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Study of benign oral lesions and serum vitamin levels in tobacco consumers

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ABSTRACT

Background: Oral mucosal lesions are seen commonly in tobacco consumers and some of them are precancerous. Study of various benign oral mucosal lesions has done in tobacco consumers. Comparison of serum Vitamin levels was done in patients with benign oral lesions in tobacco consumers and non- tobacco consumers.
Methods: Retrospective study of 100 patients with benign oral lesion with tobacco addiction was done. Control group includes tobacco non-addicts. Detail history regarding tobacco consumption was noted. Tobacco consumption pattern was studied in patients with benign oral lesion. Ear, nose, throat examination was done in detail. Oral lesion was noted. Comparison between serum levels of vitamin A, C and vitamin E in benign oral lesion of patients with tobacco addiction and tobacco non-addicts were included in the study. The results were analysed statistically by student *t*-test.
Results: Patient with tobacco consumption with lime application was more prone to develop benign oral lesions. Gingivitis was most common lesion seen in study population followed by leukoplakia. Serum vitamin A, C and E were significantly decreased in tobacco consumers with benign oral lesions as compared to tobacco non-consumers. Decreased in serum vitamin levels were seen in all benign oral lesions irrespective of its malignant potential.
Conclusions: Gingivitis was most common lesion seen in study population followed by leukoplakia. Serum level of non-enzymatic antioxidant vitamin such as vitamin A, C and E gets decreased significantly in all benign oral lesions irrespective of its malignant potential. Serum levels of the antioxidant vitamins indicate need of increased oral intake of these vitamins to protect from damage in all benign oral lesions.

Keywords: Tobacco, Addiction, Benign, Vitamin

INTRODUCTION

Stomatitis, submucus oral fibrosis, leukoplakia, erythroplakia and gingivitis are some common oral lesions presented in ENT OPD. The aetiology for these benign oral lesions is multifactorial. The etiological factors for these lesions are genetic factor, hematologic deficiencies, immunologic abnormalities, trauma, stress, tobacco and betel nut addiction and food allergy.[1,2] Some of the oral lesions having premalignant potential. Oral sub mucus fibrosis, erythroplakia, etc. are premalignant conditions. The rate of malignant transformation of sub mucus fibrosis is 7.6% over a period of 17 years.[3] Machuca et al has shown that tobacco is a significant risk factor for periodontal disease.[4] The risk of malignant transformation of oral sub mucus fibrosis in tobacco chewers is around 400 times the risk involve in non-tobacco chewers.[4] It has proven that dietary intake of fruits and vegetables rich in the vitamin A have

protective effect against development of oral malignancy. Tobacco being major risk factor for oral malignancy, afford is made to estimate the difference in serum vitamin levels in tobacco consumers with different benign oral lesions and tobacco non-consumers.

METHODS

Retrospective study of 100 patients with benign oral lesion was included in this study. Patients were attended ENT OPD in Medical college hospitals in Mumbai between January 2013 to June 2014. Patient with tobacco addiction with benign oral lesions were selected. Control group was included the patients without tobacco addiction whose age and sex were matched.

The protocol of this study was approved by Ethics Committee of Institute. Consent was given by the patient for blood investigations.

Detail ear, nose, throat examination was done. Oral lesions were diagnosed clinically. Detail history of tobacco addiction was taken. Socioeconomic status of each patient was documented during history taking. Tobacco consumption pattern in respect to sex of patient, oral lesion and socioeconomic status was studied.

Inclusion criteria

Inclusion criteria were tobacco addicts patients with oral benign lesion; tobacco addict with occasional alcohol drinker; patients between 15 years and 60 years age group; tobacco addiction in form of tobacco smoking, tobacco chewing, sniff, Shisha smoking, etc.

Exclusion criteria

Exclusion criteria were patient with malignant oral lesion; patient below 15 years age; patients with diabetes mellitus and known hormonal disorders.

Fasting blood samples (5 ml) of the patients and controls were taken between 8 and 9 in the morning. Samples were centrifuged at 3000 rpm at 4°C for 5 min and aspirated into tubes. All samples were stored in deep freezer under -55^0 C temperature until analysed for vitamins A, E and C. The reagents were brought to room temperature for at least 30 min before use.

