ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Status of Serum Vitamin C Level and Lipid Peroxidation in Smokers and Non Smokers with Oral Cancer

Nidarsh D Hegde[1]*, Suchetha Kumari[2], Mithra N Hegde[3], Mahesh Bekal[4], Priyanka Rajaram[5].

[1] Department of Oral and Maxillofacial Surgery, A.B.S.M.I.D.S, Nitte University, Mangalore, Karnataka, India.
[2] Department of Biochemistry, KSHEMA, Nitte University, Mangalore, Karnataka, India.
[3] Department of Conservative dentistry and Endodontics, A.B.S.M.I.D.S, Nitte University, Mangalore, Karnataka, India.
[4] Research Scholar, Central Research Laboratory, Nitte University, Mangalore, Karnataka, India.
[5] Department of Biochemistry, St. Aloysius College, Mangalore, Karnataka, India.

ABSTRACT

Free radical induced lipid peroxidation causes a loss of cell homeostasis by modifying the structure and functions of cell membrane. The most important characteristic of lipid peroxidation is to cause a considerable DNA-MDA adducts by interacting with cellular DNA. Lipid peroxidation by products formed under physiological and pathological conditions are scavenged by non enzymatic and enzymatic antioxidants. Mammalian cells possess elaborate antioxidant defence mechanisms to neutralize the deleterious effects of free radical induced lipid peroxidation. An imbalance between antioxidant defence mechanism and lipid peroxidation processes results in cell and tissue damage. This study was conducted on 50 patients reporting to the Dept of Oral Medicine and Radiology, A B Shetty Memorial Institute of Dental Sciences, Deralakatte, Mangalore. 5ml of blood samples of the oral cancer patients was collected by vein puncture, centrifuged to separate the serum and stored at 4^0C. Serum was tested for the level of Malondialdehyde by TBA method and was tested for level of Vitamin C by DNPH method. Data obtained were statistically analyzed by Student't' test. In this study MDA level was found to be increased significantly (P=0.05) in smokers with oral cancer when compared to that of non smokers with oral cancer. Thus MDA is involved in carcinogenesis and tumor progression. But vitamin C level in serum found to be insignificant may be due to diet of the patients.

Keywords: Lipid Peroxidation, MDA, Oral Cancer, Vitamin C

Corresponding author
Email: drhegdedental@gmail.com

ISSN: 0975-8585

INTRODUCTION

Oral cancer is a cancer found in the oral cavity and oral pharynx**.** The biggest risk factor for developing oral cancer has traditionally been smoking. Smokers are six times more likely than nonsmokers to develop mouth cancer. Approximately 90% of the people with mouth cancer are tobacco users. When burnt, cigarette smoke contains over 4000 chemicals, with over 40 of them being known carcinogens. However recent statistics indicate oral cancers in non-smokers is the fastest growing category [1, 2].

Cancer causing agents (carcinogens) in tobacco smoke damage important genes that control the growth of cells, causing them to grow abnormally or to reproduce too rapidly. Tobacco contains nicotine, which is a highly addictive psychoactive chemical. When tobacco is smoked, nicotine causes physical and psychological dependency. Cigarette smoke also contains large amounts of pro-oxidants that can directly initiate the process of lipid peroxidation, as well as deplete the body of nutrient antioxidants [3-6].

Lipid peroxidation refers to the oxidative degradation of lipids. It is a chain reaction process producing a continuous supply of free radicals/ lipid radicals, lipid alkoxy radical, lipid pereoxy radicals, which are themselves very reactive and initiates further peroxidation which lead to extensive cellular damage. Free radical induced lipid peroxidation causes a loss of cell homeostasis by modifying the structure and functions of cell membranes. In addition ,end products of lipid peroxidation may be mutagenic and carcinogenic .The end-product malondialdehyde (MDA) reacts with deoxyadenosine and deoxyguanosine in DNA forming DNA-MDA adducts .Mammalian cells poses elaborate antioxidant defense mechanisms to neutralize the deleterious effects of free radical induced lipid peroxidation [1, 2].

Patients with oral cancer show elevated levels of lipid peroxidation accompanied by antioxidant depletion [7]. An alteration in lipid peroxidation with concomitant changes in antioxidant defense system in cancer patients may be due to excessive oxidative stress [9].

Vitamin C plays a vital role in antioxidant defense. It acts as a scavenger of free radicals and impedes the detrimental chain reactions triggered by the free radicals which would otherwise culminate in tissue damage leading to oxidative stress [10]. It has been said that antioxidant nutrients may be utilized to a greater extent in oral cancer patient to counteract free radical mediated cell disturbances, resulting in a reduction in antioxidant level [9].

