression and the tumour stage.[26]

Though no association was found between Ki-67 and the nodal status in the present study, the nodal metastasis and Ki-67 expression formed a particularly interesting group. One would expect a higher proliferative activity in nodal metastasis. The proliferative rate is generally held to be higher in metastasis than in primary tumours of the same histology. However, it is important to note that the proliferation rate comprises of two portions: the growth fraction (assessable by Ki-67) and the time taken for the cell to complete the cell cycle (not assessable by Ki-67). Thus, Ki-67 expression may not reflect the true proliferation rate as it does not reveal the cell cycle length. Whether or not this is an important distinction will only be made clear from studies on tumours with adequate clinical follow-up.

Tumour differentiation based on the Broders' grading is still widely used and is prognostically useful in histopathological diagnosis for the head and neck carcinomas.[14,34] However, this tumour grading has not evolved as an important factor in planning therapeutic strategies[4] as the difference in survival rates reported for well-differentiated and the poorly-differentiated was only 6%. From the prognostic point of view, The Royal College of Pathologists have stressed that it is important to separate well-differentiated tumours from the poorly-differentiated.[34] Though the present study found association between MDM2 expression and Broders' tumour grading, this association was inconclusive as the present sample lack poorly-differentiated tumours. In other studies,[19,21] it was claimed that Ki-67 expression was connected to the level of cell differentiation where the frequencies of Ki-67 staining increased markedly in less differentiated carcinomas compared to well-differentiated carcinomas.

CONCLUSION

The tumour invasive front may be suitable for evaluating Ki-67 and but may not be the best field in assessing the expression of MDM2 at this stage due to the conflicting results on the localization of its antigen in different studies. MDM2 expression and combined Ki-67/MDM2 expression were found to be significantly associated with gender and Broders' tumour grading. These associations however, should not be regarded with any importance as the sample was bias in distribution of male and female subjects and lacks poorly-differentiated tumour.

The findings in this study suggest that the expression of Ki-67 and MDM2 at the tumour invasive front of OSCC may be further studied overcoming aforementioned limitations to show possible prognostic significance. In order to achieve this, long-term patient follow-up is required which will allow comparisons of the survival patterns between those with higher and lower Ki-67 and MDM2 expressions at the tumour invasive front. A commitment to long-term patient follow-up and good record keeping of the follow-up by clinicians will further enhance the clinical applicability of these findings.

ACKNOWLEDGEMENTS

This study was supported by a research grant (FG 726 / 2000A) from University of Malaya, Kuala Lumpur, Malaysia.

REFERENCES

1. Bankfalvi A, Piffko J. Prognostic and predictive factors in oral cancer: the role of the invasive tumour front. J Oral Pathol Med. 2000;29:291-298.
2. Platz H, Fries R, Hudec M, Jjoa AM, Wagner RR. The prognostic relevance of various factors at the time of the admission of the patient. J Oral Maxillofac Surg. 1983;11:3-12.
3. Nicolson GL. Tumor cell instability, diversification, and progression to the metastatic phenotype: from oncogene to oncofetal expression. Cancer Res. 1987;47:1473-87.
4. Roland NJ, Caslin AW, Nash J, Stell PM. Value of grading squamous cell carcinoma of the head and neck. Head and Neck. 1992;14:224-229.
5. Bryne M, Boysen M, Alfsen CG, et al. The invasive front of carcinomas. The most important area for tumour prognosis. Anticancer Res. 1998;18:4757-4764.
6. Bryne M, Thrane PS, Dabelsteen E. Loss of expression of blood group antigen H is associated with cellular invasion and spread of oral squamous cell carcinomas. Cancer. 1991;67:618-623.
7. Bryne M, Koppang HS, Lilleng R, Stene T, Bang G, Dabelsteen E. New malignancy grading is a better prognostic indicator than Broders' grading in oral squamous cell carcinomas. J Oral Pathol Med. 1989;18:432-437.
8. Bryne M, Koppang HS, Lillenf R, Kjaerheim A. Malignancy grading of the deep invasive margins of oral squamous cell carcinomas has high prognostic value. J Pathol. 1992;166:375-381.
9. Odell EW, Jani P, Sherrif M. The prognostic value of individual histologic grading parameters in small lingual squamous cell carcinomas: the importance of the pattern of invasion. Cancer. 1994;74:789-794.
10. Woolgar JA, Scott J, Vaughan ED, Brown JS, West CR, Rogers S. Survival, metastasis and recurrence of oral cancer in relation to pathological features. Ann R Coll Surg Engl. 1995;77:325-331.
11. Spiro RH, Guillamondegui O, Paulina AF, Huvos AG. Pattern of invasion and margin assessment in patients with oral tongue cancer. Head and Neck. 1999;21:408-413.
12. Sittel C, Ruiz S, Volling P, Kvasnicka HM, Jungehulsing M, Eckel HE. Prognostic significance of Ki-67 (MIB1), PCNA and p53 in cancer of the oropharynx and oral cavity. Oral Oncology. 1999;35:583-589.
13. Xie X, Angelis PD, Clausen OPF, Boysen M. Prognostic significance of proliferative and apoptotic markers in oral tongue squamous cell carcinomas. Oral Oncology. 1999;35:502-509.
14. Pindborg JJ, Reichart PA, Smith CJ, Van Der Waal I. Histological typing of cancer and precancer of the oral mucosa. World Health Organization. Berlin: Springer-Verlag. 1997;11-12.
15. Anneroth G, Batsakis J, Luna M. Review of literature and a recommended system of malignancy grading in oral squamous cell carcinomas. Scand J Dent Res. 1987;95:229-49.
16. Ng IOL, Lam KY, Ng M, Regezi JA. Expression of p21/waf1 in oral squamous cell carcinomas-correlation with p53 and MDM2 and cellular proliferation index. Oral Oncology. 1999;35:63-69.
17. Kearsley JH, Furlong KL, Waters MJ. An immunohistochemical assessment of cellular proliferation markers in head and neck squamous cell cancers. Br J Cancer. 1990;61:821-827.
18. Brown DC, Gatter KC. Monoclonal antibody Ki-67: its use in histopathology. Histopathology. 1990;17:489-503.
19. Piffko J, Bankfalvi A, Ofner D, et al. In situ assessment of cell proliferation at the invasive front of oral squamous cell carcinomas. Virchows Arch. 1996;429:229-234.
20. Roland NJ, Caslin AW, Bowie GL, Jones AS. Has cellular proliferation marker ki-67 any clinical relevance in squamous cell carcinoma of head and neck? Clin Otolaryngol. 1994;19:13-18.
21. Izawa H, Yonemitsu N, Shin T, Sugihara H. Histopathological analysis of apoptosis and expression of p53, bcl-2, bax and Ki-67 in laryngeal squamous cell carcinomas and dysplasia. Auris Nasus Larynx. 1999;26:317-330.
22. Bouzubar N, Walker KL, Griffiths K, *et al*. Ki-67 immunostaining in primary breast cancer: pathological and clinical associations. Br J Cancer. 1989;59:943-947.
23. Porschen R, Lohe B, Hengels KJ, Borchard F. Assessment of cell proliferation in colorectal carcinomas using the monoclonal Ki-67; correlation with pathohistologic criteria and influence of radiation. Cancer. 1989;64:2501-2505.
24. Agarwal S, Mathur M, Srivastava A, Ralhan R. MDM2/p53 co-expression in oral premalignant and malignant lesions: potential prognostic implications. Oral Oncology. 1999;35:209-216.
25. Partridge M, Green MR, Langdon JD, Feldman M. Production of TGF- and TGF- by cultured keratinocytes skin and oral squamous cell carcinomas-potential autocrine regulation of normal and malignant epithelial cell proliferation. Br J Cancer. 1989;60:542-548.
26. Ralhan R, Sandhya A, Meera M, Bohdan W, Nootan SK. Induction of mdm-2-P2 transcripts correlates with stabilized wild-type p53 in betel and tobacco-related human oral cancer. Am J Pathol. 2000;157:587-596.
27. Huang J-S, Ho T-J, Chiang C-P, Kok S-H, Kuo Y-S, Kuo M Y-P. MDM2 expression in areca quid chewing-associated oral squamous cell carcinomas in Taiwan. J Oral Pathol Med. 2001;30:53-58.
28. Stoll C, Baretton G, Lohrs U. The influence of p53 and associated factors on the outcome of patients with oral squamous cell carcinoma. Virchows Arch. 1998;433:427-433.
29. Schoelch ML, Le QT, Silverman S, *et al.*. Apoptosis-associated proteins and the development of oral squamous cell carcinoma. Oral Oncology. 1999;35:77-85.
30. Piffko J, Bankfalvi A, Ofner D, et al. Proliferative (MIB1, mdm2) versus anti-proliferative (p53) markers in head and neck cancer. An immunohistochemical study. Pathol Oncol Res. 1996;2:37-42.
31. Macluskey M, Chandrachud LM, Pazouki S, *et al.* Apoptosis, proliferation and angiogenesis in oral tissue. Possible relevance to tumour progression. J Pathol. 2000;191:368-375.

32. Bettendorf O, Herrmann G. Prognostic relevance of Ki-67 antigen expression in 329 cases of oral squamous cell carcinoma. ORL J Otohinolaryngol Relat Spec. 2002;64:200-205.
33. Yao L, Iwai M, Furuta I. Correlations of bcl-2 and p53 expression with the clinicopathological features in tongue squamous cell carcinomas. Oral Oncology. 1999;35:56-62.
34. Helliwell TR, Woolgar JA. Minimum dataset for head and neck carcinoma. London: Royal College of Pathologists. 2000;1-23.

CORRESPONDENCE TO:

Professor Dr. Rosnah Binti Zain,
Department of Oral Pathology,
Oral Medicine & Periodontology,
Faculty of Dentistry, University of Malaya,
50603 Kuala Lumpur, Malaysia.
Email: rosnahmz@um.edu.my

Malaysian Dental Journal (2005) 26(2) 83-86

MALAYSIAN DENTAL JOURNAL

Patients Complaints of Dental Malpractice in Malaysia Part I: Procedure of Disciplinary Inquiry

Cheah CW *Final Year Dental Student 2005, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Soon HI *Final Year Dental Student 2005, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Ngeow WC *Lecturer, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Monerasinghe E *Principal Assistant Director, Oral Health Division, Ministry of Health of Malaysia, Putrajaya, Malaysia*

Lian CB *Retired Professor Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

ABSTRACT

A system for handling complaints regarding dental malpractice was established under the Dental Act 1971 and Dental Regulation 1976. The objectives of this study were to determine the complaint process brought to the Malaysian Dental Council (MDC), the number and types of complaints received from 1997 to 2004 and the dental protection scheme that is available in Malaysia. The first objective was studied by reviewing the process of handling complaints as stated in the Dental Act 1971 and Dental Regulation 1976. A detailed description of the procedure of disciplinary inquiry of the Preliminary Investigation Committee (PIC) & Malaysian Dental Council (MDC) is outlined in this article. Reports of all complaints directed to the MDC from 1997-2004 were analysed in the second part of this study.

Key words:
dental malpractice, litigation, discipline

INTRODUCTION

The complexity of modern living's influence on the health care profession has increased the challenge to provide ethical care in dental practice. As a result, there has been a large increase in the number of allegations of negligence brought against members of the dental profession.[1]

In Malaysia, several avenues exist for complaints of dental malpractice. These include the Malaysian Dental Council (MDC), the court, the newspaper and of late, the Complaints Bureau of the Malaysian Dental Association (MDA). The Complaints Bureau of the MDA has been set up in view of the increasing number of complaints received against dental practitioners. It has to be borne in mind that the Complaints Bureau of the MDA has no legal jurisdiction in handling complaints. It serves as an arbitration board to resolve disputes in an attempt to prevent them from becoming legal issues.

In most cases dentists do not know where they stand when faced with malpractice claims by patients or when receiving letters from the solicitor alleging negligence. Thus it is the main aim of the first part of this study to look into the procedure of inquiry of the MDC on disciplinary matters. Currently, limited knowledge exists regarding the MDC's management of patients' complaints as no study or publication is available.

MATERIAL AND METHODS

The authors studied all the information provided on the process of handling complaints as stated in the Dental Act 1971 and Dental Regulation 1976.[2] This information was then synthesised into a flowchart.

RESULTS

The enactment of the Dental Act in 1971 began the era of contemporary dentistry in Malaysia. Under the authority of this Act, Parliament has entrusted the dental profession with the responsibility for its own standard of conduct, with the establishment of an executive body, called the Malaysian Dental Council (MDC). The main functions of the MDC are to keep the Dental Register, to maintain standards in the profession and to enable powers for the profession to discipline its own members.

In section 38(4), Part IV of the Act, relating to Disciplinary Proceedings, the MDC exercises disciplinary jurisdiction over all registered dentists. By virtue of the powers granted to him, the President of the MDC shall appoint a committee from among registered practitioners, known as a Preliminary Investigation Committee (PIC). The PIC shall consist of not less than 3 and not more than 6 members. The function of the PIC shall be to make

preliminary investigations into complaints or information pertaining to any disciplinary matter.

It all begins with a complaint lodged against a dentist, either directly from the patient himself, or indirectly from a third party (MDA, Ministry of Health or consumer organisation) to the MDC. All complaints are referred to the Secretary of the MDC. The secretary then forwards the complaint to the President of the MDC for his advice. The President may decide to reject the complaint if the facts do not constitute any disciplinary matter, or forward the complaint to a PIC for further investigation.

The chairperson of the PIC to which such complaints or information has been forwarded, will call for the first meeting of the PIC with the Secretary of the MDC and a legal advisor. The role of the legal advisor is to assist the Council or Committee on legal matters during the inquiry, including the interpretation of the Dental Act. The character of the complaint will then be carefully assessed. The Committee will make their decision whether to summarily dismiss the complaint if it is satisfied that:

- The name & address of the complainant is unknown or untraceable,
- It does not constitute a disciplinary matter, or
- The information/complaint is doubtful

Where there is reason to believe in the authenticity of the complaint, the PIC shall convene on another date for the first hearing. At this hearing, the attendance of the complainant accompanied by all relevant evidences or witnesses in support of the allegation is required, with or without counsel. The practitioner against whom the allegation has been made, will also be informed of the substance of the complaint and his right to be present, with or without counsel, to cross-examine such person/s who may be called at the inquiry. The duties of the PIC during the hearing are to examine the complainant and his evidences/witnesses and shall reduce to writing the statements made by them.

The PIC shall then determine if there is a case to answer or otherwise. If there is no case to answer, the PIC report will be prepared for Council and the PIC recommendation will be to take no further action. If there is a case to answer, the PIC will draft charges and invite the practitioner to answer the charges at a date, time and place to be specified.

The PIC will then recommend to the Council to either take no further action or to convene a disciplinary inquiry (where it is determined that there are sufficient grounds to support the allegation exist). Subsequently, all records of the preliminary inquiry shall be transmitted to the Council in a PIC Report.

The decision now lies with the Council. After considering the report & recommendation by the PIC, the Council shall hold a disciplinary inquiry on the practitioner, even in a case where the PIC recommends that no action be taken. The Council may also decide that no inquiry will be needed, thereafter the charges will be dropped and the case will be closed. Before holding an inquiry, the Council shall cause to be served on the practitioner a notice specifying the date, time and place of the inquiry and a copy of the charges framed by the Council after a consideration of the records submitted by the PIC.

The practitioner is then invited to answer the charges. The practitioner shall be present to make any statement as he deems necessary and call such other person/s as he may require in support of his defence. The Council shall record such further or fresh statements. At the close of the inquiry, the Council shall then deliberate and reach one of the following verdicts:

- If the Council finds that there is no case against the practitioner, it shall direct that the charges be dismissed and inform the practitioner accordingly.
- If the Council finds the practitioner guilty of any disciplinary matter specified in section 32(2) of the Act, it shall inform the practitioner of its finding and the grounds for its decision signed by the President of the MDC.

The Council shall request such practitioner to make any plea in mitigation as he deems fit. After hearing the plea in mitigation, the Council shall exercise disciplinary jurisdiction with respect to its powers as specified in section 33 of the Act by imposing any of the following punishments:

- Order the name of the practitioner to be struck off from the Register
- Order the practitioner to be suspended from the Register for such period as it may think appropriate
- Order the practitioner to be reprimanded, or
- Make any order as foresaid but suspend the application thereof, subject to such conditions as the Council may deem fit, for a period, or periods in the aggregate, not exceeding 2 years.

A practitioner who is not satisfied by the order or decision of the Council, may appeal to the High Court. The High Court may thereupon affirm, reverse or vary the order or decision, or may give such direction in the matter as it deems proper. The decision of the High Court upon such an appeal shall be final.

The procedure of the disciplinary inquiry of the PIC and the MDC is summarised in the flow chart in Figure 1(a) & (b).

DISCUSSION

The Malaysian Dental Council is a statutory body and its function is to uphold the standards of practising dentistry in Malaysia.[2] In the interest of justice, all complaints should be dealt with as expeditiously as possible.[3] This is so indicated by the establishment of the PIC. In 1999, 2 committees, PIC 1 and PIC 2 were formed in Malaysia as a result of the increased number of complaints received by the Council.

The PIC is a wholly authorised body of the MDC. It operates in accordance with the aims and principles for which they were established. It makes no decisions involving monetary compensation for the complainants, but purely acts to ensure that the quality of service and commitment is maintained among members. Monetary claims are under the jurisdiction of the civil court.

The complainant is invited only for the first hearing to present his evidence. The hearing may require several days where evidences are shown or exhibited, or require the presence of witnesses and translators as the case may be. As a result, the whole investigation may stretch over a long period of time because the hearings held are subjected to the convenience of both the complainant, witnesses and even the practitioner and his legal advisor.

Figure 1a: Procedure of Disciplinary Inquiry of PIC of MDC

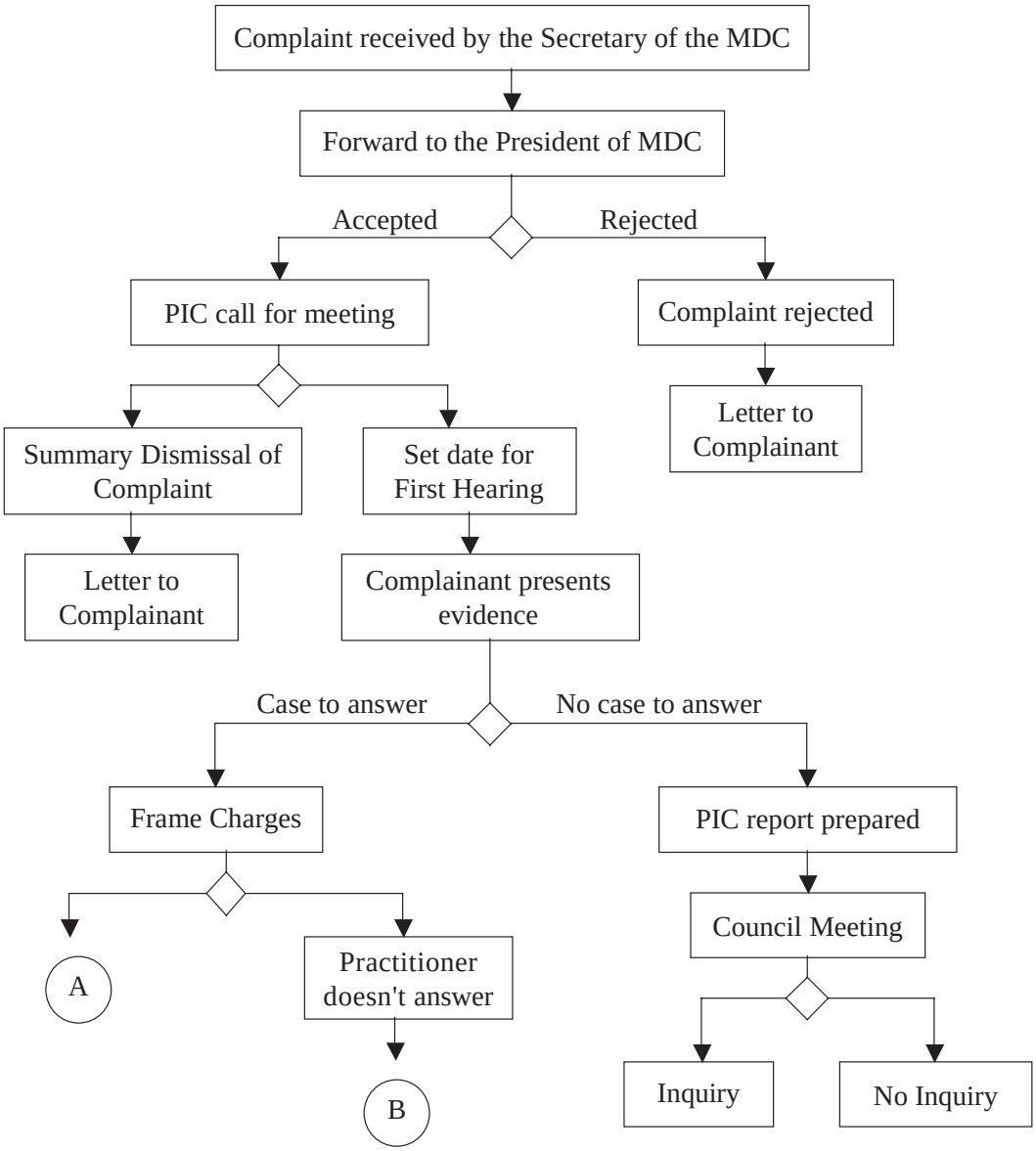
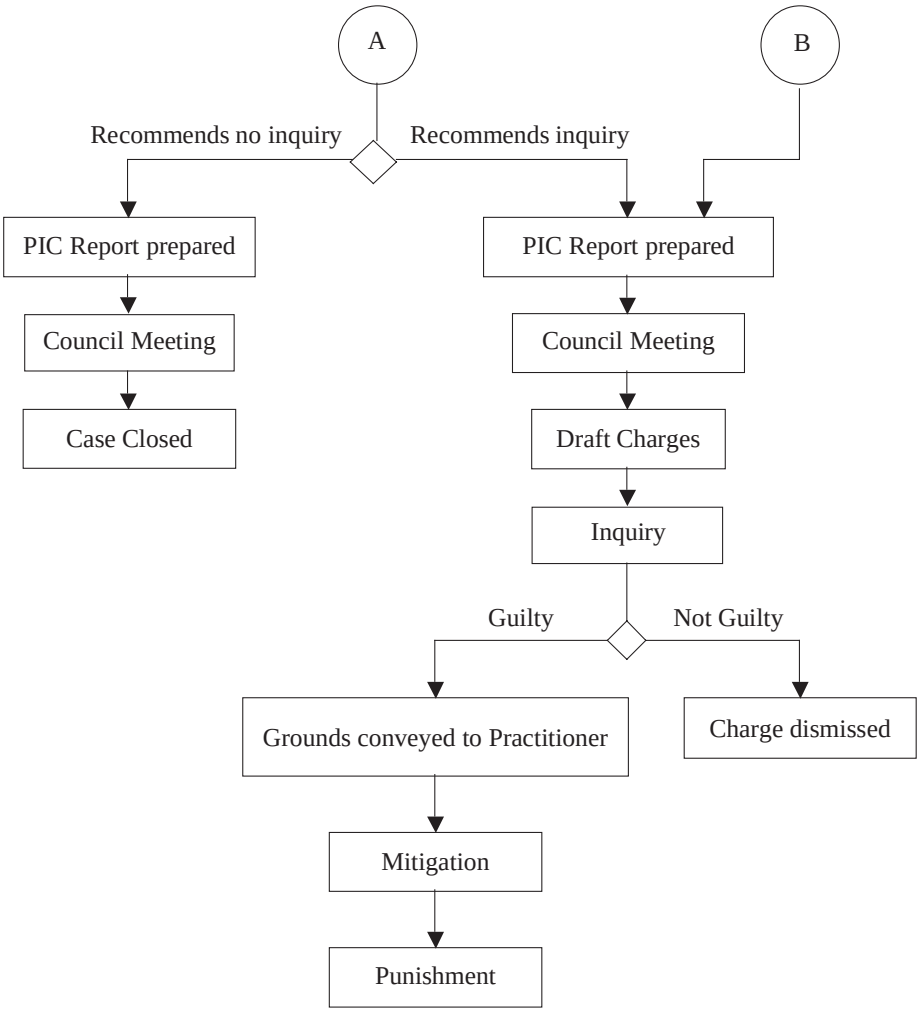


