

Assessment of Role of Oral Lycopene in Patients with Oral Leukoplakia

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ABSTRACT

Background: Oral leukoplakia is a premalignant lesion caused by smoking or smokeless tobacco. The present study was conducted to assess role of oral lycopene in patients with oral leukoplakia.

Materials & Methods: 40 clinically/histopathologically proven cases of oral leukoplakia, and healthy subjects without any oral deleterious habits were also enrolled. Determination of lycopene in blood plasma by high performance using liquid chromatography.

Results: Out of 60 subjects, males were 40 and females were 20. Group I had non- smokers, group II had smokers without leukoplakia and group III had smokers with leukoplakia. Each group had 20 subjects. The mean lycopene level in group I was 132.8 ng/ml, group II had 40.2 ng/ml and group III had 29.6 ng/ml. The difference was significant ($P < 0.05$).

Conclusion: The level of lycopene was highly reduced in patients with leukoplakia as compared to healthy subjects.

Keywords: Leukoplakia, Lycopene, Smoking.

INTRODUCTION

Oral leukoplakia (OL) is a premalignant lesion described as "a predominant white lesion of the oral mucosa which cannot be defined as any other known lesion." According to WHO 1997 Leukoplakia is defined as "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion." It is now categorized under potentially malignant disorders. It is now categorized under potentially malignant disorders.¹ Malignant potential of leukoplakia was hinted by Sugar and Banoczy way back in 1957. Association between tobacco chewing and smoking with oral leukoplakia is established beyond doubt. Tobacco smoke contains NOO radicals, which are carcinogenic. Free radical

scavengers should be the necessary part of the treatment regimen in tobacco chewers or smokers to prevent the formation, induce the remission or inhibit the progression of pre-cancerous lesions into malignancies.²

According to Warnakulasuriya et al³, the new concept of OL shall acknowledge white lesions with questionable risk of being an OL, being excluded any other pathologies or known disorders which do not present potential malignant risk such as candidiasis, lupus erythematosus, lichen planus, hairy leukoplakia, frictional keratosis, nicotinic stomatitis, and leukoedema. Nonsurgical treatment may also be considered for the management of OL. This modality offers minimal adverse effects to patients,

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especially for patients with widespread OL that involves a large area of the oral mucosa or patients with medical problems and, consequently, high surgical risks. Lycopene is a carotenoid without provitamin A action. This is a fat-soluble red pigment found in some fruit and vegetables. The greatest known source of lycopene is tomatoes, which are widely employed in cooking.⁴ The present study was conducted to assess role of oral lycopene in patients with oral leukoplakia.

MATERIALS & METHODS

The present study comprised of 40 clinically/histopathologically proven cases of oral leukoplakia, Healthy subjects without any oral deleterious habits were also enrolled. Diagnosis of oral leukoplakia was made on the basis of history and clinical features as homogenous and non-homogenous which includes speckled, nodular, verrucous or proliferative verrucous leukoplakia. Histopathological grading was done according to severity of dysplasia as follows: Mild/Moderate/Severe epithelial dysplasia or Carcinoma in situ. Determination of lycopene in blood plasma by high performance using liquid chromatography. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I: Distribution of patients.

Total- 60		
Gender	Males	Females
Number	40	20

Table II: Distribution on the basis of smoking.

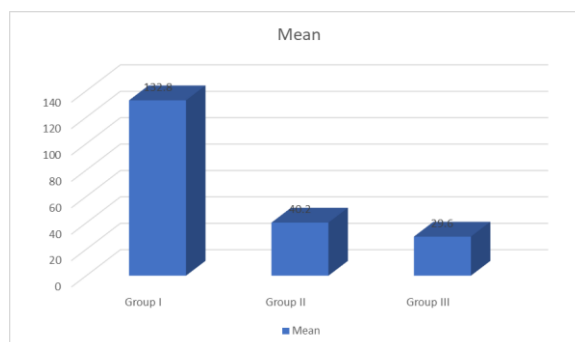
Group I	Group II	Group III
Non smokers	Smokers without leukoplakia	Smokers with leukoplakia
20	20	20

Table III: Assessment of plasma lycopene level.