METHODS AND MATERIALS

This study was conducted in the Central Research Laboratory of Nitte University after obtaining the approval from the institutional ethical committee.

Study group

Include total of 50 subjects, consisting of 25 smokers and 25 nonsmokers with oral cancer reporting to the Department of Oral Medicine and Radiology.

Exclusion criteria

- Subjects with systemic diseases.
- Subjects under medication / antioxidant supplementation.

Inclusion criteria

- Clinically diagnosed and histopathologically confirmed cases of oral cancer.
- Smokers and nonsmokers with oral cancer.

A detailed case history of the patient was taken. A case history format was filled, with a Informed consent which was duly signed by each patient.

Collection of serum

5ml of blood samples of the subjects was collected by vein puncture, centrifuged to separate the serum and stored at 4^0C.

Estimation of Malondialdehyde

Serum was tested for the level of Malondialdehyde by TBA method. Thiobuteric acid reacts with malondialdehyde (end product of lipid peroxidation) to yield a fluorescent product, which is read spectrophotometrically at 546nm.

Estimation of Vitamin C

Serum was tested for the level of level of Vitamin C by DNPH method. The intensity of the colour is measured in a spectrophotometer at 540nm.

Statistical analysis:

Results are presented as mean $\pm$ standard deviation value and statistically analyzed by Student't' test. A 'p' value of 0.05 or less was considered significant.

RESULTS

The present study involves the estimation of the levels of lipid peroxidation product malondialdehyde and Vitamin C levels in Oral Cancerous condition and their comparison

ISSN: 0975-8585

between smoking and non-smoking individuals. The results are expressed in Table-1, Fig-1 and 2.

Table 1: Comparison of serum MDA and vitamin C levels in smokers and non smokers with oral cancer

	ORAL CANCER		
	SMOKERS	NON-SMOKERS	P VALUE*
LIPID PEROXIDATION (MDA)	1.520±0.9	0.450±0.18	0.05
VITAMIN C	1.260±0.92	1.240±0.43	0.95

*P<0.05 is statistically significant. Statistical comparison were performed by Student 't' test. Data expressed as Mean±SD.

Figure1: Comparison of serum MDA levels in smokers and non smokers with oral cancer.

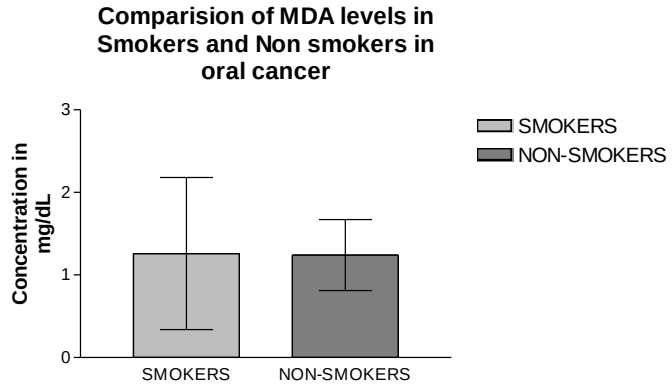
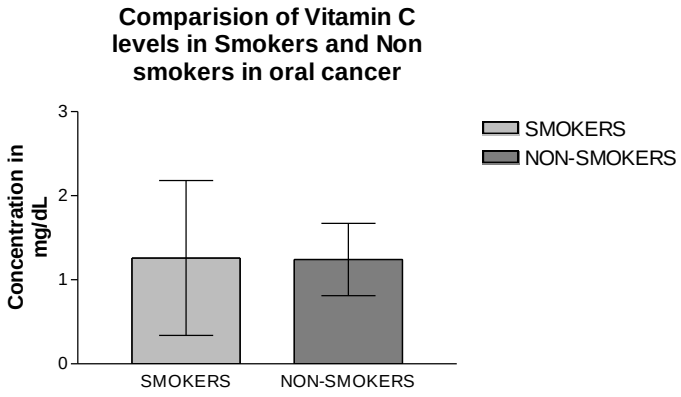


Figure2: Comparison of serum Vitamin C levels in smokers and non smokers with oral cancer



In this study MDA level was found to be increased significantly (P=0.05) in smokers with oral cancer when compared to non smokers with oral cancer. In this study Vitamin C level in smokers and non smokers with oral cancer is found to be insignificant (P=0.95).