Figure 1b: Procedure of Disciplinary Inquiry of PIC of MDC (continued)



CONCLUSIONS

This study looked into the procedure of the disciplinary inquiry of the PIC, which plays a prominent role in assisting the council to assess complaints against dentists with regards to disciplinary matters. Under the Dental Act 1971 and Dental Regulations 1976, there is a whole section that provides the procedures for dealing with complaints against registered dentists and the implementation of appropriate punishments.

ACKNOWLEDGEMENT

The authors would like to thank the Director General of the Ministry of Health of Malaysia for his kind permission to publish this paper.

REFERENCES

1. Smith BGN. Professional negligence in dentistry. Dental Update. 1990;17:359-62.
2. Laws of Malaysia. Dental Act 1971 (Act 51) & Regulation. International Law Book Series. Updated 15 August 2003.
3. Watt DG. The Dental Council of New Zealand. New Zealand Dent J. 1983;79:15-6.

Address for correspondence:

Dr. Elise Monerasinghe,
Principal Assistant Director,
Oral Health Division,
Ministry of Health of Malaysia,
Level 5, Block E10, Parcel E, Precinct 1,
Federal Government Administrative Centre,
62590 Putrajaya, Malaysia.
Tel : 603-8883 4212
Fax : 603-8888 6133
Email : kld113@moh.gov.my

Malaysian Dental Journal (2005) 26(2) 87-91

MALAYSIAN DENTAL JOURNAL

Patients Complaints of Dental Malpractice in Malaysia Part II: Analysis of Cases from 1997-2004

Cheah CW *Final Year Dental Student 2005, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Soon HI *Final Year Dental Student 2005, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Ngeow WC *Lecturer, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

Monerasinghe E *Principal Assistant Director, Oral Health Division, Ministry of Health of Malaysia, Putrajaya, Malaysia*

Lian CB *Retired Professor Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia.*

ABSTRACT

A system for handling complaints regarding dental malpractice was established under the Dental Act 1971 and Dental Regulation 1976. The objectives of this study were to determine the process of a complaint that is brought to the Malaysian Dental Council (MDC), the number and types of complaints from 1997 to 2004 and the dental protection scheme that is available in Malaysia. This second part reviews the reports of all complaints directed to the MDC from 1997-2004. A total of 48 cases were filed. There was no specific trend of prevalence of complaints throughout the years. Almost two-thirds of the reported complaints were of negligence and fraud. The dental protection scheme available in Malaysia is offered by the Medical Protection Society and Dental Protection Limited (MPS/DPL), which is a foreign-based organisation, in association with the Malaysian Dental Association (MDA).

Key words:
dental malpractice, litigation, discipline

INTRODUCTION

Increasing consumerism and public expectation of public healthcare providers, coupled with a greater readiness to question the outcomes of medical and dental care, are resulting in the rapid escalation of the frequency and size of claims around the world.[1]

Most dento-legal difficulties arise as a result of 1 of 6 main problem areas:[2]

- Failure of communication
- Patient's expectations that are too high
- Insufficient information given to patient
- Practitioners attempting work beyond their abilities
- Practitioners not disclosing details of accidents or warning of possible consequences
- Practitioners showing indignation instead of compassion when questioned by patients or their representatives

We, as dental practitioners, should take note of these problem areas, and attempt to avoid these pit falls.

To date, no study has been carried out with regards to complaints of dental malpractice in Malaysia. Hence, the objectives of the second part of this study are:

- To document and analyse the number of complaints received by the Malaysian Dental Council (MDC) from 1997-2004
- To identify the type of professional misconduct claimed
- To identify the protection organisation and malpractice indemnity available in Malaysia

It is hoped that the information obtained in this study would be useful to the dental practitioners in Malaysia.

MATERIAL AND METHODS

Data on the distribution of registered practising dentists in Malaysia was obtained from the MDC. All the case records of complaints available from the MDC were collected only from the year of 1997 to 2004 (updated as of 17/5/04). Due to the confidentiality of these data, the fourth author, who is also the Secretary of the Malaysian Dental Council undertook the task to review this data. The data were categorised as follows:

- Number of complaints reported to the MDC from 1997-2004
- Analysis of substance of complaints

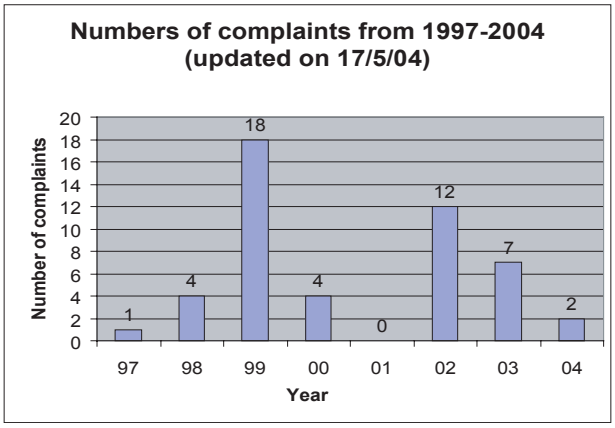
- Categorisation on complainants and practitioners by gender
- Distribution of dental practitioners in Malaysia by gender
- Distribution of Malaysian dentists and dentists with claims by gender
- Status of investigation summary of the PIC

Information regarding dental protection and malpractice indemnity was obtained from the Malaysian Dental Association (MDA). The organisation identified is based in the United Kingdom, which offers malpractice indemnity insurance and legal advice to dentists and operates 24 hours a day, in association with the MDA.

RESULTS

Number of Complaints from 1997-2004

The number of complaints across the country between 1997 and 2004 (updated as at 17/5/04) are summarised in Figure 1. The number ranged from 0-18 cases annually.



Source: MDC

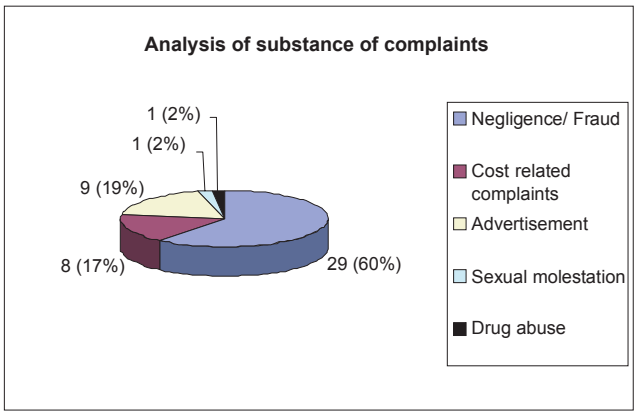
Figure 1: Numbers of complaints from 1997-2004

In 1997, only 1 complaint was received, followed by 4 in 1998. The year 1999 showed a drastic rise to 18 cases. The prevalence of complaints, however, did not increase in the following year, with only 4 cases. Surprisingly, none were received in 2001. The year 2002 showed a total of 12 complaints, which is the second highest over the 7 year period reported. Seven cases were reported in 2003 and two cases for 2004.

Analysis of Substances of Complaint

Figure 2 demonstrates that complaints were recorded most frequently in the aspect of negligence and/or fraud, which involved 29 cases (60.4%), and included wrongful extraction and the selling of medical certificates.

Advertising, with a total of 9 cases, accounted for 18.75% of the total cases. The number of cost related complaints was 8 or 16.7%. Of the categories stated, the least were sexual molestation and drug abuse (n=1; <1% each). In total, 38 cases (79.2%) were a result of professional misconduct.



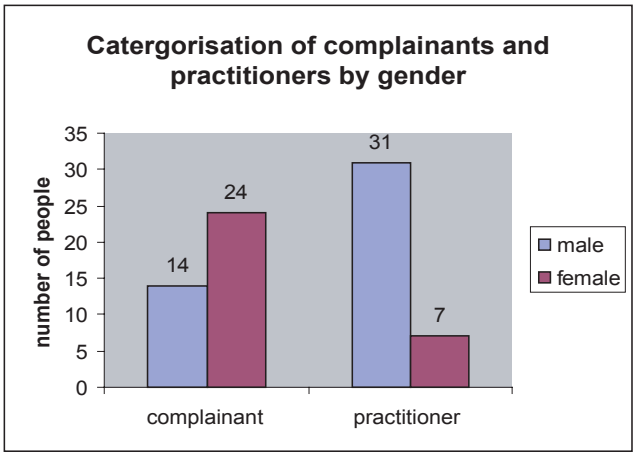
Source: MDC

Figure 2: Analysis of substance of complaints

Categorisation of Practitioners and Complainants by Gender

Analysis of the data shows that almost two thirds of the complaints (63.16%) were filed by female patients.

As for practitioners, of the complaints received, 81.6% were against male practitioners and 18.4% against female practitioners.



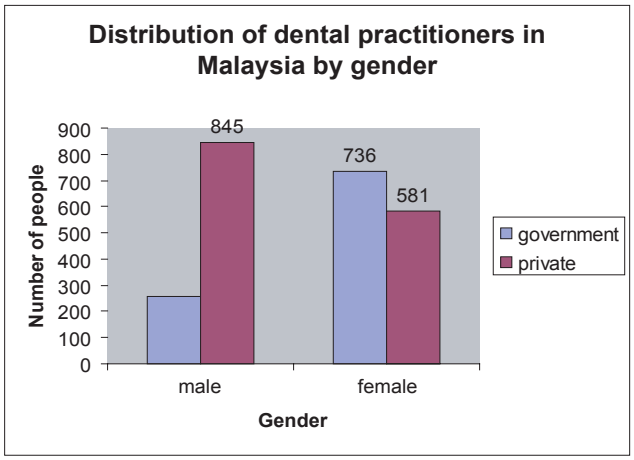
Source: MDC

Figure 3: Categorisation of practitioners and complainants by gender

Distribution of Dental Practitioners in Malaysia by Gender

As of 2004, a total of 2418 dental practitioners were serving in both the government sector and private practice. Of that total, 992 (41%) were government dentists. The majority of government dentists were female (n=736;

74%), with only 256 male dentists (26%). Almost 60% of the dentists in Malaysia were private practitioners (n=1,426). As shown in Figure 4, male dentists accounted for 845 (59%) of the private practitioners while the remaining 581 were female.

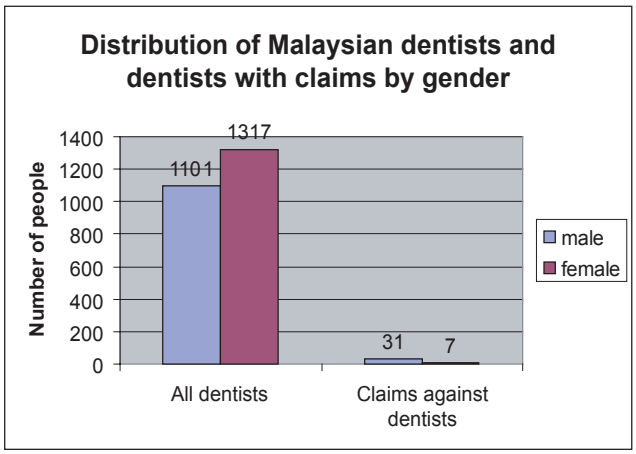


Source: MDC

Figure 4: Distribution of dental practitioners in Malaysia by gender

Distribution of Malaysian dentists and dentists with claims by gender

A total of 2,418 registered dentists were serving with the government and in private practice. Of that total, a majority were female dentists (n=1,317, 54.47%), the remaining 1,101 were male dentists. From the year 1997-2004, the total number of dentists who received complaints from patients was 38, and this accounted for 2.8% of male dentists and 0.5% of female dentists when analysed by gender.



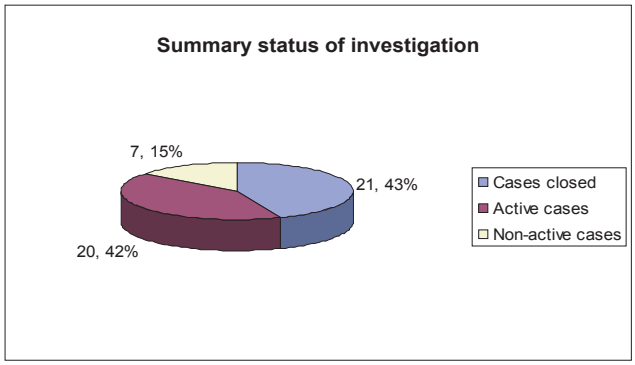
Source: MDC

Figure 5: Distribution of Malaysian dentists and dentists with claims by gender

Summary Status of Investigation

A total of 48 cases underwent investigation by the Preliminary Investigation Committees. Figure 5 illustrates that 21 cases (43.7%) had been completed or closed. The total of active cases were 20 (41.6%), of which 4 cases were under the hearing of *PIC1, 6 in *PIC2; and an additional 10 have reached the hearing stage of the MDC. Of the 7 non-active cases, 2 cases were pending PIC reports, while the remaining 5 were pending closure.

**PIC 1 and PIC 2 denotes that there are two separate PIC teams investigating different claims against different dentists simultaneously.*



Source: MDC

Figure 6: Summary status of investigation

Dental Protection and Malpractice Indemnity

The professional indemnity available in Malaysia is offered by a United Kingdom based organisation, in association with the Malaysian Dental Association (MDA); namely Dental Protection Limited (DPL), a subsidiary of Medical Protection Society (MPS).

It was established with the purpose of supporting, protecting and safeguarding the character and interests of medical and dental practitioners. It provides legal representation and advice from specialists in the field of dental litigation.

The MPS/DPL is a mutual organisation (non-profit) as opposed to other commercial insurance companies. The DPL publications, Riskwise Malaysia, and Annual Review, are well established to provide the members with a good working understanding of legal and ethical principles governing the practice of dentistry. It also promotes risk management through workshops, lectures and a series of multimedia training modules.

DISCUSSION

The doctor-patient relationship has achieved new dimensions with the increasing use of technology in medicine and dentistry. Doctors and dentists sometimes unconsciously take more risks with today's development in medical and dental technology. The issue of medical/ dental practice errors has become a symptom of this new dimension. This seems to attract much public attention especially in the mass media in some countries.[3]

In Malaysia, there are more private practitioners than government dentists in practice; however only 1 complaint was made against a government dentist. Due to lack of data on the circumstances of complaints, no appropriate conclusion can be derived as to why more private practitioner received complaints. However, another study in Turkey also showed that more dentists in private practice received complaints.[3] The study also recorded more male dentists received complaints than female dentists, which is similar to our findings.[3]

Even though the Malaysian Dental Council was established in 1971, the documentation (compiling, filing and summarising) of the complaints started only in 1997. The information contained in this paper was collected from 48 cases recorded over this period. The data obtained from this study indicated that there was an average of about 7 complaints per year, received and investigated by the MDC. There was no specific pattern in the number of complaints filed according to year, although the number of cases showed a sudden increase in 1999. Data on complaints elsewhere are hard to come by, but one that the authors found was reported in Turkey,[3] where they had only 14 cases of malpractice related to dentistry throughout a decade (1991-2000). Comparatively, the number of cases per year is higher in Malaysia and the percentage of dentists who have been complained against is also higher than the Turkish. The percentage of complaints against the population is also higher in Malaysia.

The substance of the complaints included negligence, fraud, cost-related complaints, and advertising. Negligence accounted for the highest number of complaints, and most of them were due to unfulfilled expectations of patients. There is a possibility that this problem is due in part to the lack of knowledge of contemporary treatment modalities and the breakdown of patient-dentist communication. The study in Turkey found a similar range of causes, though not entirely the same.[3]

Smith[4] stated that by following correct and prudent professional procedures all the time, dentists may be sensibly expected to avoid serious litigation. But in the medical field it has been found that most complaints against doctors were related to problems of communication, not clinical competence, and that most malpractice allegations arose from errors in communication.[5,6] Therefore, training in interpersonal skills and behaviour modification techniques is essential in improving dentist-patient relationship.

Analysis of data regarding the complainants' gender, demonstrated that more female patients filed complaints compared to male patients. According to the Population and Housing Census released in 2000, the ratio of males to females in the Malaysian population is 1.04:1. This doesn't coincide with our findings, and it may be related to the difference in the frequency of dental visits between males and females. Although the sex of the patient was a factor, no other characteristics of either the practitioner or patient could be ascertained to contribute to meaningful profiles of typical high risk malpractice complainant/receiver as these data are lacking. In contrast, the study in Turkey found more male patients lodging complaints.[3] A Danish study on the other hand, indicated that complaints were rare and that no significant association could be found between patients' inclination to complaint and their sex, education or working status.[7]

Education is an important element in the maintenance of standards. Reeves[8] suggested that dentists should be required to attend some postgraduate sessions each year. This would be advantageous for the maintenance of ethical standards, in addition to the obvious education benefits and keeping oneself updated. As for undergraduates, problem oriented ethics curriculum and behavioural science is necessary as part of the professional education.

There is only one dental protection and malpractice indemnity package available for dentists in Malaysia, which is provided by a United Kingdom based organisation. However, a few local medical practitioners are known to be forming a local body to provide for the professional indemnity needs of members in the medical field. Perhaps the dental fraternity should also look into forming their own local professional indemnity package as the cost of insurance has been escalating over the last few years. An area that may be new to Malaysian dentists is risk management.

Risk management has proven to be very successful, for instance among New York dentists in the United States.[9] With the application of risk management, that includes good record keeping, informed consent and vicarious liability as well as the latest innovations in dentistry, the DSSNY Council of Insurance managed to avoid a near disaster where dentists found difficulty in obtaining insurance cover. As a matter of fact throughout the 13 years of practice, they were even able to reverse the escalating cost of indemnity insurance.[9]

CONCLUSIONS

Although there are only a small number of complaints reported to the MDC, it is apparent that dental malpractice litigation is not new and neither will it discontinue. The result of this study could help dental practitioners to be more aware of the reasons for patient dissatisfaction.

ACKNOWLEDGEMENT

The authors would like to thank the Director General of the Ministry of Health of Malaysia for his kind permission to publish this paper.

