ORAL LESIONS FOUND IN SMOKERS

A SOCIOLOGICAL,

AETIOLOGICAL

PATHOLOGICAL

AND

FOLLOW-UP STUDY

A THESIS SUBMITTED IN CONFIRMITY WITH

THE REQUIREMENTS FOR

THE DEGREE OF

DOCTOR OF PHILOSOPHY

IN THE FACULTY OF MEDICINE,

UNIVERSITY OF PERADENIYA,

SRI LANKA.

1991

ANGELA DHARSHINI MUTHIAH

BDS (SRI LANKA)

SUMMARY

ORAL LESIONS FOUND IN SMOKERS - A SOCIOLOGICAL, AETIOLOGICAL, PATHOLOGICAL AND FOLLOW-UP STUDY

This dissertation includes studies on: 1. The knowledge, attitude, and opinions of smokers and the socio-economic aspects of smoking. 2. Detailed analysis on the aetiological aspects of oral lesions found in smokers as regards to smoking, betel chewing and mixed habits 3. Detailed pathological studies of oral lesions found in smokers from whom 141 biopsies were performed and histopathologically analysed in the first examination and 4. follow-up studies after a 2-3 year interval (1986-1989, 1987-1989) on 42 subjects from whom biopsies were obtained in the first examination and 31 subjects from whom biopsies have been obtained in the second examination. A statistically significant sample was obtained from five out of the 24 administrative districts of Sri Lanka. General hospitals situated in these districts were used as sampling centres. A total of 1648 subjects in the age groups 20 years and above were studied. Males only were studied, as very few females smoke in Sri Lanka.

Valuable data emerged on: 1. The prevalence of smoking in a Sri Lankan population, 2. The subjects knowledge, attitude and opinions relating to smoking, 3. The association between socio-economic factors and smoking, 4. The occurence of oral lesions in smokers, 5. The interactions of various factors affecting oral precancerous lesions and

oral cancer, 6. The identification of histopathological changes that can be related to clinical types of oral leukoplakia, 7. The quantitative aspects of the degree of dysplasia in oral leukoplakia, 8. The follow-up rate, 9. The ways to persuade the tobacco habitues to discontinue their tobacco habits and finally 10. The ways to evaluate the changes in the incidence and the pattern of regression of oral precancerous lesions.

The prevalence of smoking in the present study in a Sri Lankan population was 68.14%. With regard to smoking, cigarette smoking was found to be a common practice in Sri lanka. There is a significant association between attitude against smoking and prevalence of smoking ($P < .001$). Most of the smokers were having a positive antismoking attitude and they are concerned about the harmful effects of smoking on their health and the health of nearby non smokers. A significant association was found between prevalence of smoking and socio-economic factors such as educational level, marital status, occupation, income level and also with age and ethnicity ($P < .001$). But no association was found in the subjects living in suburbs, villages and towns. The degree of association for the prevalence of smoking is more with occupation, income level followed by marital status and age, education and ethnicity in the descending order.

Of the 1648 subjects, 366 lesions were found in smokers. The prevalence of leukoedema in the present study was 8.62%. The frequency of oral lesions seen among smokers

were leukoedema (12.64%), leukoplakia (12.64%), erythroplakia (0.09%), oral submucous fibrosis (0.36%), oral lichen planus (1.69%), leukokeratosis nicotina palati (3.29%) and carcinoma (1.87%). The average age for subjects with leukoedema, leukoplakia, oral submucous fibrosis, oral lichen planus, leukokeratosis nicotina palati and carcinoma was 43.90, 46.65, 42.20, 44.42, 37.97 and 51.17 years respectively (Table 21) and it was significantly higher than the average age of the rest of the sample except for oral submucous fibrosis and leukokeratosis nicotina palati. Leukoplakia was the commonest precancerous lesions found in smokers. A significant association was found between smoking and leukoplakia (χ^2 =11.85, df=1, P < .001). Most of the lesions were found in the age group 40-49 years (Table 26) and the common site of occurence was the buccal mucous membrane (Table 23).