Serum vitamin A and E was estimated by the method of Baker et al.[5]

Vitamin E was extracted into n-heptane, which reacts with ferric chloride and reduces ferric to ferrous ions. Ferrous ions then forms a red colored complex with 2, 2 dipyridyl which was read at 520 nm. A correction for ß carotene was made after reading vit E at 520 nm. ß carotene was determined in mg% by multiplying absorbance at 460 nm by 856 factor.

Method proposed by Ayekyaw was used to estimate serum vitamin C.[6]

Vitamin C from serum reacts with phosphotungstate to give a blue color that has maximum absorbance at 600 nm.

Data are expressed as mean ± standard deviation. Student t-Test was applied for statistical data analysis.

RESULTS

Male and female distribution in the study group

In present study, males were more prone to develop benign oral lesions than females. Males were addict to tobacco than females. Also Indian cultural practices such as application of lime to tobacco, Quit which includes pan with tobacco, gutkha and betal nut are more common in males. Tobacco use by males is socially accepted (Figure 1).

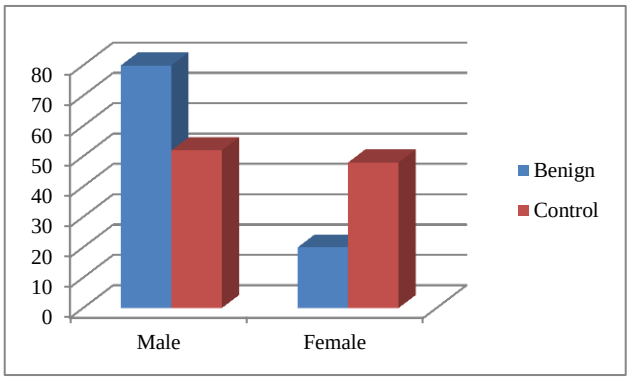

Figure 1: Male and female distribution in the study group.

Pattern of tobacco consumption

Tobacco with lime application is more common habit observed in Indian patients with benign oral lesions followed by tobacco chewing and smoking both. Use of tobacco in different forms such as gutkh, mawa, electronic cigarettes is increased because of advertisement in young population (Table 1).

Table 1: Tobacco consumption pattern.

Habit	No. of cases
Tobacco chewing	25
Tobacco+lime	48
Tobacco+pan+betalnut	08
Gutka	18
Mawa	06
Tobacco chewing+smoking	30
Smoking	15

Benign oral lesions seen in tobacco consumers

In present study, most common oral lesion was gingivitis followed by leukoplakia. Stomatitis and sub mucus oral fibrosis were other oral lesion seen. In present study, no patient was seen with erythroplakia. Tobacco addiction in the form of smoking alone is decreased as compares to tobacco chewing. So cases of melanoplakia were comparatively less in the present study (Figure 2).

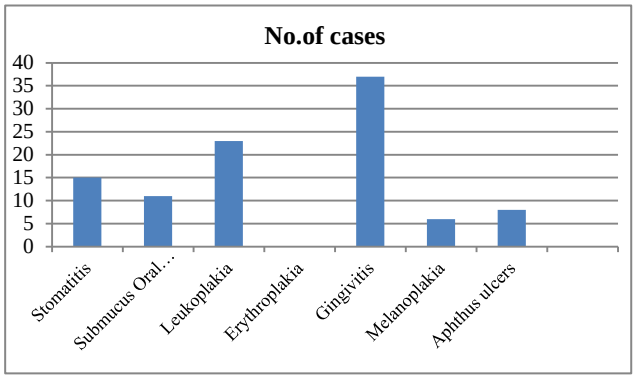

Figure 2: Benign oral lesions seen in tobacco consumers.

Comparison of vitamin levels in the benign lesion and control

Statistically significant ($p<0.01$) difference in serum vitamin levels was found in tobacco consumers with benign oral lesions and control group with non-tobacco consumers (Table 2).

Table 2: Vitamin levels in the benign lesion and control.

Mean (SD)	Vit A micromol/L	Vit E micromol/L	VitC micromol/L
Oral lesions	1.07±0.12	11.11 ± 0.64	11.02 ±1.10
Control	1.28±0.07	22.71±2.54	14.80 ±4.50
t values	t=15.04	t=44.06	t=8.11
P value	p<0.01	p<0.01	p<0.01

DISCUSSION

Depletion of non-enzymatic endogenous antioxidants can be detected in the pre-symptomatic phase of many diseases due to increased oxidative stress and elevated free oxygen radical. Vitamin A, C and E are non-enzymatic antioxidants. Anti-oxidants such as vitamin E is used to eliminating lipid peroxide radicals and vitamin E is also used in regeneration of vitamin C.[7] In diseased states and in case of stress the serum levels of these vitamins decreased. These vitamin levels can be used as biomarkers of malignant transformation of benign oral lesions and also indicators of dietary increased intake of these vitamins.