REFERENCES

1. Lewis KJ. Riskwise Malaysia Jan 2004, Dental Protection, United Kingdom.
2. Moles DR, Simper RD, Bedi R. Dental negligence: A study of cases assessed at one specialized advisory practice. Br Dent J. 1998;184:130-3.
3. Ozdemir MH, Saracoglu A, Ozdemir AU, Ergonen AT. Dental malpractice cases in Turkey during 1991-2000. J Clin Forensic Med. 2005;12:137-42.
4. Smith BGN. Professional negligence in dentistry. Dent Update. 1990;17:359-62.
5. Richards T. Chasms in communication. BMJ 1990;301: 1407-8.
6. Shapiro RS, Simpson DE, Lawrence SL, Talsky AM, Sobocinski KA, Schiedermayer DL. A survey of sued and non-sue physicians and suing patients. Arch Intern Med. 1989;149:2190-6.
7. Schwarz E. Patient complaints of dental malpractice in Denmark 1983-86. Community Dent Oral Epidemiol. 1988;16:143-7.
8. Reeves JF. Standards in Dentistry. Br Dent J 1984;156:123.
9. Feldman MJ. Dentistry and malpractice. Looking back on 13 years. New York State Dent J. 1998;64:24-5.

Address for correspondence:

Dr. Elise Monerasinghe,
Principal Assistant Director,
Oral Health Division,
Ministry of Health of Malaysia,
Level 5, Block E10, Parcel E, Precinct 1,
Federal Government Administrative Centre,
62590 Putrajaya, Malaysia.
Tel : 603-8883 4212
Fax : 603-8888 6133
Email : kld113@moh.gov.my

Malaysian Dental Journal (2005) 26(2) 92-96

MALAYSIAN DENTAL JOURNAL

Oral Maxillofacial Tumour: Preliminary Review of Cases from 1999 to 2003 Faculty of Dentistry UKM

Rosli TI, BDS (Mal), DDPHRCS (Eng), MSc (London) *Lecturer, Department of Dental Public Health, Faculty of Dentistry, UKM, Malaysia*

Hussaini HM, BDS (Mal), MDentSci (Leeds), FDSRCSEd *Head, Department of Oral Pathology and Oral Medicine, Faculty of Dentistry, UKM, Malaysia*

Ma MS, BDS (Mal), MDentSci (Leeds), FDSRCS (Eng) *Bahagian Pergigian, Angkatan Tentera Malaysia, Kuala Lumpur, Malaysia*

ABSTRACT

The aim of this study is to determine the incidence and frequency of oral maxillofacial tumour reported by Department of Oral Pathology and Oral Medicine, Faculty of Dentistry Universiti Kebangsaan Malaysia (UKM) during the five years period from 1999 to 2003. A total of 226 reports were reviewed. Majority of the specimens reported were sent by the oral surgeons and dental practitioners in the Oral Maxillofacial Unit Hospital Universiti Kebangsaan Malaysia (HUKM), Faculty of Dentistry UKM and Oral Health Division, Kementerian Pertahanan Malaysia. Two hundred and nine diagnosed tissues (87%) were of benign in nature and 17 (7.5%) were malignant. Pyogenic granuloma was the most common lesion with 23 cases (10.2%) reported, followed by squamous cell carcinoma (6.6%), fibroepithelial polyp / fibrous epulis (6.2%), mucous extravasation cyst (6.2%) and ameloblastoma (6.2%). Of the 17 malignancies retrieved, squamous cell carcinoma (n=15) was the most predominant. The relative frequencies of most oral maxillofacial tumour seen in UKM dental faculty are comparative to most similar surveys in the literature particularly with the ones in South East Asia region. Squamous cell carcinoma was ranked 2nd in the series of diagnosed lesion and was the commonest malignancy seen.

Key words:
tumour, oral, maxillofacial, Asia

INTRODUCTION

Incidence of oral maxillofacial tumour in Malaysia has not been widely reported. Most local report and surveys were done on oral precancer and cancer as well as mucosal lesions. Local data on overall incidence and frequency of odontogenic tumours, salivary gland and soft tissue tumours are not available in literatures searchable through common scholastic search engine (eg MEDLINE). The understanding of pathological conditions occurring in the oral cavity is important. Clinician usually proposed a provisional diagnosis with clinical and radiographic findings, which later confirmed or excluded by doing a histopathological examination. Therefore, the knowledge of frequency and incidence coupled with social and demographical data is pertinent not only in managing the patient but in terms of prevention and policymaking.

The establishment of National Cancer Registry (NCR) by Malaysia and most countries provide evidence that systematic collection, analysis and interpretation of cancer data essential to the planning, implementation and evaluation of clinical and public health services.[1] Proper record keeping and systematic reporting is therefore essential in any organization that does histopathological reporting. Development of minimal dataset, clinical data and report forms need to be audited from time to time. This in turn will produce good governance of record keeping and reporting.

Objectives

The objectives of this survey are; to determine the incidence and frequency of oral maxillofacial tumour reported by Department of Oral Pathology and Oral Medicine, Faculty of Dentistry UKM during the five years period from 1999 to 2003. Other objectives would be to serve as an audit of our unit, record keeping and diagnostic laboratory procedures.

MATERIALS AND METHODS

Histopathological reports of oral biopsies were retrieved from archives, dating from 1999 to December 2003. The data was then examined and sorted according to the diagnosis as reported. Demographic data such as age, race and gender were obtained. The lesions were then assorted according their type and tabulated accordingly.

RESULTS

A total of 226 reports from 1999 to 2003 were retrieved. Some reports were excluded since no proper documentation and record keeping was established before the oral pathology lab was in operation. A majority of the specimens reported were sent by the oral surgeons and dental practitioners in the Oral Maxillofacial Unit HUKM, Faculty of Dentistry UKM and Oral Health Division, Kementerian Pertahanan. A small percentage was sent by private dental practitioner and referred cases from neighbouring government clinic in Klang Valley. All of the specimens were read by an attending oral pathologist.

Subjects

Analysis of the data showed male to female patient presented at a ratio of 3:4. Thirty-one percent (n=71) of the patients were in the 1st quarter of life, 21% (n=47) in the 2nd quarter of life and 25% (n=67) were more than 50 years old.

Oral Lesions

A total of 209 (87%) diagnosed tissue reported were benign in nature and 17 (7.5%) were malignant. About 6% of lesions were reported as normal tissues. The distribution of all diagnosed tissue is shown in Table 1.

Table 1: Distribution of diagnosed benign and malignant tissues

Lesion	n	%
Benign	196	86.7
Malignant	17	7.5
Normal	13	5.8
Total	226	100

The 15 most common oral lesions reported are presented in Table 2. Pyogenic granuloma was the most common lesion with 23 cases (10.2%) reported, followed by squamous cell carcinoma (6.6%), fibroepithelial polyp / fibrous epulis (6.2%), mucous extravasation cyst (6.2%) and ameloblastoma (6.2%). Of the 17 malignancies retrieved, squamous cell carcinoma (n=15) was the most predominant. Two other malignant cases reported were salivary gland cancers (0.9%).

Forty-one percent (n=93) of the lesions were of mesencymal and inflammatory origin. Pyogenic granulomas were the most common, followed by fibroepithelial polyp/fibrous epulis. Odontogenic cysts and tumours made up 19% of all lesions diagnosed. Ameloblastomas were the most common tumour, then followed by radicular cyst and OKC.

Comparisons of diagnosed lesions by age group showed that majority of the reported cases were those in the first quarter of life. Out of 69 lesions diagnosed amongst the 1st quarter of life group, 68 were benign in nature and 1 case of malignancy. The highest percentage of malignant lesions occurred in patients aged 50 and above, accounting for 65% of all malignancies. The distribution of lesions by age group is shown in Table 3.

DISCUSSION

The faculty of Dentistry UKM was established in June 1996. Student intake commenced in 1997 and later on, minimal clinical services were assumed in order to meet the students' requirement. In 1998, Hospital Universiti Kebangsaan Malaysia (HUKM) began operating and the department of Oral Maxillofacial was established. By then, the demand for oral histopathological examination was gradually needed due to the specimens sent in by the oral surgeons. At this time, this service was facilitated with the help of an Oral Pathologist from Kementerian Pertahanan (KEMENTAH) Malaysia. An oral pathology laboratory was then established in 1999 at the Dental Faculty UKM. Most histopathological examination requests came from Dept of Maxillofacial HUKM, Dental Faculty UKM and KEMENTAH. The Department of Oral Pathology and Oral Medicine was finally established in 2003 and the yearly number of biopsies send and reported has been increasing since the early days. It has come to our attention that during the early days of establishment, record keeping was not regularly updated. Some demographic data were missing or 'not filled'. This is perhaps due to lack of permanent staff, improper filling of forms by officers and lack of experience in lab protocols. However with the establishment of the department, better governing measures have been introduced. Proper protocols and link with general pathology lab in HUKM has tremendously improved our service.

Retrospective surveys on histopathological records particularly in this Southeast Asia region were reported in literature. Singapore has published two extensive surveys, one reported by Zhao[2] covers from 1954 to 1993 and recently reported by Tay[3] from 1993-1997. Indonesia has published one survey reported by Budhy *et al.*[4] which covers east Java from 1987 to 1992. Our centre is one of the newly established tertiary referral centre for most of dental problems in Malaysia, therefore the profile of cases seen may reflect a small subset of oral pathology pattern particularly in the Klang valley. It would not be prudent to extrapolate the incidence of oral maxillofacial tumour in Malaysia from our data since most cases go to Oral Stomatology Division IMR and Universiti Malaya (UM), which has a long established oral histopathological services. It would be relevant however, to compare our

Table 2: Frequency distribution of common oral lesions

Diagnosis	No	% of all biopsies
Pyogenic Granuloma	23	10.2
Squamous Cell Carcinoma	15	6.6
Fibroepithelial Polyp / Fibrous Epulis	14	6.2
Mucous Extravasation Cyst	14	6.2
Ameloblastoma	14	6.2
Radicular Cyst	12	5.3
Granulation Tissue	9	4.0
Lichen Planus	7	3.1
Odontogenic Keratocyst	6	2.6
Non-Specific Ulcer	6	2.6
Inflammatory Cells	6	2.6
Keratosis / Leukoplakia / Epidermal Hyperplasia	6	2.6
Periapical Granuloma	5	2.2
Fibrous Hyperplasia / Denture Induced Hyperplasia	5	2.2
Dentigerous cyst	2	0.9
Mucous retention cyst	2	0.9
Epidermoid cyst	2	0.9
Others (e.g. nasopalatine cyst, pigmented lesion, fibroosseous lesion, 'skin-tag', chronic marginal gingivitis, verrucous hyperplasia, developmental disorder)	38	16.8
Normal tissue	13	5.7
Total	226	100

Table 3: Distribution of lesions by age group

	Age Group		
Lesion	1st quarter	2nd quarter	3rd quarter
Benign	68	39	43
Malignant	1	4	11
Total	69	43	54

findings with those reported surveys especially within our region. One of the earliest Malaysian series on oral maxillofacial tumour, was by Ramanathan[5] in 1979. Other surveys can be found but mostly specific to certain type of lesion or tumour type. Lian[6] in 1990 and Rengaswamy[7] in 1976 did a series on odontogenic cyst, while Siar[8,9] did a series on squamous cell carcinoma in 1985 and 1990.

Malignancies versus benign

As much as 87% of the diagnosed tissues in our centre were benign in nature and 7.5% were malignant. In Singapore, Zhao[2] reported that 6.1% of biopsied tissue were malignant and more recently Tay[3] reported 5.2% in his series. East Java, Indonesia reported 45.3% of biopsied tumours were malignant.[4,10] In comparison to Malaysia, Ramanathan[5] in 1979 reported that malignant tumours formed 22.5% of all the specimens reported. Reports from most western countries[11,12,13] would range from 1% to 5% in their series. Although our data was almost similar to Singapore, this has not taken into account malignant tissue sent to Universiti Malaya and IMR. Therefore, in terms of biopsied tissue this could well be under reporting. Data from National Cancer Registry 2003[1] reported that incidence of oral cancer is 5.2 per100,000 or forms about 1.2% of all malignancies in Malaysia.

General

Pyogenic granulomas ranked first in our series making up 10.2% of the biopsied tissues reported. Most Western series[11,12,13] including those from Singapore,[2,3] would ranked pyogenic granulomas as 10th or 11th in their series, which would make up for about 2.6% to 5 % of overall biopsied tissues.

Fibroepithelial polyp/fibrous epulis ranked third in our series with 6.2% of biopsied tissues reported, meanwhile mucocele or mucous extravasation cyst were 4th with 6.2% reported cases. This scenario is almost similar to those in other series and both are among the top 5 most common diagnosis.[2,3,11,12,13]

Odontogenic Cyst

Among cystic lesions, radicular cyst would be the most common and ranked 6th in the present series making up of 5.3%. Meanwhile, other odontogenic cysts such as odontogenic keratocyst (OKC) and dentigerous cyst were less common findings. These findings would be similar to the local series by Lian in 1990[6] and Rengaswamy in 1976.[7] Western studies[11,12,13] have reported that the incidence of OKC would be around 3% to 12%. Lian reported that the incidence of OKC in his series was 17%. The Singaporean series[3] reported the incidence of OKC to be 2.4% which is almost similar to ours (2.6%). They also concluded that radicular cyst is the most common finding among all odontogenic cysts.

Odontogenic Tumour

In the present series we only recorded one type of odontogenic tumour which is ameloblastoma. Fourteen cases of ameloblastoma were seen and this ranked ameloblastoma fifth in our series. Ameloblastomas make up about 6.2% of total number of tissues diagnosed. This figure tends to be comparable to those in most Asian series. Budhy et al from East Java, Indonesia[4] reported an incidence 6.3% in ameloblastoma. The Singaporean series by Tay[3] reported ameloblastoma to make up 5% of total oral biopsies and a previously Malaysian study[14] also reported almost a similar figure. Most African[15] or Asian series would rank ameloblastoma as their most common odontogenic tumour seen. Some western surveys[11,12,13] have found that odontome were the most common odontogenic tumour.

Oral cancer

Squamous cell carcinoma formed 6.2% of our total oral biopsies and was ranked second in our series. Squamous cell carcinoma was also the most common (89%) oral malignancy with 15 recorded cases out of 17 malignancies seen. This seems to be in agreement with the Malaysian survey by Ramanathan & Ng[5] in 1979, which reported that 91% of total cases of malignancies were squamous cell carcinoma. Budhy et al from East Java, Indonesia[10] reported 70% of their malignant cases were squamous cell carcinoma, while the Singaporean National Cancer Registry in 1997 (5 year period) reported that 87% of oral malignancies were squamous cell carcinoma.[16] Our data did not permit further analysis on gender and racial breakdown on incidence of oral squamous carcinoma, however Siar CH in 1985 and 1990[8,9] has published excellent local data on gender, racial and occurrence site of squamous cell carcinoma.

Although our data is too small for reasonable conclusion, it has shown that squamous cell carcinoma remains as the most common oral malignancy, even with the advancement of sciences. It is timely that the establishment of National Cancer Registry and Oral Cancer Research Coordinating Centre (OCRCC) at the University Malaya can better monitor the progress of oral cancer in Malaysia. Population-based surveys routinely done by the Oral Health Division as well as the National Primary Prevention and Early Detection of Oral Precancer and Cancer programme by the Oral Health Division, will also be able to observe and manage oral cancer in the population more effectively.

CONCLUSION

The relative frequencies of most oral maxillofacial tumour seen in UKM dental faculty are comparative to most similar surveys in the literature particularly with the ones in the South East Asia region. Squamous cell carcinoma ranked second in the series of diagnosed lesions and was the commonest malignancy seen. Proper record keeping, good governance and the setting up of a national dataset will definitely be able to monitor progress of oral tumours particularly the malignant ones. At the moment, we are in the process of another five years (2003-2008) audit and it would be interesting to compare the result.

ACKNOWLEDGEMENT

The authors would like to thank UKM dental faculty for their support and this research was made possible with UKM fundamental grant (DD1/2002/5).

REFERENCES

1. National Cancer Registry www.crc.gov.my/ncr
2. Zhao YY, Yeo JF. Oral pathological survey of 11,347 biopsy specimens from 1954 to 1993 in Singapore. J Oral Pathol Med. 1996;25:278
3. Tay ABG. A 5-year survey of oral biopsies in an Oral Surgical Unit in Singapore: 1993-1997. Ann Acad Med Singapore. 1999;28:665-71
4. Budhy TI, Soenarto SD, Yaacob HB, Ngeow WC. Changing incidence of oral maxillofacial tumours in East Java, Indonesia 1987-1992. British J of Oral and Maxillofacial Surg. 2001;39:210-213
5. Ramanathan K, Ng KH. Malignant oral tumours in Peninsular Malaysia – A preliminary report on 2,263 cases. Med J Malay. 1979;33:342-345
6. Lian CB. Cyst of the jaw, analysis of cases from the faculty of Dentistry, University of Malaya. Mal J Sur. 1990;9:82-87
7. Rengaswamy V. Clinical Statistics of Odontogenic cysts in West Malaysia. Br J Oral Surg. 1977;15:160-165
8. Ng KH, Siar CH, Ramanathan K, Murugasu P, Chelvanayagam P.I. Squamous cell carcinoma of the oral mucosa in Malaysia – Any changes? Southeast Asian J Trop Med Publ Health. 1985;16:602-606
9. Siar CH, Ng KH, Mah CF, Ling CC. Oral squamous cell carcinoma in Peninsular Malaysia. Asian Med J. 1990;33: 697-703
10. Budhy TI, Soenarto SD, Yaacob HB, Ngeow WC. Changing incidence of oral maxillofacial tumours in East Java, Indonesia 1987-1992. Part 2: Malignant tumours British J of Oral and Maxillof Surg. 2001;39:460-464
11. Krutchkoff DJ, Chen J, Eisenberg E, Katz RV. Oral cancer: a survey of 566 cases from the University of Conneticut Oral Pathology Biopsy Service, 1975-1986. Oral Surg Oral Med Oral Pathol. 1990;70:192-8
12. Weir C, Davenport WD, Skinner RL. A diagnostic and epidemiologic survey of 15,783 oral lesions. J Am Dent Assoc. 1987;115:439-41
13. Thompson CC. A six year regional report on the oral tumour registry and lesions diagnosed in the School of Dentistry Biopsy Service University of Oregon Health Sciences Centre (Portland, Oregon). J Oral Med. 1981;36:11-4
14. Boon LC, Abu Kasim NH. Jawbone tumours. Malaysian J Surg. 1984;8:120-126.
15. Chidzonga MM, Lopez VM, Portilla Alvarez AL. Orofacial biopsies: a survey of 1,732 cases seen over a 10 year period. Cent Afr J Med. 1996;42:102-8
16. 1997 Health Facts, Singapore. Singapore Ministry of Health, 1997.

Address for correspondence:

Dr Haizal Mohd Hussaini
BDS (Mal), MDentSci (Leeds), FDSRCS (Edin)
Head Dept of Oral Pathology and Oral Medicine
Faculty of Dentistry,
Universiti Kebangsaan Malaysia
Jalan Raja Muda Abdul Aziz
50300 Kuala Lumpur, Malaysia.

Malaysian Dental Journal (2005) 26(2) 97-104

MALAYSIAN DENTAL JOURNAL

Prevalence of Periodontal Disease among Adults in Trivandrum District, Kerala, India

Jayakrishnan R *Lecturer, Division of Community Oncology, Regional Cancer Centre, Trivandrum, Kerala, India*

Sarma PS *Additional Professor, Achutha Menon Centre for Health Science Studies, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India*

Thankappan KR *Additional Professor, Achutha Menon Centre for Health Science Studies, Sree Chitra Tirunal Institute for Medical Sciences and Technology, Trivandrum, Kerala, India*

ABSTRACT

***Background*: Periodontal disease is a risk factor for tooth loss and systemic diseases like cardiovascular disease. We conducted a study to estimate the prevalence and correlates of periodontal disease among adult population in rural Trivandrum district of Kerala state. *Methods*: A community based, cross-sectional survey of 504 individuals in the 30-50 year age group (Mean age 39.9±7.1 years) was conducted using a cluster sampling technique. A pre-tested structured questionnaire was followed by clinical examination of the oral cavity and periodontal disease was diagnosed on the basis of WHO Community Periodontal Index of Treatment Needs (CPITN). Bivariate associations were examined using the Chi-square test, while multiple logistic regression was used to evaluate the association of select risk factors with periodontal disease. *Results*: The overall prevalence of periodontal disease in our study was 65.3%(95%CI: 61.1-69.4). The prevalence of periodontal disease was highest in the 45-50 year age group (OR=5.3; 95%CI 3-9.6) compared to the 30-34 year reference group and also among current tobacco smokers (OR=2.6; 95%CI 1.3 - 5.4) compared to non smokers. We noted a progressive nature of the disease with increase in age. *Conclusions*: In our study, approximately two thirds of the adult population had periodontal disease. Educational efforts targeted to avoid smoking in non-smokers and to quit smoking among smokers should be considered at the community level. Dental checkups are highly recommended in an early age considering the progressive nature of the disease with age.**

Key words:
periodontology, periodontal disease, prevalence, India

INTRODUCTION

Periodontal disease afflicts many individuals at some point in life and is a major cause of tooth loss in adults.[1] Periodontitis or inflammation of the periodontium results from the extension of inflammatory process initiated in the gingiva to the supporting periodontal tissues resulting in bone damage, loosening and eventual loss of teeth.