The interactions of age, tobacco smoking and betel chewing on oral precancerous lesions and oral cancer aetiology was analysed by 'Discrete Multivariate Analysis'. A hierachy of log linear models were sequentially fitted until a good fit was obtained. The difference in the likelihood ratio statistic between two successive models served as a test for the interaction term included in the larger model. This study confirmed that age, smoking duration, smoking quantity and betel chewing jointly affect the presence of oral precancerous lesions and oral cancer. The magnitude of interaction in the three factor effect was higher than the other interaction terms (P< 0.05). The lesions were found only in a few subjects 6 (0.53%) when (i) they smoke

very few cigarettes, beedis or cigars, (ii) when they did not chew any betel, (iii) when the smoking duration was below 10 years and (iv) when the subjects were below the age of 45 years (Table 29).

Out of the total of 102 biopsies from oral leukoplakia, 29 (28.43%) were homogenous leukoplakias, 63 (61.76%) were nodular leukoplakias and 10 (9.80%) were ulcerated leukoplakias. There was a correlation between the clinical types of leukoplakia: viz, homogenous leukoplakia, nodular leukoplakia and ulcerated leukoplakia and their histopathological characteristics. The variations on the clinical types of leukoplakia had a high statistical significance for histopathological changes such as orthokeratosis, parakeratosis and hyperplasia and acanthosis ($P < 0.01$). With respect to ulceration however the variation was statistically significant at 5% level. The incidence of epithelial dysplasia was 30.39%. Nodular leukoplakias showed the highest percentage of epithelial dysplasia (80.65%). Statistical analysis showed its incidence to be significantly higher ($P < 0.05$) in the nodular type than in the other forms. The main dysplastic features noted in this study, in descending order were increased nuclear/ cytoplasmic ratio, nuclear hyperchromatism, disturbed maturational sequence, basal cell hyperplasia and prominent nucleoli. The peak incidence of epithelial dysplasia was 45.66% and found in the 50-59 year age group (Fig.105). In this study, the subjects with oral leukoplakia and dysplastic changes decreased with age after attaining a peak incidence (Fig.105).

The follow-up rate in this study was low(29.79%)due to unavoidable circumstances prevailing during the follow-up survey. Of the total lesions re-examined 30 (71.43%) were leukoplakias. There was also one erythroplakia, one oral submucous fibrosis and four oral lichen planus. There were also three growths on the oral mucous membrane and three oral cancers in the follow-up examination. The subjects who had completely discontinued their tobacco habits or changed their tobacco habits for the better were remarkably high for the smoking habit group (71.43%) when compared with the betel chewing habit group (42.42%) (Table 46).

The 30 leukoplakias were made of 9 homogenous leukoplakias, 17 nodular leukoplakias and 4 ulcerated leukoplakias. The behaviour of the 17 nodular leukoplakias showed that 9 were persistent, 3 each were recurrent lesions and regressive lesions and 2 lesions changed to homogenous leukoplakia. Of the 9 persistent nodular leukoplakias, 2/3 showed an increase in the size of the lesions during the examination period.

Of the 30 leukoplakias 7 (23.33%) regressed, while 10 (33.33%) either changed from one type to another or recurred within the examination period. A further 13 (43.33%) remained persistent. In the persistent group 5/13 (38.46%) had given up all their smoking and chewing habits. Of the subjects who showed regression of their lesions, 4 were smokers and 3 were mixed habitues in the first examination. In the follow-up study, 3 of them had given up the habit fully. There were still 2 smokers and 2 mixed

habitues left (Table 49).

Histopathological examination show that out of 19 cases of persistent and changed leukoplakias the histopathological appearances improved after 2-3 years. Dysplastic oral mucosal lesions regressed from 13 to 2 and these dysplastic lesions turned into hyperplasia and acanthosis (13 in the second examination). In the second examination there were also 2 lesions each of carcinoma-in-situ and invasive squamous cell carcinoma. The invasive squamous cell carcinomas were clinically diagnosed as nodular leukoplakias.