DISCUSSION

Cigarette smoking is a serious health problem worldwide. Smoking has been strongly implicated as a risk factor for chronic obstructive pulmonary diseases and cancer. Cigarette smoke contains a large number of oxidants [13]. Many of the adverse effects of smoking may result from oxidative damage to critical biologic substances. Free radical induced lipid peroxidation causes a loss of cell homeostasis by modifying the structure and functions of cell membrane. The most important characteristic of lipid peroxidation is to cause a considerable DNA-MDA adducts by interacting with cellular DNA. Lipid peroxidation by products formed under physiological and pathological conditions are scavenged by non enzymatic and enzymatic antioxidants. Mammalian cells possess elaborate antioxidant defence mechanisms to neutralize the deleterious effects of free radical induced lipid peroxidation. An imbalance between antioxidant defence mechanism and lipid peroxidation processes results in cell and tissue damage [2, 14]. Hence elevated levels of serum malonialdehyde are usually seen in oral cancer group [7, 16].

In this study MDA levels is found to be increased significantly in oral cancer patients when compared with healthy subjects. The levels of MDA were higher in tobacco users than non users.

These high levels of serum MDA in oral cancer patients directly reflects increased oxidative stress and lipid peroxidation, which might be due to the interaction of various carcinogenic agents, generating free radicals to a greater extent in these patients beyond their defending power or may be due to poor antioxidant system existing in these individuals [2,16].

Vitamin C is an important non-enzymatic antioxidant. This free radical scavenger protects the cell against toxic oxygen radicals. Enhanced lipid peroxidation with concomitant decrease in antioxidants is indicative of oxidative stress that provides evidence of relationship between lipid peroxidation and oral cancer [6].

In this study vitamin C level in oral cancer patients and healthy subjects is found to be insignificant. This insignificant change may be due to high intake of Vitamin C through diet.

CONCLUSION

Our study showed that, the serum levels of MDA were higher in tobacco users than non users. This provides a compelling evidence that smoking causes oxidative modification of biologic components in humans i.e., lipid peroxidation .These findings indicate the role of free radicals in pathogenesis and cancer. Insignificance of Vitamin C level in oral cancer patients and

healthy subjects may be due to high intake Vitamin C through diet, as we had not controlled Vitamin C intake of the study group.

REFERENCES

[1] Manoharan S, Kolanjiappan K, Suresh K, Panjamurthy K. I J Med Res 2005; 122:529-534.
[2] Manoharan S, Baskar AA, Manivasagam T, Subramanian P. Singapore Med J 2005;46(4): 184.
[3] Nair UJ, Oshima H, Friesen M, Croisy A, Bhide SV, Bartisch H. Carcinogenesis 1985; 6:295-303.
[4] Subapriya R, Kumaraguruparan R, Nagini S, Thangavelu A. Toxicology Mechanisms and Methods 2003; 13:77-81.
[5] Sun Y. Free Radical Biology & Medicine 1999; 8(6):583-99.
[6] Syed Sultan Beevi S, Muzib Hassanal Rasheed A, Geetha A. Jpn J Clin Oncol 2004; 34(7):379–385.
[7] Khanna R, Thapa PB, Khanna HD, Khanna S, Khanna AK, Shukla HS. Medical Journal 2005; 3[4(12)]:334-339.
[8] Aebi H, Bergmeyer H. Methods in enzymatic analysis. New York Academic Press 1983; 276-286.
[9] Reza Mahdavi, Elnaz Faramarzi, Ensiyeh Seyedrezazadeh, Mohammad Mohammad zadeh and Masoud Pourmoghaddam E. Humana Press Inc., Friday, January 16, 2009.
[10] Ames NB. Proc Natl Acad Sci, USA 1993; 90:7915-22.
[11] Anissa KS, Sangita RP, Pramod SS, Ajit VS, Anup SH, Anand KG. I J Clin Biochem 2009; 24(3): 307-311.
[12] Balwant RA. Pesq Bras Odontoped Clin Integr 2008; 8(1):123-125.
[13] Balwant R, Simmi K, Rajnish J, Anand SC Adv Med Dent Sci 2008; 2(1):7-8.
[14] Church DF, Pryor WA. Environ Health Prespect 1985; 64:111-126.
[15] Kollanjiappan K, Prasad MPR, Krishna TP, Reddy GA. Eur J Cancer 1995; 31B:41-48.
[16] Beena P Patel, Upendra M Rawal, Tina KD, Rakesh MR, Shilin N, Shukla MD, Pankaj M, Shah MD, Prabhudas S Patel. Sage journals 2005.