Tobacco consumption leads to increased oxidative stress and depletion of non-enzymatic antioxidants. As tobacco consumption is leading preventable cause of oral malignancy, serum vitamin levels were compared with benign oral lesions some of which are malignant potential in tobacco consumers and non-tobacco consumers.

In present study, males were prone for benign oral lesions. As tobacco consumption is accepted socially for males, more males were consuming tobacco in Indian population. Similar results were seen in study done by Neville, et al and Agrawal, et al.[8,9] Study done by Mathew, et al had shown that 2% visitors at dental college, Manipal had oral submucus fibrosis with male dominance between 40 to 60 years age group.[10] In controversy, Study done by Allon, et al had shown equal gender distribution in benign oral mucosal tumors.[11]

In the present study, the commonest habit seen for tobacco consumption was tobacco with lime chewing followed by tobacco chewing and smoking both. As tobacco with lime application is common habit observed in study population. Application of lime damage the integrity of oral mucosa and chances of oral lesions were increased. As per study done by Sujatha, the commonest habit seen was smoking, 39.2% followed by smokeless tobacco use 28.1%.[12]

Aljabab, had shown tobacco smoking (69%) as most common tobacco consumption pattern in Aljouf province of Saudi Arabia, followed by smokeless tobacco (31%).[13]

In present study, most common oral lesion was gingivitis followed by leukoplakia. Stomatitis and sub mucus oral fibrosis were other oral lesion seen. According to Napier, et al the buccal mucosa is affected in 25% of cases, followed by the mandibular gingiva in 20% cases. Other oral lesions were seen in rest 35% cases.[14] As per study done by Sujatha, 40% subjects with tobacco user had no clinically detectable changes in their oral mucosa.[12] Leukoplakia was commonest mucosal lesion seen in 14% tobacco users.[12] Oral leukoplakia is seen in the middle and older age group people. 90% oral leukoplakia were seen in tobacco/areca nut users.[15] Leukoplakia was commonest mucosal lesion seen in 14% tobacco users.[12] Study done by Chandra, et al had divided patients into groups based on their tobacco habits and oral mucosal lesions were studied.[16] Oral mucosal lesions were seen two third of the tobacco chewers.[16]

Study done by Baig, et al had shown that Oral lesions caused by constant exposure to Gutkha were associated with HPV infection in most of the cases. Study done by Baig, et al had shown that 78% gutkha chewers with oral lesions. According to Baig, et al, oral ulcers was seen in 25%, rough mucosa was seen in 62%, sub-mucosal fibrosis was seen in 24%, leukoplakia was seen in 20% and erythroplakia was seen in 10.6% of Gutkha chewers.[17]

Study done by Agrawal, et al had shown that Benign lesions were the common Oral Lesions.[9] According to Agrawal, et al tongue was most commonly affected 29.32% followed by tonsil in 22.56% cases, buccal mucosa in 20.32% cases, floor of mouth in 10.53% cases, palate in 9.02% cases, lower lip in 6.02% cases, upper lip in 1.50% cases and vestibule in 0.75% cases.[9]

Vitamin A, C and E are free radical scavengers which protects the cell against toxic free radicals. In this study, vitamin A, C and E levels in the patients having benign oral lesions were significantly less when compared to control. Other reason being vitamin E used by body for prostaglandin production.[18,19] Prostaglandins plays important role in inflammation. In a study done by Krajcovicova et al had shown decreased values of Vitamin C and Vitamin E levels.[20] Decreased levels of vitamin C were also observed in a study done by Nidarsh et al.[21] el Attar had shown dose dependent effect of vitamins C and E on prostaglandin synthesis which plays major role in inflammatory process.[22]

In present study, serum levels of vitamin A, C and E were decreased significantly in all patients irrespective of diagnosis of benign oral condition.

CONCLUSION

Gingivitis was most common lesion seen in study population followed by leukoplakia. Serum level of Non-enzymatic antioxidant vitamin such as vitamin A, C and E gets decreased significantly in all benign oral lesions irrespective of its malignant potential. Serum levels of the antioxidant vitamins indicate need of increased oral intake of these vitamins to protect from damage in all benign oral lesions.