Group	Mean	P value
Group I	132.8	0.02
Group II	40.2	
Group III	29.6	

Table III, graph I shows that mean lycopene level in group I was 132.8 ng/ml, group II had 40.2 ng/ml and group III had 29.6 ng/ml. The difference was significant ($P < 0.05$).

Graph I: Assessment of plasma lycopene level.



DISCUSSION

Leukoplakia is the most common pre-cancerous lesion in the oral cavity. Association between tobacco chewing and smoking with oral leukoplakia is established beyond doubt. Tobacco smoke contains NOO radicals, which are carcinogenic.⁵ Antioxidants especially lycopene exhibits highest physical quenching rate constant with singlet oxygen. Lycopene has been found to be least 3-fold more effective than β -carotene in preventing cell death by quenching of NOO radicals. It also protects DNA damage induced by 1-methyl 3-nitro-1-nitrosoguanidine and H₂O₂.⁶

Lycopene has the unusual feature of becoming bound to chemical species that react to oxygen, thus being the most efficient biological antioxidantizing agent.⁷ Due this property, studies have been actively conducted with lycopene, to find out whether or not it could be an alternative to protect patients against the damaging effects of free radicals. In addition to its antioxidantizing property, lycopene also has the capacity to modify intercellular exchange junctions, and this is considered to be an anticancer mechanism.⁸ In vitro experiments have shown the inhibition of the process of human neoplastic cellular growth by lycopene, since this protein interferes in growth factor receptor signaling and, thus, in cellular cycle progression, as previously demonstrated for neoplastic cells from the prostate gland.⁹ The present study was conducted to assess role of oral lycopene in patients with oral leukoplakia.

In present study, out of 60 patients, males were 40 and females were 20. We found that group I had non- smokers, group II had smokers without leukoplakia and group III had smokers with leukoplakia. Each group had 20 subjects. Shivakumar et al¹⁰ in their study a total of 180 individuals were included in the study with two main groups namely cases and controls: clinically/ histopathologically proven cases of oral leukoplakia (60) and age and sex matched control group divided into two categories nonsmokers (60) and smokers without leukoplakia (60). The age of the patients varied between 31-70 years. Mean plasma lycopene levels for the leukoplakia patients (smokers) was 32.86 ± 21.42 ng/ml, and for controls who were smokers it was 42.70 ± 26.54 ng/ml, however the mean levels for non-smokers was 146.6 ± 52.45 ng/ml which was significantly more.

We found that mean lycopene level in group I was 132.8 ng/ml, group II had 40.2 ng/ml and group III had 29.6 ng/ml. Panwar et al¹¹ found that the age ranged between 32-65 years. In the study group out of 30 patients of leukoplakia 16 patients were with clinically homogenous variety of leukoplakia and 14 with the speckled variety. Histopathologically out of 30 patients 13 had mild dysplasia and 17 with moderate dysplasia. The mean plasma lycopene level values were 137.4 ± 62.745 , 40.07 ± 23.956 and 29.46 ± 17.497 in non- smokers, smokers without leukoplakia and smokers with leukoplakia respectively. Plasma lycopene level was significantly higher in non-smokers than smokers without leukoplakia and smokers with leukoplakia at 99% confidence level but there is non-significant difference in smokers without leukoplakia and smokers with leukoplakia.

Nagao T et al¹² in their study found that the mean serum lycopene and β -carotene levels among male cases were significantly lower than those detected for controls. The results demonstrated that high serum levels of β -carotene and lycopene reduce the risk of leukoplakia in males. In their study this was confirmed in the bivariate analysis but the significance of lycopene was not apparent in the logistic regression analysis.

CONFLICTS OF INTEREST

The authors declare they have no potential conflict of interests regarding this article.

CONCLUSION

Authors found that the level of lycopene was highly reduced in patients with leukoplakia as compared to healthy subjects.

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