Modern periodontal epidemiology rediscovered focal infections and indicated that general health has a crucial impact on the periodontal health. Periodontal disease in turn has a major impact on the general health status of the patient.

A strong association has been shown between periodontal disease and coronary heart disease,[2-4] as well as between periodontal disease and cerebrovascular diseases (stroke).[5]

While a significant proportion of population is susceptible to periodontitis, it appears that there is a large segment of population that is not susceptible to the severe forms of periodontitis. This observation points to the risk factors that modulate susceptibility or resistance to destructive periodontitis.[6,7]

Studies had shown that tobacco smoking is a risk factor for lung cancer and cardiovascular diseases. The World Health Organization has estimated that tobacco related mortality is steadily increasing and the figure is expected to rise to 10 million deaths per year by 2020 or early 2030 with 70% of deaths occurring in developing countries.[8]

Current data has shown that dental plaque is the cause of periodontitis and cigarette smoking is a known risk factor, which adversely influences periodontal health.[9-11] There is a growing body of scientific evidence indicating that nicotine contributes to the progression of periodontal disease, and is detrimental to healing following periodontal therapy.[12] A recent study reported that exposure to environmental tobacco smoke has deleterious effects on the periodontium, resulting in periodontal disease.[13] Studies conducted to determine the effects of smokeless tobacco on the periodontium had shown marked gingival recession in smokeless tobacco users but were unable to confirm a consistent association between use of smokeless tobacco and generalized periodontal diseases.[14, 15] From the Indian context, very limited studies were reported to determine the impact of tobacco consumption on the periodontium.

In the Indian sub-continent, tobacco use in betel quid chewing is very popular especially in the rural areas and the consumption pattern is entirely different from the rest of the world.[16] So far, no study has been conducted to

estimate the prevalence of periodontal disease in rural Kerala. Kerala is relatively a small state in the southwest corner of the Indian sub-continent representing 3% of India's total population of 1027 million.[17] Health conditions in Kerala have always been better than those in other parts of India. This continued improvement has been tied to the population educational attainments and also to health sector reforms in the state.[18] In spite of remarkable achievements in the health sector, tobacco smoking and chewing are widely practiced in Kerala. The prevalence of any form of tobacco use in Kerala among people above 15 years in rural area in 1988 was 44.6% among males and 13.4% among females.[19]

Our objectives were to estimate the prevalence of periodontal disease among adult population in rural Trivandrum district, Kerala and to compare the periodontal disease status among tobacco smokers, chewers and nonusers of tobacco.

Subjects and Methods:

We conducted a cross-sectional survey of subjects in the 30-50 years age group in three Community Development Blocks (CDB) i.e. administrative units within districts of rural Trivandrum district, Kerala. We selected one CDB randomly from each geographical regions of high-land, mid-land and low-land in Trivandrum district.

Two village Panchayaths were randomly selected from each CD block and all the wards in each of the selected Panchayath were included for the study. The Panchayath is the lowest level of administrative system below the CD block. Each Panchayath is further divided into a number of wards ranging from 8-15. Trivandrum district was selected for the study because the researcher is a native of this district and is familiar to the study area. Other factors like feasibility of doing the study within a limited time period and financial resources, also influenced the selection of the district.

People in the 30-50 year age group were selected because they were in the prime working age group, more mobile and exposed to several risk factors such as tobacco chewing, smoking and alcohol consumption.

Sample size

The sample size calculation was based on the findings of a previous study conducted in a selected population in urban Trivandrum district, which assessed the periodontal conditions of a selected population in the 35-44-age group.[20] This study had shown that around 75% of the population had periodontal pockets. Based on similar prevalence rate, a minimum sample size required was 288 and assuming a design effect of 1.5 for cluster sampling, the minimum sample size required was estimated as 434. For this, one cluster was taken from each ward of a Panchayath and each cluster consisted of 7-10 people depending on the number of wards in each Panchayath. In certain wards there were houses with 3 or 4 people in the 30-50 age group, and all of them were included in the study. A total of 66 wards (clusters) and 504 individuals were included in the study.

Tools for data collection

Verbal informed consent was obtained from subjects and data collection was done through a community based cross sectional survey with the help of a pre-tested structured questionnaire, followed by clinical examination of the oral cavity by one of the investigators (RJ). Besides general information like age, sex, occupation, education and income of the subject, the questionnaire was designed to collect information concerning the use of smokeless tobacco, other forms of tobacco, alcohol consumption, past dental history and oral hygiene habits. The participants were classified into four groups-those who consume both forms of tobacco (smoking and chewing), smokers alone, chewers alone and non-users of tobacco. Smokers were defined as those who had smoked any tobacco product at the time of survey. The same definition was applied to define tobacco chewers and alcohol users.

We further categorized participants into three groups based on their level of education. The first group comprised of illiterates and those up to 5 years of schooling, the second group between 5 and 10 years of schooling and third group having more than 10 years of schooling. Occupation of the individual was graded on the basis of type of work the individual was handling and also the wage pattern of the individual.

Socio-economic status was assessed on the basis of household income, considering the inflow from all possible sources of income in the household. Oral hygiene habits of each individual was assessed on the basis of method of cleaning, frequency of cleaning and the materials used for cleaning the oral cavity. For clinical examination we used mouth mirrors, Community Periodontal Index of Treatment Needs (CPITN) probe and cidex solution for sterilization.

Case Definition

In this study, periodontal disease was diagnosed on the basis of CPITN index developed by the WHO/FDI which describes the prevalence of periodontal conditions and as a screening test to identify patients who need complex or simple treatment.[22-24] The index assesses the periodontal condition of a subject based on the following periodontal disease markers namely:[25]

Code 0. Healthy
Code 1. Bleeding observed, directly or by using a mouth mirror, after probing
Code 2. Calculus detected during probing, but all of the black band on the probe visible.
Code 3. Pocket 4-5 mm (gingival margin within the black band on the probe)
Code 4. Pocket 6 mm or more (black band on the probe not visible)

According to the index criteria set, subjects falling under code 3 category or code 4 category were considered as having periodontal disease.[26]

Intra oral examinations were conducted by the principal investigator (RJ), who was assisted by a dental hygienist student in all subjects to reduce inter-observer variations. Each subject was examined on an ordinary chair in adequate natural light using a mouth mirror and CPITN probe.

Since only one investigator (RJ) was involved in the assessment of periodontal disease, there was no intra-observer variation in this study. The single observer being a trained and experienced person, intra-observer variation was expected to be minimum, although not formally assessed in this study.

Statistical analysis

Statistical analysis of the study was done using SPSS version 6-software program. Chi-square test and in certain cases Chi-square trend test was used. Multiple logistic regression analysis was used to assess the association of select risk factors with prevalence of periodontal disease adjusting for other predictor variables. The predictor variables were age, tobacco consumption habits, alcohol consumption, education level, oral hygiene habits and socio-economic status. A p-value of 0.05 or less was taken to indicate a statistically significant association.

RESULTS

Study Sample Characteristics

The total study sample consisted of 290 males and 214 females in the age group 30-50 years (mean age 39.9±7.1 years). Participants were categorized into four groups according to their age groups i.e. 30-34 years, 35-39 years, 40-44 years and 45-50 years. In certain houses women were alone and they expressed their unwillingness to participate in the study and that resulted in lower percentage of female participants in the study. Half of the population in the study sample was in the lowest household income group and nearly one fourth of the total sample population was found to be tobacco smokers. Majority of the subjects used toothpaste and toothbrush for cleaning their teeth and three fourths of them cleaned their teeth once in a day. (Table 1)

Prevalence of Periodontal disease

The overall prevalence of periodontal disease in the study population was estimated to be 65.3% (95% CI 61.1-69.4). Deep periodontal pockets (code 4) were found in 18% and shallow periodontal pockets (code 3) were found in 47.3% of the total sample population (Table 2).

A significant difference was observed in the prevalence of periodontal disease between men (70%; 95%CI 64.7- 75.3) and women (58.9%; 95%CI 52.3-65.5). With reference to socioeconomic status (SES) as assessed by household income, we found that 74.5% of the participants belonging to the lowest SES group were affected by periodontal disease ($p<0.001$), compared to the highest SES group.

A larger proportion of the subjects used toothpaste and toothbrush while one fourths of the subjects relied on charcoal and finger for cleaning the oral cavity. It was found that periodontal disease was more prevalent among finger and charcoal users in the study sample (Table 3).

When comparing the four different age groups, a stepwise increase in the prevalence of periodontal disease was noted. The prevalence of periodontal disease had increased with age (Table 4).

In the analysis of the association between tobacco consumption pattern and periodontal disease, prevalence of periodontal disease was highest among those who used both forms of tobacco (smoking and chewing) followed by smokers alone and chewers alone; providing a gradient between non-users of tobacco and those who used both forms of tobacco (Table 4).

Multivariate analysis

The results of multivariate analysis are presented in Table 5. Periodontal disease prevalence had increased with increasing age. It was observed that 45-50 age group had 5.3 fold risk of prevalence of periodontal disease compared to 30-34 year age group. The other variable which had a striking influence in the prevalence of periodontal disease, was tobacco smoking. We observed that smokers alone had 2.6 times risk of getting periodontal disease when compared to non-users of tobacco.

Treatment needs of the population

When considering the treatment needs of the population it should be noted that healthy periodontium was not observed in any of the subjects (Table 6). The WHO Global Oral Health Data Bank also highlights the fact that subjects with healthy periodontal tissues in the 35-44 year age group was found to be very minimal in India and Bangladesh.[27] Though we considered CPITN 3 (shallow pockets) and CPITN 4 (deep pockets) only as diseased periodontium in our study, CPITN 2 also requires treatment in the form of scaling and oral hygiene instructions. In general, though both smoking and chewing has a detrimental effect on the periodontium, the severity of periodontal destruction with respect to periodontal pocketing was more among smokers than in chewers.

Table 1. Study sample characteristics

Variable	Total sample (N=504)	Female (N=290)	Male (N=214)
Age group(yrs)			
30-34	146	77(52.7)	69(47.3)
35-39	94	65(69.1)	29(30.9)
40-44	95	60(63.2)	35(37.2)
45-50	169	88(52.0)	81(48.0)
Education			
Up to primary school (<5yrs)	184	92(50.0)	92(50.0)
High school (5-10yrs)	221	144(65.1)	77(34.8)
College (>10yrs)	99	54(54.5)	45(45.5)
House hold income/month			
<2000 Rs	282	158(56.0)	124(44.0)
2000-5000 Rs	114	57(50.0)	57(50.0)
>5000 Rs	108	75(69.4)	33(30.6)
Tobacco users			
Smokers and chewers	80	77(96.2)	3(3.8)
Smokers alone	112	112(100.0)	0 (0.0)
Chewers alone	66	16(24.2)	50(75.8)
Non users	246	85(34.6)	161(65.4)
Alcohol users			
Users	121	120(99.17)	1(0.83)
Non users	383	170(44.3)	213(55.7)
Oral hygiene habits			
1. Materials used			
Charcoal &others	129	74(57.4)	55(42.6)
Tooth paste users	375	216(57.6)	159(42.4)
2. Methods Used			
Finger	131	72(55.0)	59(45.0)
Tooth brush	373	218(58.4)	155(41.6)
Frequency of cleaning			
Once	394	243(61.7)	151(38.3)
Twice	110	47(42.7)	63(57.3)

*Figures in parenthesis are percentages

Table 2. Prevalence of Periodontal disease

Disease	Males	Females	Total
Periodontal Disease	70.0(64.4, 75.2)	58.9(52.0, 65.5)	65.3(60.9, 69.4)
Deep pockets	19.3(14.9, 24.3)	16.4(11.7, 22.0)	18.1(14.8, 21.7)
ShallowPockets	50.7(44.8, 56.6)	42.5(35.8, 49.5)	47.2(42.8, 51.7)

*Figures in parenthesis are 95% confidence intervals

Table 3. Correlates of Periodontal disease: Results of bivariate analyses

Variable	Total sample	Disease present	P value
Sex			
Males	290	203 (70)	
Females	214	126 (59)	<0.001·
House hold income (Rs)			
<2000	282	210 (74.5)	
2000-5000	114	61 (53.5)	<0.001·
>5000	108	58 (53.7)	
Oral hygiene habits			
Method of cleaning	Finger-131	106 (80.9)	
	Tooth brush-373	223 (59.8)	<0.001·
Materials for cleaning	Charcoal & Others -129	104 (85.7)	<0.001·
	Tooth paste-375	225 (60)	
Frequency of cleaning/day	Once -394	273 (69.3)	
	Twice-110	56 (50.9)	<0.001·
Alcohol users			
Users	121	95 (78.5)	
Non users	383	234 (61.1)	<0.001·

·Chi-square test

Table 4: Prevalence of periodontal disease in relation to different age groups and tobacco consumption habits

Age group (years)	Total sample (n=504)	Disease present (n=329)	Odds ratio	P value
30-34	146	66 (45.2)	1.00	
35-39	94	57 (60.6)	1.87	<0.001·
40-44	95	70 (73.6)	3.89	
45-50	169	136 (80.4)	5.00	
Tobacco users	**Total sample**	**Disease present (n=329)**	**Odds ratio**	**P value**
Non users of tobacco	246	135 (54.8)	1	<0.001·
Chewers alone	66	42 (63.6)	1.44	
Smokers alone	112	88 (78.5)	3.01	
Using both forms	80	64 (80)	3.29	

*Chi-square trend test
Figures in parenthesis are percentages

Table 5. Multivariate logistic regression results of periodontal disease on select risk factors

Variables	Referent category	Odds ratio	95% CI	p-value
Age group				
40-44 years	30-34 years	3.3	(1.58, 5.54)	<0.001
45-50 years		5.3	(3.01, 9.96)	<0.001
Tobacco use				
Smokers	Non users	2.65	(1.29, 5.41)	0.006

Variables considered in the logistic model and found not significant were sex, education status, household income, oral hygiene habits and alcohol consumption.

Table 6. Percentage distribution of dentate subjects in different age groups by highest CPITN code number recorded, Periodontal status (partial mouth recording)

Age (yr.)	No. of dentate Subjects	CPITN code number recorded				
		No Disease (0)	Bleeding only (1)	Calculus (2)	Shallow pockets(3)	Deep Pockets(4)
30-34	146	0	6.2	48.6	40.4	4.8
35-44	189	0	3.7	29.1	47.6	19.6
45-50	169	0	0	19.5	52.1	28.4

DISCUSSION

It was found that prevalence of periodontal disease increases in the middle years of life, which is the major reason for teeth loss in adults. Tobacco consumption has got substantial influence on the periodontal health and disease of a society and smoking is one of the most important risk factor for periodontal disease. Even though the pathogenesis of periodontitis in smokers is incompletely understood, epidemiological data indicate a direct effect. The gradual worsening of the periodontal conditions with increasing age reflected the chronic progressive nature of periodontal disease.[20, 28]

Our study in Kerala found that tobacco chewing particularly in the form of betel quid (pan) chewing is very common in rural areas and that the overall picture of periodontal diseases collected from 6 Panchayaths in Trivandrum district resembled the CPITN data obtained from other parts of the world. We also found that age, gender, tobacco consumption habits, oral hygiene habits, alcohol, education and socioeconomic status having a direct bearing on the prevalence of periodontal disease. Compared to non-users of tobacco, prevalence was higher among chewers and highest among those who consumed both forms of tobacco.

It was found that tobacco smoking and age were associated with periodontal disease. Although prior studies had not been conducted in rural Kerala, but when compared to a previous study conducted in a selected population in urban Trivandrum district in 1990,[20] we found that the disease prevalence was less in our study with reference to the 35-44 year age group category (67.2% when compared to 77% in the urban population). Perhaps this may be due to the improvement in socioeconomic status in Kerala since 1990,[29] which had its impact on lowering the periodontal disease prevalence.

Based on the WHO Global Oral Health Data Bank, the periodontal disease status with reference to periodontal pockets in the 35-44 year age group was found to be around 60% in India and Bangladesh in the year 1989-1990.[27] Similarly, our study had shown 67.2% prevalence of periodontal disease in rural Kerala in this age group (Table 5). The periodontal disease prevalence in our study indicates that, the entire population needs oral hygiene instructions, over a half needs scaling and 18.1% needs complex periodontal treatment. In spite of the remarkable achievements in the health sector, it was seen that oral health care was not given much importance by the adult population in rural Trivandrum.

CONCLUSIONS

In our community based study, around two thirds of the adult population had periodontal disease. The disease prevalence was more among older age groups when compared to younger age groups. The emergence of smoking as a risk factor for periodontal disease should be dealt with utmost priority. Educational efforts targeted to avoid smoking in non-smokers and to quit smoking among smokers should be considered at the community level and dental checkups should be introduced at an early age and continuously done considering the progressive nature of the disease with age.

Policy implications

The study highlights the poor state of periodontal health in rural Trivandrum. This may be related to lack of proper dental health knowledge among rural population, expensive treatment procedures and lack of sufficient preventive and therapeutic dental services. Dental treatment procedures are inherently expensive and as a result of this, many communities are not able to afford even essential treatment procedures. In order to overcome this problem, the provision of dental care should be extended to rural population through the network of primary health centres. Educational efforts at national and local levels are needed to focus both public and professional attention on the problem of periodontal diseases and its association with tobacco consumption. An obvious implication is that smoking prevention and giving-up smoking among smokers should be included in dental public health education program for the purpose of preventing periodontal disease. Considering the progressive nature of the disease dental check ups should be started at an early age.

ACKNOWLEDGEMENTS

We are thankful to Dr. K.Nandakumar, Head of the Department, Dental College Trivandrum for his technical assistance in formulating this project. We are also indebted to Mr. S. Nasiruddin, dental hygienist for assisting in clinical examination of the participants and the officials of the Panchayaths in lending their co-operation in overcoming the challenges of the field survey.

REFERENCES

1. Burt BA, Ismail AI, Eklund SA. Periodontal disease, tooth loss and oral hygiene among older Americans. Community Dent Oral Epidemiol. 1985;13:93-96.
2. Arbes SJ Jr, Slade GD, Beck JD. Association between extent of periodontal attachment loss and self reported history of heart attack: an analysis of NHANES III data. J Dent Res. 1999;78:1777-82.
3. Beck J, Garcia R, Heiss G, Vokonas PS, Offenbacher S. Periodontal disease and Cardiovascular disease. J Periodontol. 1996;67:1123-37.
4. Joshipura KJ, Rimm EB, Douglass CW, Trichopoulos D, Ascherio A, Willett WC. Poor Oral health and coronary heart disease. J Dent Res. 1996;75:1631-36.
5. Semmelweis Orvostudomanyi Egyetem, Parodontologiai Klinika, Budapet. The effect of smoking on the spread and frequency of periodontal disease. Forgov SZ. 1999;92:99-110.
6. Johnson NW, Griffiths GS, Wilton JMA, Maiden MFJ, Curtis MA, Gillett IR, Wilson DT, Sterne JAC. Detection of high risk groups and individuals for periodontal diseases: Evidence for the existence of high risk groups and approaches to their detection. J Clin Periodontol. 1988;15:276-90.
7. Sheiham A. Public Health aspects of periodontal disease in Europe. J Clin Periodontol. 1991;18:362-69.
8. Tobaco or Health: A Global status report. World Health Organisation Geneva. 1997:1-3.
9. Weinstein RL, Francetti L, Maggiore E, Marchesi G. Alcohol and smoking. The risk factors for the oral cavity. Minerva Stomatol. 1996;45:405-13.
10. Bergstorm J, Eliasson S, Dock J. A 10 year prospective study of tobacco smoking and periodontal health. J Periodontol. 2000;71:1338-47.
11. Machuca G, Rosales I, Lacalle JR, Machuca C, Bullon P. Effect of cigarette smoking on periodontal status of healthy young adults. J Periodontol. 2000;71:73-78.
12. Qandil R, Sandhu HS, Matthews DC. Tobacco smoking and periodontal disease. J Can Dent Assoc. 1997;63:187-92, 194-95.
13. Arbes SJ Jr, Agustsdottir H, Slade GD. Environmental tobacco smoke and periodontal disease in the United States. Am J Public Health. 2001;91:253-57.
14. Offenbacher S, Weathers DR. Effects of smokeless tobacco on the periodontal, mucosal and caries status of adolescent males. J Oral Pathol. 1985;14:169-81.
15. Robertson PB, Walsh M, Greene J, Ernster V, Grady D, Hauck W. Periodontal effects associated with the use of smokeless tobacco. J Periodontol. 1990;61:438-43.
16. Bhonsle RB, Murti PR, Gupta PC. Tobaco habits in India. In: Prakash C Gupta, James E Hamner III and PR Murti (eds). Proceedings of an international symposium on control of tobacco related Cancers and other Diseases Jan 15-19, 1990, Tata Institute of Fundamental Research, Mumbai. Oxford University Press, Mumbai. 1992:25-46.
17. Census of India 2001, Provisional Population Total: India-Part-I, Ministry of Home affairs, Government of India, New Delhi.
18. Zachariah KC, Irudaya Rajan S, Sarma PS, Navaneetham K, Gopinathan Nair PS, Misra US. Geographic, Socio-Economic and Demographic Back Ground: Kerala state and Sample Districts. In: Demographic Transition in Kerala in the 1980's, Centre for Development Studies, Thiruvananthapuram. 1994:9-38.
19. Vaidya GS, Naik DU. Tobacco addiction in India. Tob Control. 1995;1:194-205.
20. Anil S, Hari S, Vijayakumar T. Periodontal Conditions of a selected population in Trivandrum district, Kerala, India. Community Dent Oral Epidemiol. 1990;18:325.
21. Spolsky Vladimir. The epidemiology of gingival and periodontal disease. In: Carranza Jr (ed). Glickman's Clinical Periodontology. (7th ed). W.B. Saunders company, 1990:312-13.
22. Cutress TW, Hunter PB, Hoskins DI. Comparison of the Periodontal Index (PI) and Community Periodontal Index of Treatment needs (CPITN). Community Dent Oral Epidemiol. 1986;1:39-42.
23. Baelum V, Papapanou PN. CPITN and the epidemiology of periodontal disease. Community Dent Oral Epidemiol. 1996;24:367-68.
24. Croxson LJ. A simplified periodontal screening examination. The Community Periodontal Index of Treatment Needs (WHO) in general practice. Int Dent J. 1984;34:28-34.
25. World Health Organization. Oral Health Surveys: basic methods. World Health Organisation Geneva (4th ed) 1997;36-38.
26. Gamonal JA, Lopez NJ, Aranda W. Periodontal conditions and Treatment needs by CPITN in the 35-44 and 65-74 year old population in Santiago, Chilie. Int Dent J. 1998;48: 96-103.
27. Observed Periodontal Conditions measured by CPITN at age 35-44 years. SEARO (WHO Global Oral Data Bank, 1 January 1994). WHO Oral Health Country Profile Programme. Division of non-communicable Diseases/Oral Health. School of Dentistry, Nigate University, Japan. http://www.dent.niigata-u.ac.jp.
28. Gaengler P, Goebel G, Kurbad A, Kosa W. Assessment of Periodontal disease and dental caries in a population survey using the CPITN, GPM/T and DMF/T indices, Community Dent Oral Epidemiol. 1988;16:236-39.
29. Aravindan KP, Kunhikannan TP. Health transition in rural Kerala 1987-1996. Kerala Sastra Sahithya Parishad, Chalappuram, Kozhikode, Kerala, India 2000:17-19.