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REFERENCES

1. Porter SR, Scully C, Pedersen A. Recurrent aphthous stomatitis. Crit Rev Oral Biol Med. 1998;9:306–21.
2. Scully C, Gorsky M, Lozada-Nur F. The diagnosis and management of recurrent aphthous stomatitis:A consensus approach. J Am Dent Assoc. 2003;134:200–7.
3. Trivedy CR, Craig G, Warnakulasuriya S. The oral health consequences of chewing areca nut. Addict Biol. 2002;7:115-25.
4. Machuca G, Rosales I, Lacalle JR, Machuca C, Bullon P. Effect of cigarette smoking on periodontal status of healthy young adults. J Periodontol. 2000;71:73-8.
5. Baker H, Frank O. Determination of serum αtocopherol. In: Gowenlock, McMurry. Varley's Practical Clinical Biochem; London. 6th ed; 1968: 902-903.
6. Ayeqaw. A simple colorimetric method for ascorbic acid determination. Clinica Chem Acta. 1978;86:153-7.
7. Kline K, Yu W, Sanders B. Vitamin E and Breast Cancer. J Nutr. 2004;134:3458S–62S.
8. Neville B, Damm D, Allen C, Bouquot J. Oral and Maxillofacial Pathology. 3rd edition. St. Louis, Mo, USA: Saunders/Elsevier; 2009.
9. Agrawal R, Chauhan A, Kumar P. Spectrum of Oral Lesions in A Tertiary Care Hospital. J Clin Diagno Res. 2015;9(6):EC11-3.
10. Mathew AL, Pai KM, Sholapurkar AA, Vengal M. The prevalence of oral mucosal lesions in patients visiting a dental school in Southern India. Indian J Dent Res. 2008;19(2):99–103.
11. Allon I, Kaplan I, Gal G, Chaushu G, Allon DM. The clinical characteristics of benign oral mucosal tumors. Med Oral Patol Oral Cir Bucal. 2014;19(5):e438-43.
12. Sujatha D, Hebbar PB, Pai A. Prevalence and correlation of oral lesion among tobacco smokers, tobacco chewers, areca nut and alcohol users. Asian Pac J Cancer of Prev. 2012;13:1633-7.
13. Aljabab MA, Aljbab AA, Patil SR. Evaluation of Oral Changes Among Tobacco Users of Aljouf Province, Saudi Arabia. J Clin Diagnos Res. 2015;9(5):ZC58-61.
14. Napier SS, Speight PM. Natural history of potentially malignant oral lesions and conditions: an overview of the literature. J Oral Pathol Med. 2008;37(1):1–10.
15. Suarez P, Batsakis JG, El-Naggar AK. Leukoplakia: still a gallimaufry or is progress being made?—a review. Adv Anatomic Pathol. 1998;5(3):137–55.
16. Chandra P, Govindraju P. Prevalence of oral mucosal lesions among tobacco users. Oral health Prev Dent. 2012;10:149-53.
17. Baig S, Lucky MH, Qamar A, Ahamad F, Khan S, Ahmed W, et al. Human papilloma virus and oral lesions in gutka eating subject in Karachi. J Coll Physicians Surg Pak. 2012;22:135-8.
18. Wang Y, Huang DS, Eskelson CD, Watson RR. Long term dietary vitamin E retards development of retro virus-induced disregulation in cytokine production. Clini Immunol Immunopathol. 1994;72:70-5.
19. Omach EH, Kidao S, Sanders BG, Kline K. Effects of RRR-α-tocopherol suucinate on IL-1 and PGE2 production macrophages. Nutr Cancer. 1993;20:205-14.
20. Krajcovicova KM, Paukova V, Bacekova M, Dusinka M. Lipid peroxidation in relation to vitamin C and vitamin E levels. Cent Eur. J Publ Health. 2004;12(1):46-8.
21. Nidarsh H, Sucheta K, Amita H, Prasanna C, Neeriksha R. Lipid peroxidation and vitamin C levels in saliva of oral precancerous patients an invitro study. RJPBCS. 2011;2(2):13-8.

22. el Attar TM, Lin HS. Effect of vitamin C and vitamin E on prostaglandin synthesis by fibroblasts and squamous carcinoma cells. Prostaglandins Leukot Essent Fatty Acids. 1992;47(4):253-7.

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