Address for corresponde:

Dr. R. Jayakrishnan
Lecturer,
Division of Community Oncology,
Regional Cancer Centre,
Trivandrum, Kerala, India
Tel : 91-471-2522 2299
Email : jayrish71@yahoo.com

Malaysian Dental Journal (2005) 26(2) 105-107

MALAYSIAN DENTAL JOURNAL

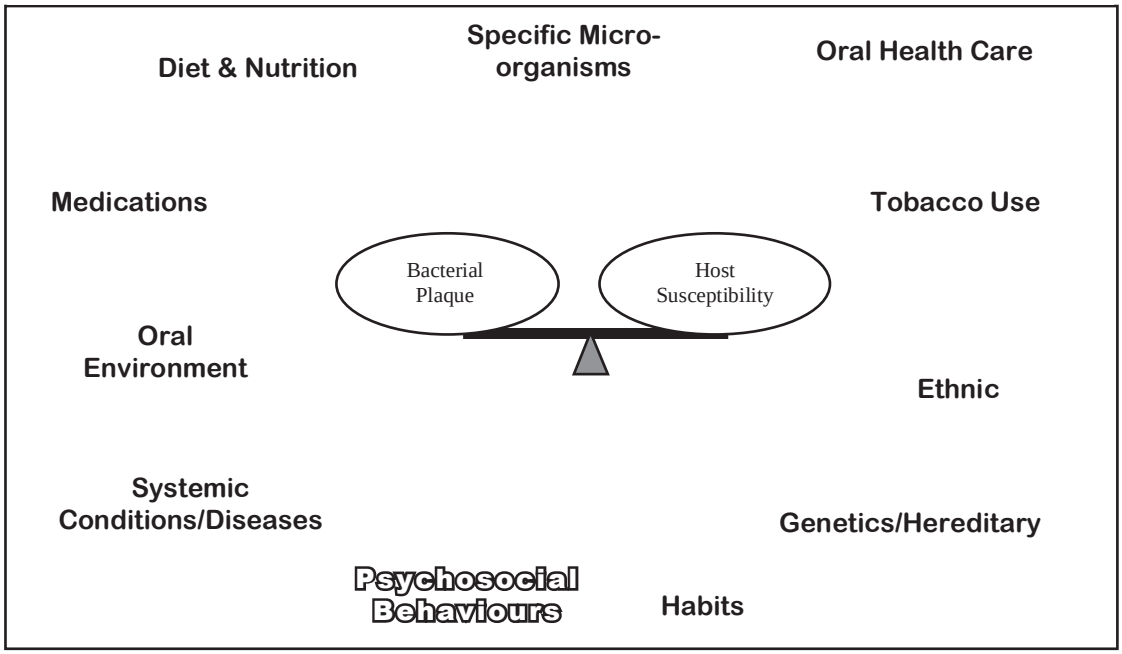
The Expert Says …

Prevalence of Periodontal Disease In Malaysian Population

Mohd Said S. *Lecturer, Department of Periodontology, Faculty of Dentistry, UKM, Kuala Lumpur, Malaysia.*

For many decades, periodontal disease is one of the commonest oral problems reported worldwide. Although the aetiology of periodontology are mainly bacterial plaque and host susceptibility to the disease, there are many factors that contribute to its occurrence and progression. These include specific micro-organisms for example most commonly *Actinobacillus actinomycetemcomitans, Porphoromonas gingivalis, Prevotella intermedia;* poor oral health care by individuals, smoking status, genetic influence, particular habits such as bruxism, stress, distress and poor coping behaviours, particular systemic diseases and conditions such as diabetes mellitus, hormonal influence, specific medications, diet and malnutrition. These factors, singly or in combination, influence both the aetiological components either directly or indirectly and causing imbalance between them. Thus explain why periodontal disease still remains as one of the major oral diseases and is difficult to manage.

Diagram 1: Aetiology and contributing factors to periodontal disease.



Malaysia, a country located in the south-east of Asia, has a total population of 26.64 million. The country has a multiracial population living together in harmony. This unique feature of the country contributes greatly to the diversity of culture and practice of its population. Peninsular Malaysia, consists mainly of indigenous group including the Malays (65.1%), followed by the Chinese (26.0%) and Indians (7.7%), while in East Malaysia i.e. Sabah and Sarawak, the majority population is made up of the Ibans (30.1%), Kadazan Dusun (18.4%), Chinese (26.7%) and other minority indigenous groups such as the Bajaus and Malays. The proportion of population of Malaysia below 15 years old in 2000 was 33.3% while those 65 years and above was reported as 3.9%. Median age for Malaysians is 23.6 years.

In 2000, a National Oral Health Survey for Adults (NOHSA) was carried out in Malaysia utilizing the Community Periodontal Index (CPI) score and the highest score of each subject. It showed that out of the total 9.7 million individuals screened, only 9.8% have healthy gingiva while the remaining had periodontal problems of various severity including gingivitis (4.5%), shallow probing pockets of 4-5mm (20%) and those with deeper probing pockets (5.2%). Half of the population had calculus in their mouth.

The survey also showed that Malaysian males had more incidence of periodontal tissue breakdown when compared to females. More females experience gingivitis though, and healthy gingiva. The difference between these percentages can perhaps be explained by influence of various contributing factors as mentioned and illustrated in the diagram above.

Table 1: Estimated Percentage of Dentate Population With Highest Recorded Community Periodontal Index (CPI) Score in 2000. (National Oral Health Survey for Adults 2000)

Variables	N	Healthy (0)	Bleeding on Probing (1)	Calculus (2)	Shallow Pockets, 4-5mm (3)	Deep Pockets, >6mm (4)	Excluded sextants (x)
All	9,661,565	9.8	4.5	57.5	20.0	5.2	3.0
Gender:							
Male	4,376,028	7.9	3.7	56.0	22.5	7.0	2.9
Female	5,285,534	11.4	5.2	58.8.	17.9	3.6	3.1
Age Group:							
15-19	1,553,415	25.8	11.2	60.0	2.9	0.1	0.0
20-24	1,074,073	15.6	6.5	69.5	7.5	0.8	0.0
25-29	945,677	9.2	4.7	69.1	15.2	1.5	0.4
30-34	1,059,338	7.0	3.8	63.5	21.5	4.0	0.3
35-44	2,186,492	5.0	2.8	54.9	28.5	7.2	1.7
45-54	1,544,320	4.3	2.1	51.1	27.9	10.2	4.4
55-64	806,704	3.9	1.3	44.6	31.6	9.5	9.1
65-74	379,810	2.6	1.5	40.7	27.1	9.2	19.0
75+	111,736	1.2	0.9	45.4	19.8	5.9	26.8
Ethnic Group:							
Malay	5,087,557	9.0	3.4	57.8	21.4	5.6	2.7
Chinese	2,576,510	12.0	6.9	56.7	18.1	3.9	2.4
Indian/Pakistani	875,432	11.5	5.1	52.8	21.4	7.1	2.2
Kadazan	195,964	1.4	6.0	63.3	17.5	5.9	5.8
Iban	326,740	12.0	3.0	54.8	19.0	5.9	5.3
Other Indigenous Group	509,740	6.2	3.0	64.1	16.1	3.8	6.8
Others	89,687	5.6	3.1	72.4	12.3	1.3	5.4

When observed across age groups, the prevalence of periodontal disease seems to increase with increasing of age. However, it cannot be concluded that prevalence of the disease progresses with increasing age, but it is seen as a cumulative effect of the disease with increasing age.

Earlier literature in the 1960s, in which Periodontology is a relatively a new subject in dentistry, similar inference was made as such that *'periodontal disease is a major global problem affecting the majority of adults after the age of 35-40 years and that gingivitis if not treated, will progress to destructive periodontitis as the subjects aged and with impairment of oral hygiene measures'* (reviewed by Scherp 1964). With development of new knowledge as well as research findings, we now know that in presence of bacterial plaque, individual susceptibility to periodontal disease determines the occurrence and progression of periodontal tissue breakdown. Series of studies by Löe and co-workers involving the Sri Lankan and Swedish population, told us that gingivitis do not necessarily progress to periodontitis, even with increasing age and different oral hygiene care and status. Also, as indicated by Douglass and Fox (1993), *prevalence of periodontal disease should be expected to increase, rather than decrease, as long as the disease is measured by means of cumulative clinical attachment loss*.

The Malaysian Indians appear to have the highest proportion of deep probing pocket depths of more than 6mm (7.1%), followed by the Kadazans and Ibans (5.9% each) and Malays (5.6%), while Chinese has the least (3.9%). Malaysian population due to its diversity in ethnicity and has various cultural lifestyle and practices. Among others are betel-nut chewing, particular smoking habits and diets. Although these are well-known fact, strong evidence of their influence on the occurrence and progression of periodontal disease has yet to be studied.

As a conclusion, the prevalence of periodontal disease among Malaysian population is 5.2%, which is lesser than those reported worldwide (10-15% deep pockets of >6mm). With better understanding of the nature of the disease, clinicians and researchers can look closer at the contributing factors as well as individual risk of the patients. Periodontal disease will perhaps remains as one of the major global oral health problems in this millennium as we find more and better approaches to prevent and treat the disease.

REFERENCES

1. Population Statistic 2006. Department of Statistics Malaysia. www.statistics.gov.my
2. Population Distribution and Basic Demographic Characteristics Report - Population & Housing Census 2000. November 2001. Department of Statistics Malaysia
3. National Oral Health Survey of Adults (NOHSA) 2000. Oral Health Division, Ministry of Health. November 2004.
4. Scherp HW. Current Concepts In Periodontal Disease Research: Epidemiological Contributions. J Am Dent Assoc. 1964;68:667-675.
5. Anerud A, Löe H, Boysen H, Smith M. The Natural History of Periodontal Disease In Man. Changes In Gingival Health and Oral Hygiene Before 40 Years of Age. J Periodontal Res. 1979;14:526-540.
6. Hujoel PP, Löe H, Anerud A, Boysen H, Leroux BG. The Infromativeness of Attachment Loss On Tooth Mortality. J Periodontol. 1999;70:44-48.
7. Douglass C.W., Fox C.H. Cross-sectional Studies In Periodontal Disease: Current Status and Implications of Different Index Systems. J Clinical Periodontol. 1993;20: 656-661.

Author:

Dr Shahida Mohd Said,
Lecturer,
Department of Periodontology,
Faculty of Dentistry,
Universiti Kebangsaan Malaysia,
Jalan Raja Muda Abdul Aziz,
50300 Kuala Lumpur, Malaysia.
Tel : +603 4040 5764 / 5745
Fax : +603 4040 5797
Email : shahidasaid@hotmail.com

Malaysian Dental Journal (2005) 26(2) 108-115

MALAYSIAN DENTAL JOURNAL

Smoking Cessation for Adolescents: Opinions and Experience of Malaysian Government Dental Practitioners

Dr Yahya NA BDS (Malaya) MSc (London) DDPHRCS (Eng) *Lecturer, Dental Public Health Department, Dental Faculty, UKM, Malaysia.*

Croucher RE BSc MA PhD *Professor in Community Oral Health/Senior Tutor, Dental Postgraduate Taught Courses, Institute of Dentistry, Barts and The London, Queen Marry's School of Medicine and Dentistry, Queen Mary, University of London, England.*

ABSTRACT

AIMS: To assess the nature and extent of adolescent smoking cessation support and identify barriers for tobacco prevention amongst the government dental practitioners of the Ministry of Health and Ministry of Education serving in Kuala Lumpur, Malaysia. OBJECTIVE: This study therefore investigated the awareness of government dental practitioners in the Federal Territory of Kuala Lumpur, Malaysia on their role in adolescent smoking cessation. MATERIALS AND METHODS: A self-administered questionnaire was mailed to 125 government dental practitioners in the Ministry of Health and the Ministry of Education (University of Malaya and Universiti Kebangsaan Malaysia). RESULTS: The overall response rate was 63%. Only 26.4% of the dentists routinely record the smoking status of adolescent patients in their clinical practice. Almost 96% of the dentists believe that the dental team should encourage adolescent patients to stop smoking and slightly more than half believe the dental team is effective in helping adolescents to stop smoking. Lack of knowledge on the subject of smoking cessation in general was the most frequently reported barrier to discussing smoking with adolescent patients aged between thirteen and sixteen years old (54.2%). Almost 85% perceived that discussing an adolescent's smoking habits is part of their job. Only fifteen respondents (20.8%) had assisted an adolescent patient aged between thirteen to sixteen in smoking cessation, whilst nine dentists (12.5%) have taken part in other adolescent cessation activity. While a majority of dentists (69.4%) reported having actually read some information on smoking cessation, only 2.8% of dentists reported having taken a course on smoking cessation. CONCLUSION: While most dentists do believe that they have a role in ad lescent smoking cessation and are convinced that smoking is a major health issue, they are not confident of their ability to practice it.

Key words:
Smoking cessation, adolescents, survey

INTRODUCTION

Smoking is a significant public health problem world wide. Tobacco smoking is the single most important cause of preventable ill-health.[1] The most common smoking related diseases are lung and oropharyngeal cancer, cardiovascular diseases, chronic airway diseases and cancers of other sites such as the oesophagus, pancreas and bladder.[2] In addition to being implicated in coronary heart disease, lung cancer and other cancers, smoking has profound effects in the mouth. Smoking is estimated to account for 92 percent of cancers of the lip, oral cavity and pharynx, and maternal smoking has been associated with an increased risk of cleft palate in infants.[3] A causal relationship between smoking and periodontitis has been recognized by the United States Surgeon General.[4] Damage to periodontal tissues is significantly greater in smokers than in non-smokers. Refractory periodontitis has been shown to occur almost exclusively among current smokers and smoking cessation is recommended prior to periodontal treatment.[5] Since smoking was not identified as one of the main risk factors for chronic periodontitis, for most of the 20th century, a distorted understanding of periodontitis resulted.[6] Stained teeth, halitosis and soft tissue changes in the mouth may be especially pertinent to young people as strong motivators to quit smoking.[7]

At least one in three teen-age smokers will die prematurely as a result of smoking. Growing evidence indicates that children are smoking more and starting to smoke at younger age. The Second National Health and Morbidity Survey in Malaysia in 1996 found a prevalence of smoking among teenagers aged 12 to 18 years of 30.7% for males and 4.8% for females. Because most smokers begin using tobacco during adolescence, preventing and reducing tobacco use among youth has substantial long term health benefits to society.[8] Survey results indicate an increase in smoking incidence beginning at age 11 and peaking at age 18. However, if adolescent tobacco use is prevented, it is unlikely to be initiated in adulthood.[9]

Dentists are well placed to recognise smokers and dentists can identify the impact of tobacco use in the mouth.[10] Dentists are increasingly being urged to use their contacts with the public to support tobacco cessation policies and prevent the uptake of tobacco habits by those

not yet habituated, particularly children and adolescents.[11] Despite this, there has been little attempt to study the role of the dentist in smoking cessation for adolescents. This is of special importance for a country such as Malaysia. The World Tobacco Marketfile describes teenage smoking in Malaysia as "an acute problem", stating that approximately 12% of boys and 2% of girls smoke, up from 10% of boys and 1% of girls in 1985.[12] This is much lower however than other studies which have found smoking prevalence rates amongst Malaysian youth ranging from 30% to 60%. Experimentation rates are extremely high; a study by the Ministry of Youth and Sports, conducted in 1994, suggested that 71% of young people had experimented with cigarettes.[13]

Zulkifli[14] argues that prevalence studies may even underestimate the real prevalence, as smoking is a disciplinary offence for school students. The highest smoking prevalence was found amongst 'lepak' youth (young people who are unemployed and/or not in school). A Ministry of Culture, Youth and Sports survey on this group found a smoking prevalence of 80%, which was much higher than in school students.[14]

This study therefore investigated the awareness of government dental practitioners in the Federal Territory of Kuala Lumpur, Malaysia on their role in adolescent smoking cessation.

MATERIALS AND METHODS

A self-administered mail questionnaire was used to carry out the survey. Permission to carry out this survey of Malaysian government dental practitioners in Kuala Lumpur was sought from the Oral Health Division Director, Ministry of Health Malaysia. Following confirmation of this support, a self-administered mail questionnaire was assembled. The questionnaire used was originally developed for a Swedish study.[15]

The questionnaire consisted of three sections with nineteen closed questions and two open ended questions. In the first section five questions were used to assess the practice of identifying adolescent smoking in the clinical setting and whether they believe the dental team should encourage adolescent patients to stop smoking and whether they are effective in doing so. 'Yes' or 'no' response options were available for the questions in this section.

The second section had seven questions designed to identify the perceived barriers to discussing smoking with adolescent patients aged thirteen to sixteen years. The potential suggested barriers were; lack of time, lack of knowledge on smoking cessation in adolescents and in general, not perceiving discussing an adolescent's smoking habit as part of the job, whether smoking is a major health issue, feeling comfortable to ask adolescents about their tobacco use and whether they would prefer to refer adolescents to therapist who specialise in smoking cessation.

In the third section there were four questions assessing smoking cessation consultation experiences with adolescents aged 13-16 years old. These questions offered the dichotomous response options of 'yes' or 'no'. One open-ended question was included regarding what information they would like to receive about smoking cessation.

The final section had five questions to ascertain the respondent's personal details, ie gender, year born, year of initial dental qualification, place of practice and smoking status.

After completion of all the questions, the respondents were asked to place the questionnaire in the reply envelope provided for collection.

The questionnaire was piloted on six government dental practitioners from the Ministry of Health and Ministry of Education (University of Malaya and Universiti Kebangsaan Malaysia) who were pursuing their postgraduate studies in London.

A letter introducing the study and assuring potential participants of confidentiality along with a self-administered mail questionnaire was delivered to the 125 government dental practitioners from the Ministry of Health and the Ministry of Education (University of Malaya and National University of Malaysia) in the Kuala Lumpur region who were in possession of a current Annual Practicing Certificate. The list of their names and practicing addresses was obtained from the Oral Health Division, Ministry of Health Malaysia.

The collection of the questionnaire was carried out between May-June 2003. Resource constraints prevented reminders being sent to those who did not initially respond to the questionnaire.

Data was entered for analysis using SPSS. Frequencies and percentages were used to determine the distribution of responses for each variable. Percentages for each variable were based on the number of respondents for each question. Associations were tested using the chi-squared test. A significance level of 0.05 was adopted.

RESULTS

Of 125 government dental practitioners identified, only 114 could be contacted and were given the questionnaire. Seventy-two out of 114 returned the questionnaire, an overall response rate of 63%. Details relating to the profile of respondents are presented in Table 1. More than half of the respondents (60.6%) were working in the university (University of Malaya and Universiti Kebangsaan Malaysia), while 38.0% were working with the Ministry of Health in the government clinics and hospitals (Table 1). One respondent did not state his/her place of practice.

Fifty-three respondents were female (73.6%) and nineteen (26.4%) were male. Half of the respondents were 36 years (50.7%) (Table 1). There was a statistical significant relationship between gender and age of the respondents with P value less than 0.05. However, there was no statistical significant relationship between gender and place of practice.

Table 1: Characteristics of the study population.*

	Male		Female		Total	
	N	%	N	%	N	%
Age (median 36.0)						
36 years or less	6	31.6	29	54.9	35	50.7
>36 years old	13	68.4	21	39.6	34	49.3
Years of working experience (median 12 years)						
Less than 12 years	6	31.6	27	50.9	33	47.8
12 years or more	13	68.4	23	43.4	36	52.2
Place of practice						
Government clinics/hospital	4	21.1	23	43.4	27	38.0
University	14	73.4	29	54.7	43	60.6
Others	1	5.3	0	0	1	1.4
Smoking status						
Smoker/ex-smoker	11	57.9	0	0	11	15.3
Non smoker	8	42.1	53	100	61	84.7

*Denominators vary as a result of missing information.

Thirty-three (47.8%) respondents had less than twelve years of working experience with six being males and twenty-seven being females. Meanwhile, thirty-six (52.2%) respondents had twelve years or more working experience with thirteen males and twenty-three were females. There was no statistical significance between the gender and working experience.

Eleven male dentists were either current or ex-smokers. Fifty-three respondents, mostly female, reported they had never smoked. These differences were statistically significant (Table 1).

Table 2 reports the respondents' practice of identifying adolescent smoking in their clinical routine. Only nineteen (26.4%) dentists routinely record the smoking status of adolescent patients in the clinical practice. Although it was not common practice to identify adolescent smokers, sixty-nine (95.8%) dentists believed that the dental team should encourage adolescent patients to stop smoking and slightly more than half believe the dental team is effective in helping adolescents to stop smoking (Table 2).

Twenty-seven (37.5%) of the dentists reported asking their adolescent patients whether or not she/he smokes even though they had no symptoms related to tobacco use compared to 90.3% of dentists questioning their adolescent patient when they have symptoms related to tobacco use (Table 2).

Table 3 shows the barriers for discussing smoking with adolescent patient. Five potential barriers were identified in the study as preventing discussion on smoking. Lack of knowledge on the subject of smoking cessation in general was the most frequently reported barrier for discussing smoking with adolescent patients aged between thirteen to sixteen years (54.2%). The respondents might have some knowledge of smoking cessation but dentists would prefer to refer to therapists who are specialised in smoking cessation, as reported by 55.6% of the respondents (Table 3).

56.9% of dentists did not perceive that discussion with adolescents about smoking cessation was too time consuming. Moreover, 84.7% perceived that discussing adolescent's smoking habits is part of their job and that a majority of dentists (76.4%) do feel comfortable asking adolescents about their tobacco use. This was probably due to the fact that 95.8% of them were convinced that smoking is a major health issue.

Table 4 showed the smoking cessation consultation experiences and education among the government dental practitioners. Only fifteen respondents had assisted an adolescent patient smoking cessation, while nine dentists (12.5%) have ever taken part in other adolescent cessation activities. While a majority of dentists (69.4%) reported having actually read some information on smoking cessation, only two dentists (2.8%) reported having taken a course on smoking cessation.

DISCUSSION

In this study there were more female respondents, reflecting the distribution of dental practitioners in Malaysia.71.6% of female dentists are practicing in the public sector.[16]

Most respondents reported that they supported a role in adolescent smoking cessation. Their actual practice was however, limited. Recording adolescent smoking status was unlikely. Most of them seem to be more likely to identify tobacco use routinely if the adolescent patients had tobacco related symptoms.

There are limited studies on adolescent smoking cessation activities by dentists to compare with theses findings. However, these findings are consistent with other studies on adult smoking cessation by dentists.[15] Only 26.4% of the respondents in this Malaysian study routinely record the smoking status of adolescent patients, which is slightly more than the findings by John et al. (1997) of dentists' practices in the UK Oxford region.[1] In Glasgow, 5.8% of the respondents reported always doing this and 8% of dentists in a Finnish study always inquired whether their patients smoked.[17]

In this study, female dentists working in the government clinic / hospitals were more likely not to record their adolescent patients' smoking status despite reporting that the dental team should encourage adolescents to stop smoking. Over 80% of female dentists reported that they did not routinely record their adolescent patients' smoking status. Male respondents were more likely to routinely record the smoking status of adolescent patients. Conversely, Klein et al (2001) reported higher rates of screening for regular smoking by female physicians than male physicians during routine adolescent visits for medical care.[8] Klein et al. (2001) also found that the physician's gender was not significantly associated with smoking screening and counselling behaviour.[8]

This study has identified that lack of knowledge was a key barrier for discussing smoking with adolescent patients. This led to more than half of the respondents preferring to refer the adolescent patient to a therapist who has specialized in smoking cessation even though the majority of them reported feeling comfortable asking adolescents about their tobacco use. Similar findings were also reported from other studies. Chestnutt and Binnie (1995) identified lack of time as a major hindrance to providing advice on smoking cessation.[17] A review of the results of studies among dental care providers regarding tobacco cessation in the past two decades reported that the most significant barrier remains a lack of education in dentists and hygienists on cessation activities during their years of training.[18] However, the availability of specialist tobacco cessation counselors has not been established.

Table 2: Dentist's practice of identifying adolescent smoking by gender.*

Questions		Male N (%)	Female N (%)	Total N (%)
Do you routinely record the smoking status of adolescent patient	**Yes**	9(47.4)	10(18.9)	19 (26.4)
	No	10(52.6)	43(81.1)	53 (73.6)
Do you believe the dental team should encourage adolescent patients to stop smoking	**Yes**	18(94.7)	51(96.2)	69 (95.8)
	No	1(5.3)	2(3.8)	3 (4.2)
Do you believe the dental team is effective in helping adolescent patients to stop smoking	**Yes**	12(63.2)	26(49.1)	38 (52.8 0
	No	6(31.6)	26(49.1)	32 (44.4)
Do you ask your adolescent patient whether or not she/he smokes even though they have no symptoms related to tobacco use	**Yes**	10(52.6)	17(32.1)	27 (37.5)
	No	9 (47.4)	36(67.9)	45 (62.5)
Do you ask your adolescent patient whether or not she/he smokes when they have symptoms related to tobacco use	**Yes**	16(84.2)	49(92.5)	65 (90.3)
	No	3(15.8)	4(7.5)	7 (9.7)

*Denominators vary as a result of missing information.

Table 3: Barriers for discussing smoking with adolescent patient aged 13-16 years old.*

QUESTIONS	RESPONSE OPTIONS	
	**SA/A N (%)	**NAND/D/SD N (%)
Discussions with adolescents about smoking cessation are too time consuming	29 (40.3)	41 (56.9)
I do not perceive discussing adolescent's smoking habits to be part of my job	8 (11.1)	61 (84.7)
I am not convinced that smoking is a major health issue	3 (4.2)	69 (95.8)
I feel comfortable asking adolescents about their tobacco use	55 (76.4)	17 (23.6)
I would prefer to refer adolescents to therapists who are specialised in smoking cessation	40 (55.6)	32 (44.4)
I feel I lack knowledge on the subject of smoking cessation in general	39 (54.2)	33 (45.8)
I feel that I have plenty of knowledge on the subject of smoking cessation in general	15(20.8)	57(79.2)

**SA-Strongly Agree; A-Agree; NAND-Neither Agree nor Disagree;D-Disagree; SD-Strongly Disagree.

Table 4: Smoking cessation consultation experiences and own education with adolescents aged 13-16 years old.*

QUESTIONS	RESPONSE OPTIONS	
	Yes N (%)	No N (%)
Have you ever assisted an adolescent patient in smoking cessation	15 (20.8)	57 (79.2)
Have you ever take part in other adolescent cessation activity, such as school campaigns	9 (12.5)	63 (87.5)
Have you taken a course in smoking cessation	2 (2.8)	70 (97.2)
Have you read any information on smoking cessation	50 (69.4)	22 (30.6)

*Denominators vary as a result of missing information.

This study shows that whilst most dentists do believe that they have a role in adolescent smoking cessation and are convinced that smoking is a major health issue, they are not confident of their ability to practice smoking cessation. These results are similar to another study by Shenkin et al[19] which reported that paediatric dentists consider tobacco cessation an important role in their practices, but do not consider themselves well prepared to shoulder this responsibility.

Smoking cessation consultation experiences and education activity is limited among Malaysian dentists. The majority of dentists have never assisted an adolescent patient in smoking cessation and never taken part in adolescent smoking cessation activities. Most dentists have not taken a course on smoking cessation but more than half have read some information on the subject. This suggests that, currently, courses in smoking cessation are not part of the undergraduate or postgraduate dental curriculum in Malaysia and are not integrated into the Continuous Professional Development programmes for qualified dentists. Alternatively, these courses may be available, but are not perceived to provide appropriate preparation.[20] Clear evidence is now available, from a meta-analysis of the literature, as to the appropriate content of educational and practice based strategies designed to increases the involvement of primary health care practitioners in the treatment of tobacco dependence.[21] Educational interventions for practitioners in training, combined with outreach practice-based support are proposed as an effective strategy.

There were several limitations in conducting this study. The survey sample was small and might not represent the entire population of dentists in Malaysia. The generalizability of this study is limited to dentists employed by the Ministry of Health and the Ministry of Education in the Federal Territory of Kuala Lumpur region, which is also the capital city of Malaysia. Other dentists from the Ministry of Defence and private practitioners were not included in the study. The list of dentists that were received from the Oral Health Division, Ministry of Health Malaysia was not current. Some of the respondents from the list could not be contacted due to changes of workplace, resignation or being on sabbatical leave. Coverage errors occur when mailing lists are incomplete, biased, inaccurate or out of date.[22]

The questionnaire was developed from previous studies conducted on issues in adult smoking cessation activities. Five barriers to smoking cessation activities were assessed in this study. There were more barriers reported from other studies than the five barriers that were used in this survey. These include lack of finance,[17] lack of insurance coverage[23] and fear of alienating patients.[24] The questionnaire that was adapted focused more on individual factors such as level of education.

There was limited time available for this study. Control of the distribution of the questionnaire was limited due to the geographic distance from where the questionnaire was sent. Follow-up of the questionnaire was not possible due to the limited time for distribution and collection of the questionnaire. It was not possible to distribute reminders to the respondents.

CONCLUSION

Smoking remains the most common preventable cause of death in the developing world. It is proposed that dentists should play a prominent role in promoting smoking cessation within their communities. A lack of education was the most significant barrier to the carrying out smoking cessation activities.

Further research with a larger national sample is recommended which could incorporate potential confounders that were not identified in this study.

REFERENCES

1. John J.H., Yudkin P., Murphy M., Ziebland S., Fowler G.H. Smoking cessation interventions for dental patients-attitudes and reported practices of dentists in the Oxford region. Br Dent J. 1997;183:359-64.
2. Kronebusch K. Smoking: human and economic costs. Cancer Inv. 1989;7:463-477.
3. Squier C. Introduction: Tobacco, human disease and the role of the dental profession. J Dent Assoc. 2001;65:303-5.
4. U.S. Department of Health and Human Services. The Health Consequencces of Smoking: A Report of the Surgeon General – Executive Summary. U.S. Department of Health and Human Services, Centres for Disease Control and Prevention, National Centre for Chronic Disease and Health Promotion, Office on Smoking and Health, 2004.
5. Johnson GK, Hill M. Cigarette smoking and the periodontal patient. J Peridontol. 2004;75:196-209.
6. Hujoel PP, del Aguila MA, DeRouen TA, Bergstrom J A hidden periodontitis epidemic during the 20th century? Community Dent Oral Epidemiol. 2003;31:1-6.
7. Watt RG, Johnson NW, Warnakulasuriya KA. Action on smoking-opportunities for the dental team. Br Dent J. 2000;189:357-60.
8. Klein JD, Levine LJ, Allan MJ. Delivery of smoking prevention and cessation services to adolescents. Archives of Pediatrics & Adolescent Medicine 2001;155:597-602.
9. Hovell MF, Jones JA, and Adams MA. The feasibility and efficacy of tobacco use prevention in orthodontics. J Dent Educ. 2001;65:348-53.
10. Monaghan N. What is the role of dentists in smoking cessation? Br Dent J. 2002;193:611-612.
11. Smith T.A., House R.F. Jr., Croghan I.T., et al. Nicotine patch therapy in adolescent smokers. Pediatrics 1996;98:659-667.
12. World Tobacco File [web page](2000): http://www.marketfile.com
13. The Star, January 4 1997.
14. Ahmad Z, Jaafar R, Musa R. Cigarette smoking among Malaysian youth: problems and prospects, Proceedings of the Malaysian Society of Health 21st Scientific Symposium, Kuala Lumpur 1997.

15. Helgason AR, Lund KE, Adolfsson J, Axelsson S. Tobacco prevention in Swedish dental care. Community Dent Oral Epidemiol. 2003;31:378-85.
16. Oral Health Division, Ministry of Health Malaysia. Oral Healthcare in Malaysia. MOH / K / GIG / 4.2001(BK) 2001.
17. Chesnutt IG, Binnie VI. Smoking cessation counselling-a role for the dental profession? Br Dent J. 1995;179:411-5.
18. Warnakulasuriya S. Effectiveness of tobacco counselling in the dental office. J Dent Educ. 2002;66:1079-87.
19. Shenkin D, Horowitz AM, Drury TF, Kanellis M. Attitudes of pediatric dentists towards tobacco intervention children and adolescents: a pilot survey. Pediatr Dent. 2003;25:53-60.
20. Fried J L, Reid B C, DeVore L E. A comparison of health professions student attitudes regarding tobacco curricula and interventionist roles. J Dent Educ. 2004;68:370-7.
21. Anderson P, Jane-Llopis E. How can we increase the involvement of primary health care in the treatment of tobacco dependence? A meta-analysis. Addiction 2004;99:299-312.
22. American Statistical Association (2000): What is a survey? : www.amstat.org/sections/srms/whatsurvey.html
23. Gerbert B, Coates T, Zahnd E, Richard RJ, Cummings SR. Dentists as smoking cessation counselors. J Am Dent Assoc. 1989;118:29-32.
24. O'Keefe J, Lessio A, Kassirer B. A pilot smoking cessation program involving dental offices in the Borough of East York, Ontario: an initial evaluation. J Can Dent Assoc. 1995;61:65-7.

Address for correspondence:

Dr Nurul Asyikin Yahya
BDS (Malaya) MSc (London) DDPHRCS (Eng)
Lecturer, Dental Public Health Department,
Dental Faculty, Tingkat 2, Blok Q,
Universiti Kebangsaan Malaysia,
Jalan Raja Muda Abdul Aziz
50300 Kuala Lumpur, Malaysia.
Tel : 603-4040 5854

Malaysian Dental Journal (2005) 26(2) 116-120

MALAYSIAN DENTAL JOURNAL

Average Outpatient Treatment Time by Dental Officers at the Malaysian Armed Forces

Hussin ZA BDSc (Qld) DPHD (Syd) *Health Branch, Army Field Headquarters, Ministry of Defence Malaysia.*

Tuti NMD BDS (Mal) MPH (Michigan) *Department of Dental Public Health, Faculty of Dentistry, UKM, Malaysia.*

ABSTRACT

Knowledge of time taken to provide treatment for dental patients may assist in the estimation of manpower requirements. As such, this study was carried out to find out the average time taken by dental officers of the Dental Services of the Malaysian Armed Forces (DSMAF) to attend to complaints of walk-in patients. Forms were distributed to seventeen dental officers of various bases throughout the country. These centers had comparable chairs, equipments and staff. A sample size of 1780 attendances comprising of 63.1% military personnel, 12.4% family members and 24.5% civilians was obtained. The longest average time taken by an operator is 25.4 minutes whilst the shortest, 9.4 minutes. Overall, the average treatment time is 16.6 minutes with a standard deviation of 10.3 minutes. There is no significant difference in treatment times between local (Malaysian) and foreign graduates. The average treatment time performed by operators who had worked longer appeared to be significantly shorter than those with less working experience.

***Key words*:**
treatment time, dentistry

INTRODUCTION

Knowledge of the time taken by dental surgeons in attending to their patients is useful in the planning of community-based dental service programmes. It gives an indication as to the number of patients that can be seen in a day. Together with data on disease prevalence, treatment needs and service utilization, it provides a quantitative avenue for estimating the amount of man-hours of work to be done[1] and consequently, manpower requirements. A dentist to population ratio can then be worked out based on the amount of work to be done and the ability of the dentist to undertake them.

The time taken by a dental surgeon to attend to his patients' complaints depends on numerous factors – among others, his training and experience, the procedure or procedures that he performs, the assistants who are with him, the equipments that he has and his practice habits and standards.

This study was conducted to find out the average time taken by dental officers of the Dental Service of the Malaysian Armed Forces (DSMAF) to attend to complaints of non-appointed walk-in patients. The type and number of procedures done on patients were to the operators' discretions, mainly guided by what they usually do in attending their daily sick reports.

This "treatment time" refers to the time from the moment the patient steps into the treatment room to the time he leaves it. It does not take into account the interval between dismissal of one patient to the entrance of the next. It does not seek to differentiate in a detailed manner the procedures performed as innumerous combinations are possible. No attempt is made to standardise or define procedures to be performed by these officers. They are to carry out their duties as they would do in their daily practices, based on respective complaints or cases presented. Outpatient treatment time was chosen as it is felt that after facing similar "situations" everyday dental officers will develop fairly consistent clinical habits and work pace most comfortable to them.

MATERIALS AND METHODS

Forms were distributed to seventeen dental officers of various bases throughout the country, whose dental centres performed outpatient functions daily. These dental centres also had comparable chairs, equipments and staff especially the dental surgery assistants.

Patients were asked to fill in their times of arrival and the times they were called into the surgery. Upon completion of respective attendances, dental officers were to fill in the times that patients left their chairs. They were also to record the procedures done. Participating dental officers were told to make their recordings as per daily attendances and not to choose their patients. They were to compile about 100 cases each. No distinction was to be made between military, family and civilian cases. Data analysis was carried out using Microsoft Excel 2000 and SPSS 11.5 for Windows (2001).

RESULTS

Seventeen dental officers comprising of fourteen males and three females responded to this study. Their working experiences range from a year to twenty years with an average of 9.4 years. Ten received their undergraduate training locally whilst seven received theirs overseas. Details are as per Table 1.

Table 1: Distribution of dental officers

Training and Experience		Number of personnel
Local		10
Number of years in practice	1	2
	3	2
	6	2
	9	3
	13	1
Overseas		7
Number of years in practice	1	1
	6	1
	17	2
	19	2
	20	1

A sample size of 1780 attendances comprising of 63.1% military personnel, 12.4% family members and 24.5% civilians was obtained. Sample size variation between operators is between 95 and 112. The longest average time taken by an operator is 25.4 minutes whilst the shortest, 9.4 minutes (Table 2). Overall, the average treatment time is 16.6 minutes with a standard deviation of 10.3 minutes. Median is 15.0 minutes whilst the mode is at 10.0 minutes.

Table 2: Average treatment time of each operator

OPERATOR	Number (n)	Mean (mins)	SD (mins)
A	107	25.4	17.0
B	111	24.1	13.8
C	102	21.0	13.0
D	103	20.2	10.4
E	98	20.1	16.2
F	108	19.6	12.6
G	105	18.6	8.7
H	107	17.1	8.4
I	112	16.9	13.8
J	95	14.3	11.7
K	111	13.3	12.0
L	104	13.2	8.0
M	108	12.5	4.7
N	111	12.0	7.4
O	101	10.9	7.2
P	97	10.7	4.5
Q	100	9.4	5.4
ALL	**1780**	**16.6 mins**	**10.3 mins**

For further examination, procedures performed by the operators were grouped into the following categories viz extractions, fillings, examinations, scaling and prophylaxis, denture works, dressings, oral hygiene instruction, minor oral surgery, root canal therapy and miscellaneous (Table 3).

Table 3: Categories of dental treament

CATEGORIES	
Extractions	All extractions (single, multiple) of permanent and primary teeth, retained root(s) or combination done in one session, not involving the raising of flaps.
Fillings	All fillings i.e. amalgam, composite resins, combination (single, multiple) with or without pins done in one session.
Examinations	All clinical examinations followed by advice, medication or referral for example, routine annual check-ups, pre course check up, examination of a complaint followed by prescription mainly to control pain and inflammation e.g. cases of acute pericoronitis, ulcers.
Scaling and prophylaxis	Single, multiple or all quadrants.
Denture works	Bite registrations, try-ins, impression takings, issues
Dressings	All temporary dressings.
OHI	Chairside oral hygiene instruction.
Minor oral surgery	Extractions involving raising of flaps.
Root canal treatment	Procedures involving pulp chamber and canals performed in particular sessions.
Miscellaneous	For cases such as dry socket, post-op minor oral surgery reviews, irrigation and drainage.

The average times taken for each category are as per Table 4.

Table 4: Average time for dental procedures

PROCEDURE	**Number (n)**	**%**	**Mean (mins)**	**SD (mins)**
Extractions	581	32.6	15.4	10.4
Fillings	546	30.7	18.6	10.7
Examination	250	14.0	9.1	7.9
Scaling and Prophylaxis	159	8.9	19.9	10.8
Denture works	71	4.0	17.0	10.4
Dressings	66	3.7	15.9	8.3
Oral Hygiene Instruction	39	2.2	8.6	3.9
Minor Oral Surgery	23	1.3	51.4	25.2
Root Canal Treatment	16	0.9	29.4	11.1
Misc	29	1.6	15.7	19.5
TOTAL	**1780**	**100**	**16.5**	**11.9**

DISCUSSION

In the Dental Service of the Malaysian Armed Forces (DSMAF) non-appointed patients are seen during morning sick calls which start at 8:00 a.m. and end usually between 10:00 a.m. and 11:00 a.m. The rest of the day is reserved for appointments of follow-up cases. Treatments are not only restricted to those that relief pain and assuage discomfort but also non-emergency procedures such as scaling and polishing, precourse and predischarge examinations which the operators feel will best serve their patients' interests when performed at those visits rather than at later appointments.

The average treatment time found in this study is 16.6 minutes. Inter-operator comparisons showed variations that may be attributed to their different training backgrounds and years of experience. Comparison between local and overseas graduates showed that local graduates averaged at 16.59 minutes whilst overseas graduates, 16.26 minutes. However t-test results did not show any statistically significant difference between the average treatment times of these two groups, ($p>0.05$) (Table 5).

Table 5: Average treatment time by local and overseas graduates.

		Mean (minutes)	Std. Deviation	p-value
Treatment Time	Local (Malaysia)	16.59	12.29	0.568
	Overseas	16.26	11.20	

In Table 6, operators' years of working experience are divided into two categories i.e. those who have worked for ten years or less and those who have worked for more than ten years. The average treatment time for operators who have worked for more than ten years is found to be significantly shorter than those who have worked for fewer number of years (t-test, $p<0.005$)

Table 6: Average treatment time result experience officers

		Mean (minutes)	Std. Deviation	p-value
Treatment Time	10 years or less	17.25	12.8	<0.005
	More than 10 years	14.97	9.8	

No comparison is made between male and female operators as there are only three female dental officers out of seventeen in the group.

Of the 1780 attendances in this study 1185 or 66.6% were caries-related (based on the total number of extractions, fillings and dressings done). Similar findings were also observed by Singh[2] and Wan Hussain[3] in 1996 respectively, i.e. nearly 70% and 63.8% of cases coming in were consequences of caries. Hussin[4] however found a lower rate of 50% which was comparable with what Cheong and Chong[5] saw in the Singapore Armed Forces (52%). These variations in the data are possibly due to the different diagnostic criteria used, interexaminer variability and respective reasons for collecting the data. Greater service accessibility and awareness are also likely contributors to the higher percentage in 1996 and this study as compared to that of 1988.

Extraction formed the main bulk of treatment done (32.6%, n = 581 Table 4) followed by amalgam/composite fillings (n = 546), examination (n = 250), scaling and prophylaxis (n = 159). Other treatments include denture-related works, dressings, oral hygiene instruction, minor oral surgery, root canal treatment and other miscellaneous procedures such as post-operation suture removal and reviews. The time to do extraction averaged at 15.4 minutes (standard deviation 10.4 minutes); fillings 18.3 minutes (standard deviation 10.0 minutes); examination 14.1 minutes; scaling and prophylaxis 8.9 minutes. No attempt has been made to put weightage to the procedures according to their complexity as advocated by Chartier and Burrow[6] as in this instance the concern is mainly on average treatment time.

Taking the overall average treatment time of 16.6 minutes found in the study, an eight-hour working day with a one-hour lunch break, one can estimate that on average a dental officer can see 25 – 26 patients in a day (7 x 60 ÷ 16.6). This number will however be less when the interval between dismissal of one patient to the entrance of the next and other short breaks are taken into account.

The Dental Service of the Malaysian Armed Forces is not only entrusted with providing outpatient service but also the attainment and maintenance of dental fitness of soldiers. As most outpatient attendees at DSMAF centres are symptomatic patients,[2,7-11] an examination of caries prevalence will provide an estimate of the impending workload to render the soldiers dentally fit.

A survey by the Ministry of Health found caries prevalence among Malaysian adults to be 90.7% (Oral Health Division, Ministry of Health Malaysia 2001)[12] whilst a study by Samsuri[10] in 1999 on a group of Malaysian Armed Forces personnel showed caries prevalence of 87.2%. In a unit of 1000 men and taking 90% caries prevalence, one would expect 900 members of the unit to be affected. If a DT index of 0.6 decayed tooth per person is taken this will mean a workload of 149.4 man-hours (900 x 0.6 x 16.6 minutes ÷ 60). On an 8-hour working day, a single dental officer will need 18.7 days to make the unit dentally fit. This will further increase when other considerations such interpatient time, rest periods and other treatment needs such as scaling and prophylaxis and dentures are taken into account. An appreciation can then be made on the number of days or the number of dental officers required to prepare troops before their deployment date.

Time taken to perform various dental procedures is a useful tool in estimating clinical man-hour and manpower requirements in community-based set-ups. It should be noted that generalisation of data from this study to the whole dental fraternity has to be done with caution due to the non-random sampling. However this is a less serious concern as information of use can still be derived from the results. It has been advocated that of more practical importance is the consistent use of the set of values over a period of time so that programme monitoring can be achieved against measurable reference points.[6]

CONCLUSION

The average treatment time taken by DSMAF officers to treat their outpatients is 16.6 minutes per patient. There is no significant difference in treatment times between local (Malaysian) and foreign graduates. However, the average treatment time performed by operators who had worked longer appeared to be significantly shorter than those with less working experience. However, many confounding variables should be studied more closely before concluding that there is a relationship between years of experience and length of treatment time.

This treatment time will be a useful tool in estimating manpower requirements. especially in community-based programmes. For the military it assists in time and space planning of getting troops dentally fit. Interpatient time and finer definitions of procedures will need to be addressed if more accurate time estimates are required.

REFERENCE

1. Mahoney GD. The operational dental officer in the ADF. ADF Health, J Aust Defence Force. 2003;4:40-44.
2. Singh D. Needs and demand for dental care among Malaysian Armed Forces personnel – Master in Community Dentistry thesis, Faculty of Dentistry, University Malaya, Kuala Lumpur 1996.
3. Wan Hussain NMZ. Prevalence and impact of orofacial pain and discomfort among Malaysian Army personnel. Master in Community Dentistry thesis, Faculty of Dentistry, University Malaya, Kuala Lumpur 1996.
4. Hussin ZA. Dental Fitness. Malaysian Medical and Dental Corps Annual Magazine. 1987:29-33.
5. Cheong YH, Chong LL. Dental survey on soldiers of an infantry Battalion of Singapore Armed Forces (SAF) at the time of completion of national service. Sing Dent J. 1988;13:27-30.
6. Chartier A, Burrow D. Relative Standardised Time Units – Utilisation in the management of public dental health programmes. South Australian Dental Service. Dec 2000.
7. Faki B. Dental services necessary or not. Mirror and Probe Dental Service Malaysian Armed Forces Bulletin. 1989;13:13-4.
8. Indrasanan S. Oral health status of army personnel. Mirror & Probe. 1989;1:27-28.
9. Halina H. The effectiveness of the Active Dental Support programme in the Malaysian Armed Forces. Master of Community Dentistry thesis, Faculty of Dentistry University of Malaya. 1995.
10. Samsuri N. Oral Health and related impacts among commandos in Malaysian Armed Forces. Master of Community Dentistry thesis, Faculty of Dentistry University of Malaya. 1999.
11. Borhan J. Periodontal treatment needs among Malaysian Armed Forces personnel. Master of Community Dentistry thesis, Faculty of Dentistry University of Malaya. 1995.
12. Oral Health Division, Ministry of Health Malaysia. Oral health status impacts and treatment needs of Malaysia adults. Preliminary report 2001.

Address for correspondence:

Dr. Tuti Ningseh Mohd Dom
BDS (Mal) MPH (Michigan)
Department of Dental Public Health,
Faculty of Dentistry,
Universiti Kebangsaan Malaysia (UKM),
Jalan Raja Muda Abdul Aziz.

Malaysian Dental Journal (2005) 26(2) 121-129

MALAYSIAN DENTAL JOURNAL

A One-year Study of Peri-implant Tissue Status of Branemark Implants

Yunus N, BDS (Glasgow), MSc (Bristol) *Associate Professor & Head of Department, Department of Prosthetic Dentistry, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia*

Taiyeb-Ali TB, BDS (Malaya), MSc (London), FDSRCS(Edin) *Professor, Department of Oral Medicine, Oral Pathology and Periodontology, Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia*

Manan NM, BDS (Malaya) *Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia*

Zakaria N, BDS (Malaya) *Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia*

ABSTRACT

It is relevant to observe the clinical response of peri-implant tissues over time in order to determine whether they respond in any regular pattern in maintaining health. It is therefore the aim of this study to compare peri-implant tissue status around implants at the time of prosthesis placement and at subsequent intervals. A one-year study was conducted on implant patients provided with various fixed and removable prostheses in the maxilla and/or the mandible. There were 47 implants (Mark II, Branemark system, Nobelbiocare, Gotenberg, Sweden) placed in 12 patients. Baseline measurements were obtained during prosthesis insertion and follow-up examinations at 3, 6 and 12 months intervals. Evaluations of plaque scores (PS), bleeding on probing scores (BOP), periodontal probing pocket depths (PPD), and recession as well as radiographic assessments were conducted. Bone changes were determined from the radiographs by an examiner using predetermined fixed reference points. A repeated measures ANOVA and post-hoc t-test were used to compare the clinical parameters between maxillary and mandibular implants and between implants supporting fixed and removable prostheses at all time intervals over the 1 year period. The results showed that PS was lowest at baseline and highest at 12 months. BOP and PPD recorded the highest value at 3-months interval and recession around implants increased from baseline to 12 months. Comparisons around implants supporting fixed and removable prostheses showed mean PS and recession at all time intervals to be significantly different ($p<0.05$) while comparison between maxillary and mandibular implants indicated only the mean PS to be significantly different ($p<0.05$). Comparison of bone loss around implants at the one year interval showed a higher proportion of implants supporting removable prostheses (46%) exhibited bone loss as compared to implants supporting fixed prostheses (13%). A higher percentage of implants in the maxilla (48%) exhibited bone loss as compared to the mandibular implants (13%). Conclusions: The highest values for BOP, PPD and recession for all implants were observed at the 3-month interval after prosthesis placement. Greater PS and gingival recession were observed in implants supporting fixed as compared to removable prostheses. Greater PS was observed in maxillary compared to mandibular implants. Bone loss was detected in a higher percentage of implants supporting removable prostheses and around maxillary implants.

Key words:
Implant, soft tissue, prosthetic

INTRODUCTION

The surge in the use of dental implants was initiated by Professor Branemark and co-workers (1969) in Sweden.[1] Following that, various animal and clinical studies on osseointegration were conducted. Now with the relatively higher success rate and predictibility, implant-supported prostheses are widely accepted as a treatment modality or option in oral rehabilitation.

The use of implants was initially described for edentulous patients mainly to improve denture retention[2-4] but at present, the application has been extended to partially edentulous and single-tooth situations.

There are various clinical periodontal parameters used to evaluate the peri-implant tissue response. The practice of regular assessment of periodontal parameters over a period of time during the maintenance phase will allow the detection of signs of inflammation so that early interception by appropriate means will be possible.

Lang et al.[5] considered periodontal probing as a reasonable clinical diagnostic procedure to monitor the status of peri-implant tissues and together with radiographic parameters, are useful predictors for peri-implant status alteration.[6] It is also relevant to observe the clinical response of the tissues surrounding implants to determine whether tissue responses in any regular pattern in the maintenance of health over a period of time. However one has to be cautious in interpreting the soft tissue response to plaque, as peri-implant mucosa of implants at certain locations were found to have greater

risks to plaque-induced inflammation as compared to the gingiva in natural dentition.[7] This may be of importance in terms of maintenance for implant patients as the standard hygiene recall and treatment regime may need to be revised in order to minimize peri-implantitis and bone loss.

Therefore, the intention of the study was to observe the biologic response of peri-implant tissues and to compare the peri-implant tissue status between implants supporting fixed and removable prostheses and between maxillary and mandibular implants over a one year period.

MATERIALS AND METHODS

The study was carried out over a one year period. All the patients enrolled for the study were treated at the Faculty of Dentistry, University of Malaya, Kuala Lumpur, Malaysia. A total of fifteen patients were selected for the study, and of these patients, two patients did not turn-up for follow-up. One patient died; hence twelve patients (7 males, 5 females) remained in the study. The mean age of the patients was 52.2 years with the range between 35-68 years. This study evaluated 47 implants of the Branemark System MK II type of implants (Nobelbiocare, Gotenberg, Sweden). Fifteen implants were placed in the maxilla and 31 implants in the mandible. In terms of the prosthesis types, 31 implants supported 8 units of fixed prostheses and 16 implants supported 6 units of removable prostheses (Table 1).

All implants utilized for the prostheses were clinically osseointegrated and had been maintained throughout the study period. Some implants had been lost during the healing period prior to abutment connection and prosthesis construction. A 2-stage surgery with 3-6 months submerged healing according to the Branemark original protocol was followed.

At the second-stage surgery, healing abutments were connected. Abutments were connected at 2-3 weeks after the second stage surgery and prostheses were inserted between 2-6 weeks later. The soft tissue parameters were measured on all the implants at prosthesis insertion (baseline) and subsequently at 3, 6 and 12 months. The radiographic assessments were made at baseline and at 12 month.

Plaque score

Plaque score (PS) was used to quantify the amount of plaque retained on the 4 aspects of each implant using Waite's Plaque Index (1983) utilizing anodized Waite's Plaque Probe. The following score was used for the assessment:

Score 0 = No plaque
Score 1 = Thin plaque detected with Waite's probe
Score 2 = Moderate layer of plaque covering the anodized probe tip
Score 3 = Heaped plaque (added index in the present study to accommodate the observation)

Bleeding on probing

Assessment of bleeding on probing (BOP) was done using Gingivitis Index and the following score was used:

Score 0 = No bleeding on probing
Score 1 = Slow bleeding on probing
Score 2 = Copious bleeding on probing
Score 3 = Spontaneous bleeding present before probing

Peri-implant probing pocket depth

The peri-implant probing depth (PPD) was measured to the nearest millimeter at mesial, distal, buccal and lingual aspects of each implant.

Table 1. Location of implants and types of prostheses constructed

		No. of implants
Location of implants	Maxilla	15
	Mandible	32
	Total	47
Type of prostheses	Fixed	31 (8 units)
	Removable	16 (6 units)
	Total	47 (14 units)

Gingival Recession

The determination of the changes in the height of gingival margin was made relative to the baseline, with reference to the known height of abutment as in baseline casts. The recession assessments were done on the midbuccal and midlingual surfaces of the implant.

Radiographic assessment of marginal bone loss

Periapical radiographs were generally used to assess the marginal bone loss. In some patients panoramic radiographs were used when there were difficulties with the alignment of the periapical film. In order to correct the dimensional distortion, the apparent dimension of each implant on the radiograph was compared with the known implant length. The marginal bone level assessment was done at the mesial and distal implant sites.

The clinical parameters were measured by a periodontist (second author) and the radiographic assessments performed by the other two authors. Intra-examiner and inter-examiner agreement on bone level was initially obtained. The reproducibility assessment was performed twice within 2-week interval and the intra- and inter-examiner reproducibility was more than 86%. Peri-implant bone change was analyzed as categorical data with dichotomous outcome (loss or no loss of bone).

Statistical analyses were made using SPSS 11.0 for Windows with repeated measures analyses of variance (ANOVA) and t-test.

RESULTS

Table 1 depicts the distribution of the implants in both the maxilla and the mandible and the type of prostheses constructed in relation to these implants. The mean values and standard deviations of the clinical parameters recorded around 47 implants at baseline, 3 months, 6 months and 12 months intervals are shown in Table 2. Figures 1, 2, 3, 4 show the graphical representation of the values for PS, BOP, PPD and recession respectively.

Statistical analysis using ANOVA comparing the clinical parameters around implants supporting fixed and removable prostheses at all time intervals over the 1 year period (Table 3) showed there was a significant difference in PS and recession values ($p<0.05$). Post-hoc t-test comparing the mean values of PS and recession around implants supporting fixed and removable prostheses showed there was a significant difference in the values with $p < 0.001$ for PS and $p < 0.05$ for recession (Table 3). Statistical analysis using ANOVA comparing the clinical parameters around maxillary and mandibular implants at all time intervals over the 1 year period (Table 4) showed there was a significant difference in PS value ($p< 0.05$). No significant difference was observed for other parameters such as BOP, PPD and recession ($p> 0.05$). Post-hoc t-test comparing the mean PS values between mandibular and mandibular implants showed there was a significant difference in the values with $p < 0.01$ (Table 4).

The percentage of bone loss around implants supporting either fixed or removable prostheses and for maxillary or mandibular implants is shown in Table 5 & 6.

DISCUSSIONS

The PS value for all implants in this study was lowest at baseline and highest at the 12-months interval. There was an increase in PS values observed in patients treated with implant-supported prostheses despite routine follow-up. Patient motivation and instructions in meticulous oral hygiene may need to be reinforced with greater emphasis using suitable techniques during the issue of these prostheses and during the follow-ups.

The mean BOP score for all implants at all time intervals in the study ranged from 0.63-0.83 and the mean PPD varied from 2.18-2.74mm, with the highest values of these clinical parameters recorded at the 3-month assessment. This may probably be due to the adjustment period for patients in the initial phase of implant-supported prosthesis usage whereby maintenance of oral hygiene and peri-implant tissue health is being appreciated

Table 2. Mean values of the clinical parameters for all implants at various time intervals

Clinical Parameter	Baseline Mean (SD)	3 month Mean (SD)	6 month Mean (SD)	12 month Mean (SD)
PS	0.19 (0.23)	0.43 (0.45)	0.32 (0.35)	0.47 (0.48)
BOP score	0.63 (0.48)	0.83 (0.50)	0.74 (0.45)	0.71 (0.51)
PPD (mm)	2.42 (1.47)	2.74 (1.28)	2.18 (1.20)	2.29 (1.07)
Recession (mm)	0.41 (0.70)	1.08 (1.31)	0.84 (0.98)	0.92 (1.03)

Figure 1. **Mean plaque score (PS) around implants at all time intervals**

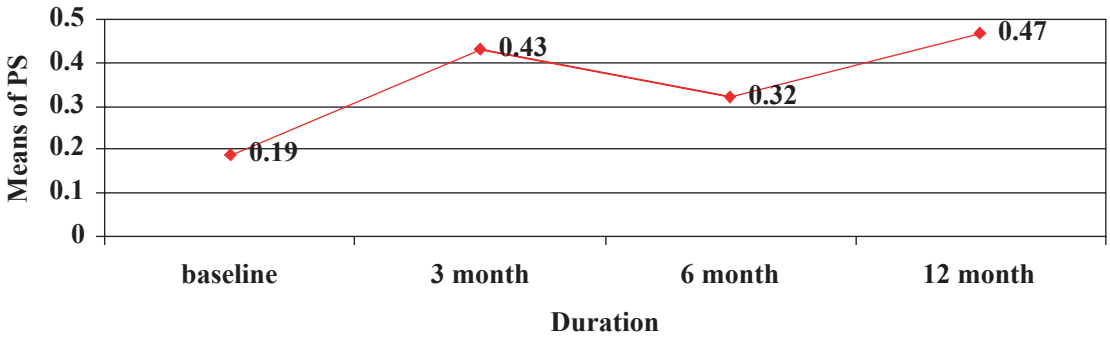


Figure 2. **Mean bleeding on probing (BOP) score around implants at all time intervals**

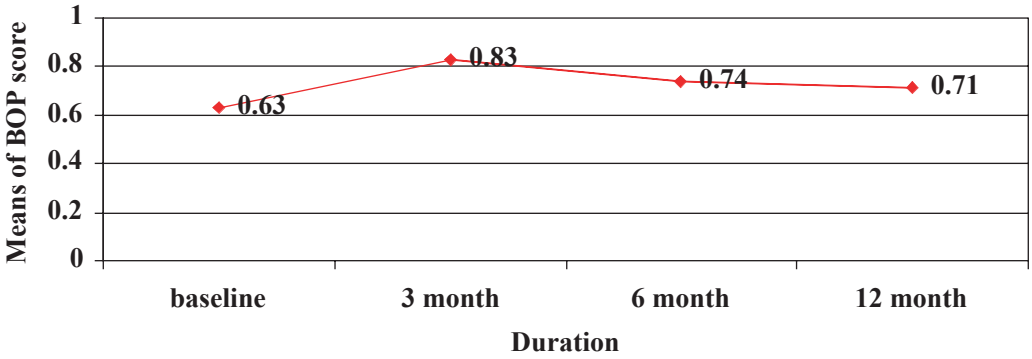


Figure 3. **Mean peri-implant probing depth (PPD) around implants at all time intervals**

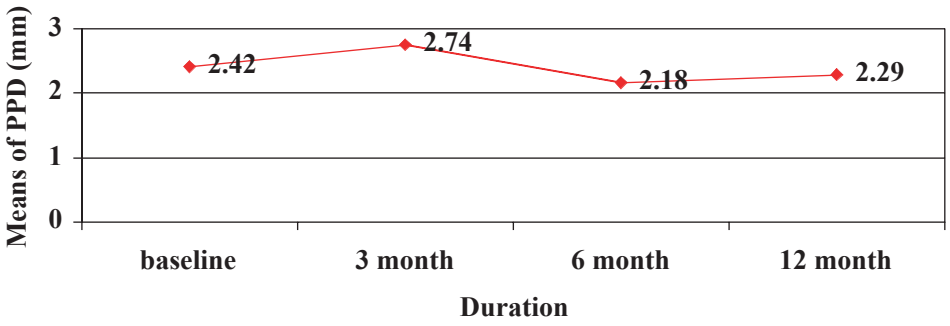


Figure 4. **Mean gingival recession around implants at all time intervals**

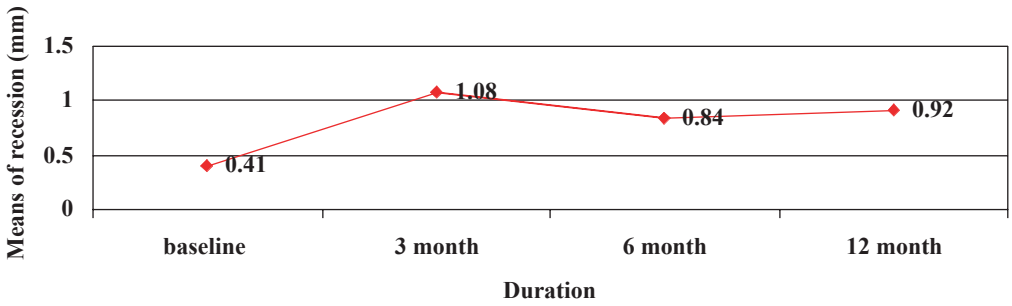


Table 3. The mean values of the clinical parameters at all time intervals in implants supporting fixed and removable prostheses with the results of ANOVA and post-hoc test comparing the mean values between implants supporting fixed and removable prostheses.

Clinical Parameter	Fixed Mean (SD)	Removable Mean (SD)	ANOVA		t-test
			F- value	p-value (Significance)	p-value
PS	0.44 (0.42)	0.16 (0.27)	20.88	0.00 (S)	P<0.001
BOP score	0.73 (0.48)	0.71 (0.49)	0.03	0.86 (NS)	-
PPD (mm)	2.26 (1.30)	2.68 (1.16)	3.91	0.05 (NS)	-
Recession (mm)	0.92 (1.04)	0.57 (1.02)	4.39	0.04 (S)	P<0.05

Table 4. The mean values of the clinical parameters at all time intervals in maxillary and mandibular implants with the results of ANOVA and post-hoc test comparing the mean values between maxillary and mandibular implants.

Clinical Parameter	Maxilla Mean (SD)	Mandible Mean (SD)	ANOVA		t-test
			F- value	p-value (Significance)	p-value
PS	0.16 (0.28)	0.44 (0.42)	21.98	0.00 (S)	P<0.01
BOP score	0.64 (0.43)	0.76 (0.50)	2.58	0.11 (NS)	-
PPD (mm)	2.34 (1.34)	2.43 (1.24)	0.20	0.66 (NS)	-
Recession (mm)	0.59 (1.01)	0.91 (1.06)	3.75	0.06 (NS)	-

Table 5. Percentage of bone loss (%) around implants supporting fixed and removable prostheses at various intervals.

Duration	Type of prosthesis	% bone loss
0 month	Fixed	9
	Removable	20
6 month	Fixed	8
	Removable	50
12 month	Fixed	13
	Removable	46

Table 6. Percentage of bone loss (%) around implants in the maxilla and mandible at various intervals.

Duration	Type of prosthesis	% bone loss
0 month	Mandible	7
	Maxilla	29
6 month	Mandible	8
	Maxilla	42
12 month	Mandible	13
	Maxilla	48

by the patient. A comparison of BOP and PPD values at all time intervals around maxillary and mandibular implants showed no significant difference even though there was a significant difference in the mean PS values. Higher PS score was seen in the mandibular implants probably due to easier access for hygiene in the upper implant regions. Similarly no significant difference was observed in these parameters (BOP & PPD) between implants supporting fixed and removable prostheses, although greater amount of plaque was observed in the former. Higher plaque accumulation seen may be explained by the difficulties in cleaning between abutments or between the abutments and the pontics or teeth in fixed restorations.

It was generally believed that plaque accumulation would induce negative peri-implant tissue response.[9-11] The findings of the present study seem to concur with previous findings[12] whence no evidence to support a correlation between oral hygiene and mucosal health was detected. A one year study of peri-implant tissue response also showed no correlation between plaque and PPD.[13] Hence the role of plaque per se in contributing towards peri-implant mucosal health and long-term implant success may not be significant and probably be questionable. Although many researchers consider the mucosal response to be correlated to marginal bone loss and osseointegration,[14-16] others have questioned the influence of plaque in implant success.[17] Henry et al.[18] has suggested that although the role of bacterial plaque in the pathogenesis around implants is still uncertain, it should however be controlled.

The soft tissue response around implants may not be similar to that around the natural dentition as reported by Ericsson & Lindhe[19] who found BOP in the majority of clinically healthy implant sites. A study by Papaioannou et al.[20] demonstrated that more than half of the implant sites demonstrated bleeding on probing. As a matter of interest the majority of implants sites in our study also exhibited BOP. The underlying connective tissue around implants is different from the natural teeth with respect to the orientation of the collagen fibres of the peri-implant mucosa which run parallel with the surface of the abutment[21] in comparison to the natural dentition whereby they run perpendicular and are inserted into the root surface. This collagen barrier formed around implants may provide minimal resistance to probing resulting in bleeding. Therefore one has to be cautious in interpreting the response of soft tissue around implants to plaque, as peri-implant mucosa of implants at certain locations may have greater risks to plaque-induced inflammation as compared to the gingiva in natural dentition.[7]

This study also showed that recession increased from baseline to the 12-months interval. The amount of soft tissue recession observed which was in the range of 0.41-1.08 mm was within the range quoted by Touati & Guez[22] and Small & Tarnow.[23] The greatest change in the present study was observed at the 3-month interval after prosthesis placement. Other studies[24,25] observed the apical soft tissue displacement to occur mainly during the first 6 months after second stage surgery although in another study[23] majority of recession occurred within the first 3 months after implant exposure. The time taken for the soft tissue to heal and mature is of clinical importance which from the present study and other studies indicate the first 3-6 months as being crucial. For this reason, clinical protocol should take into account at least 0.5-1.0 mm recession. Construction of final prosthesis may need to be deferred until it is stabilized so that the margin of the prosthesis can be established without further changes particularly in relation to fixed prostheses in the esthetic zone. This may aid in providing long-term esthetic results in the final fixed prosthesis.

In this study, the mean recession values at all time intervals around maxillary and mandibular implants were found to be not significantly different. And this was in agreement with that reported.[23] However, there have been conflicting results from other studies on the soft tissue changes around implants. In one study,[26] it was observed that more recession occurred in the maxillary as compared to the mandibular implants while still in another study[24] more recession resulted in the mandibular implants. This variability may be explained by several factors that may influence healing around implants for example patient factors, manufacturer's design in implant fixture and abutment, surgical technique and prosthesis type and design.

Peri-implant bone levels were measured at baseline and at 12-months interval. Dichotomous observation of bone loss was made in this study since bone gain was not observed. When the implants were grouped as either maxillary or mandibular implants, it was found that a higher proportion of implants with bone loss was found in the maxilla in comparison to mandibular implants. This result concurred with findings by Penarrocha et al.[27] where implants placed in the completely edentulous mandibles demonstrated less bone loss as compared to implants in the edentulous maxilla. Poorer bone quality in the maxilla may have attributed to these findings. In other long term studies,[2,25,28] where the Branemark implant system was utilized, it was similarly observed that implants in the maxilla appeared to have less favourable prognosis than implants in the mandibles.

This study showed that a higher proportion of implants supporting removable prostheses (46%) showed bone loss as compared to implants supporting fixed prostheses (13%). Makkonen et al.[29] in a 5-year prospective study, also reported lower implant survival rate in the removable prostheses and at the 3-year assessment they observed more marginal bone loss in this implant group as compared to the implants supporting fixed prostheses. Removable prosthesis is normally a second choice option to fixed prosthesis because of the presence of poor bone quality and quantity. As a result of this initial indication, the prognosis and success rate of implants supporting removable prosthesis is less favourable.

One limitation of the present study was the inability to assess the effect of various prosthesis designs on the clinical parameters due to the limited sample size. A larger sample might yield more definitive comparisons and aid in greater understanding of the soft tissue response. As there have been modifications and changes to the implant/ abutment design of the Branemark system, the sample size could not be increased.

However, assessments of soft tissue contours are important towards achieving acceptable esthetics, function and physiological harmony between the tissues and prosthesis constructed. The health of the soft tissues should be achieved during the issue of the definitive prosthesis which should be maintained thereafter. The current trend in clinical implantology is to provide restorations as soon as possible to meet patients' desires. Clinician following the recommendations of implant manufacturers for almost immediate implantation and function should always be aware of the possible soft tissue alterations and its impact on the final prostheses.

CONCLUSIONS

The highest values of bleeding on probing, peri-implant probing depth and recession around all implants were observed at the 3-months interval after prosthesis placement. Greater plaque score and gingival recession were observed in implants supporting fixed as compared to removable prostheses. Greater plaque score was observed in mandibular versus maxillary implants. Bone loss was detected in a higher proportion of implants supporting removable prostheses and maxillary implants.

ACKNOWLEDGEMENT

This study had been funded by research grants Vote F (1995 and 2001), University of Malaya, Kuala Lumpur.

REFERENCES

1. Branemark PI, Adell R, Breine U, Hansson BO, Lindstrom J, Ohlsson A. Intra-osseous anchorage of dental prostheses. I. Experimental studies. Scand J Plast Reconstr Surg. 1969;3:81-100.
2. Adell R, Lekholm U, Rockler B, Branemark PI. A 15-year study of osseointegrated implants in the treatment of the edentulous jaw. Int J Oral Surg. 1981;10:387-416
3. Babbush CA, Kent JN, Misiek DJ. Titanium plasma-sprayed (TPS) screw implants for the reconstruction of the edentulous mandible. J Oral Maxillofac Surg. 1986;44:274-82.
4. Mericske-Stern R, Milani D, Mericske E, Olah A. Periotest measurements and osseointegration of mandibular ITI implants supporting overdentures. A one-year longitudinal study. Clin Oral Implants Res. 1995;6:73-82.
5. Lang NP, Wetzel AC, Stich H, Caffesse RG. Histologic probe penetration in healthy and inflamed peri-implant tissues. Clin Oral Implants Res. 1994;5:191-201.
6. Tawse-Smith A, Perio C, Payne AG et al. One-stage operative procedure using two different implant systems: a perspective study on implant overdentures in the edentulous mandible. Clin Implant Dent Relat Res. 2001;3:185-93
7. Toijanic JA, Ward GB, Gewerth ME Banakir ML. A longitudinal clinical comparison of plaque-induced inflammation between gingival and peri-implant soft tissues in the maxilla. J Periodontol. 2001;72:1139-45.
8. Waite IM, Jalil R, Cornick DER. A probe for plaque measurement. Dental Practice. 1983;21:1.
9. van Steenberghe D, Klinge B, Linden U, Quirynen M, Herrmann I, Garpland C. Periodontal indices around natural teeth and titanium abutments: a longitudinal multicenter study. J Periodontol. 1993;64:538-41.
10. Berglundh T, Lindhe J, Marinello C, Ericsson I, Liljenberg B. Soft tissue reaction to de novo plaque formation on implants and teeth. An experimental study in the dog. Clin Oral Implants Res. 1992;3:1-8.
11. Lekholm U, Adell R, Lindhe J, Branemark PI, Eriksson B, Rockler B, et al. Marginal tissue reactions at osseointegrated titanium fixtures. (II) A crosssectional retrospective study. Int J Oral Maxillofac Surg. 1986;15:53-61.
12. Apse P, Zarb GA, Schmitt A, Lewis DW. The longitudinal effectiveness of osseointegrated dental implants. The Toronto Study: peri-implant mucosal response. Int J Periodontics Restorative Dent. 1991;11:94-111.
13. Rungcharassaeng K, Lozada JL, Kan JYK, Kim JS, Campagni WV, Munoz CA. Peri-implant tissue response of immediately loaded, threaded, HA-coated implants: 1-year results. J Prosthet Dent. 2002;87:173-181.
14. Teixeira ER, Sato Y, Akagawa Y, Kimoto T. Correlation between mucosal inflammation and marginal bone loss around hydroxyapatite-coated implants: a 3-year cross-sectional study. Int J Oral Maxillofac Implants. 1997;12:74-81.
15. Block MS, Kent JN. Long-term follow-up on hydroxyapatite-coated cylindrical dental implants: a comparison between developmental and recent periods. J Oral Maxillofac Surg. 1994;52:937-43
16. Kirsch A, Mentag PJ. The IMZ endosseous two phase implant system: a complete oral rehabilitation treatment concept. J Oral Implantol. 1986;12:576-89.
17. Smith DE, Zarb GA. Criteria for success of osseointegrated endosseous implants. J Prosthet Dent. 1989;62:567-572.
18. Henry PJ, Tolman DE, Bolender C. The applicability of osseointegrated implants in the treatment of partially edentulous patients: three-year results of a prospective multicenter study. Quintessence Int. 1993;24:123-9.
19. Ericsson I, Lindhe J. Probing depth at implants and teeth. An experimental study in the dog. J Clin Periodontol. 1993;20:623-7.
20. Papaioannou W, Bollen CM, Van Eldere J, Quirynen M. The adherence of periodontopathogens to periodontal probes. A possible factor in intra-oral transmission? J Periodontol. 1996; 67:1164-9.
21. Berglundh T, Lindhe J, Jonsson K, Ericsson I. The topography of the vascular systems in the periodontal and peri-implant tissues in the dog. J Clin Periodontol. 1994;21:189-93.

22. Touati B and Guez G. Immediate implantation with provisionalization. From literature to clinical implications, Pract Proced Aesthet Dent. 14, 2002, 69
23. Small PN, Tarnow DP. Gingival recession around implants: A 1-year longitudinal prospective study. Int J Oral Maxillofac Implants. 2000;15:527-532.
24. Bengazi F, Wennstrom JL, Lekholm U. Recession of the soft tissue margin at oral implants. A 2-year longitudinal prospective study. Clin Oral Implants Res. 1996;7:303-10.
25. Adell R, Lekholm U, Rockler B et al. Marginal tissue reactions at osseointegrated titanium fixtures (1). A 3-year longitudinal prospective study. Int J Oral Maxillofac. Implants. 1986;15:39-52.
26. Ekfeldt A, Eriksson A, Johansson LA. Peri-implant mucosal level in patients treated with implant-supported fixed prostheses: a 1-year follow-up study. Int J Prosthodont. 2003;16:529-32
27. Penarrocha M, Palomar M, Sanchis JM, Guarinos J, Balaguer J. Radiologic study of marginal bone loss around 108 dental implants and its relationship to smoking, implant location, and morphology. Int J Oral Maxillofac Implants. 2004;19:861-7.
28. Jemt T, Chai J, Harnett J, Heath MR, Hutton JE, Johns RB, McKenna, S, McNamara DC, van Steenberghe D, Taylor R, Watson RM, Herrmann. A 5-year prospective multicenter follow-up report on overdentures supported by osseointegrated implants. Int J Oral Maxillofac Implants. 1996;11:291-8.
29. Makkonen TA, Holmberg S, Niemi L, Olsson C, Tammisalo T, Peltola. A 5-year prospective clinical study of Astra Tech dental implants supporting fixed bridges or overdentures in the edentulous mandible. Clin Oral Implants Res. 1997;8:469-75.

Address for correspondence:

Assoc. Prof. Dr. Norsiah Yunus,
Head,
Department of Prosthetic Dentists,
Faculty of Dentistry,
University of Malaya
50603 Kuala Lumpur, Malaysia.
Tel : 603-7967 4884
Fax : 603-7967 4535
Email : norsiah@com.edu.my

Malaysian Dental Journal (2005) 26(1) 130-131

MALAYSIAN DENTAL JOURNAL

Instructions to contributors

Original articles, editorial, correspondence and suggestion for review articles should be sent to:

Associate Professor Dr. Ngeow Wei Cheong,
Editor, Malaysian Dental Journal
Malaysian Dental Association
54-2, Medan Setia 2, Plaza Damansara
50490 Kuala Lumpur, Malaysia

Authors are requested to submit three copies of their typescript and illustration and also copy of their work in a file on a newly formatted 3.5 inch floppy disk.

The editor cannot accept responsibility for any damage or loss of typescripts. The editorial currently is unable to support electronic submissions; apart from the use of e-mailing for correspondence.

A paper is accepted for publication on the understanding that it has not been submitted simultaneously to another journal in the English Language.

Rejected papers will be returned to authors.

The editor reserves the right to make editorial and literary corrections. Any opinion expressed or policies advocated do not necessarily reflect the opinion or policies of the editors.

Copyright

Authors submitting a paper do so on the understanding the work has not been published before. The submission of the manuscript by the authors means that the authors automatically agree to sign exclusive copyright to the Editor and the publication committee if and when the publication is accepted for publication. The copyright transfer agreement will be sent to you by the editor with your proofs. A copy of the agreement must be signed by the principal author before any paper can be published.

We assure that no limitation will be put on your freedom to use material contained in the paper without requesting permission provide acknowledgement is made to the journal as the original source of publication.

Presentation of manuscript

Manuscript should be submitted in journal style. Spelling preferably be either British or American. Articles typed in double spacing throughout on good, white A4 paper with a margin of at least 3 cm all around. Type only on one side of the paper. Three copies of the typescript and illustration should be submitted and the authors retain a copy for reference.

In addition to the typed manuscript we also require every article be submitted as a file on a newly formatted 3.5 inch floppy disk. The manuscripts and the file on the disk must be identical and the disk should not contain other files. The disk must be clearly labeled with the title of the article, the names of the author(s) and the computer operating system (eg DOS, Macintosh) and WP version (word 2.0, WordPerfect 5.0) used. Files should not be converted to ASCII format.

Full papers

Papers should be set out as follows with each beginning in a separate sheet: title page, summary, text, acknowledgements, references, tables, caption to illustrations.

Title page. The title page should give the following information: 1) title of the article; 2) initials, name and address of each author, with higher academic qualifications and positions held; 3) name, address, telephone, fax and e-mail address.

Text. Normally only two categories of heading should be used: major ones should be typed in capital in the centre of the page and underlined; minor ones should be typed in lower case (with an initial capital letter) at the left hand margin and underlined.
Do not use he or she if the sex of the person is unknown; e.g. 'the patient'.

References. The accuracy of the references is the responsibility of the author. References should be entered consecutively by Arabic numerals in superscript in the text. The reference list should be in numerical order on a separate sheet in double spacing. Reference to journals should include the author's name and initials (list all authors when six or fewer; when seven or more list only the first three and add ‘et al.’), the title of paper, Journal name abbreviated, using index medicus abbreviations, year of publication, volume number, first and last page numbers (ie. Vancouver style).
For example: Ellis A, Moos K, El-Attar. An analysis of 2067 cases of zygomatico-orbital fractures. J Oral Maxillofac Surg 1985, 43: 413-417.
Reference to books should be sent out as follows: Scully C, Cawson RA. Medical Problems in Dentistry 3rd edn. Wright 1993:175.

Tables. These should be double spaced on separate sheets and contain only horizontal rules. Do not submit tables as photographs. A short descriptive title should appear above each table and any footnotes, suitably identified below. Care must be taken to ensure that all units are included. Ensure that each table is cited in the text.

Illustrations

Line illustration. All line illustrations should present a crisp black image on an even white background (127 mm x 173 mm or 5 x 7 inches) or no larger than 203 mm x 254 mm or 8 x l0 inches.
Photographic illustrations and radiographs. These should be submitted as clear lightly contrasted black and white prints (unmounted) sizes as above. Photomicrographs should have the magnification and details of the staining technique shown. Radiographs should be submitted as photographic prints carefully made to bring out the details to be illustrated, with an overlay indicating the area of importance.
All illustration should be carefully marked (by label pasted on the back or by a soft crayon) with figure number and authors name and the top of the figure should be indicated by an arrow. Never use ink of any kind. Do not use paper clips as these can scratch or mark illustrations. Caption should be typed, double spaced on separate sheets from the manuscript.
Patient confidentiality. Where illustrations must include recognizable individuals living or dead and of whatever age, great care must be taken to ensure that consent for publication has been given. Otherwise, the patient’s eyes or any indentifiable anatomy should be covered.
Permission to reproduce, borrowed illustration or table or identifiable clinical photographs. Written permission to reproduce, borrowed material (illustrations and tables) must be obtained form the original publisher and authors and submitted with the typescript. Borrowed material should be acknowledged in the caption in this style. 'Reproduced by the kind permission of..... (publishers) from /....(reference)'.

Page Proofs

Page proofs are sent to the author for checking. The proofs with any minor corrections must be returned by fax or post to the editor within 48 hours of receipt.

Proprietary names

Proprietary names of drugs, instruments etc. should be indicated by the use of initial capital letters.

Abbreviations and units

Avoid abbreviations in the title and abstract. All unusual abbreviations should be fully explained at their first occurrence in the text. All measurements should be expressed in SI units. Imperial units are also acceptable.

Offprints

Ten free offprints are supplied to the author. An offprint order form will be sent to the author with the page proof.