www.jmscr.igmpublication.org Impact Factor 5.84

Index Copernicus Value: 83.27

ISSN (e)-2347-176x ISSN (p) 2455-0450

crossref DOI: https://dx.doi.org/10.18535/jmscr/v5i6.06



Original Article

A Study of Urea, Uric Acid and Creatinine Pathogenesis of Oral Submucous Fibrosis

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Abstract

Oral Submucous Fibrosis (OSMF) is a high risk precancerous condition, chronic debilitating disease of the oral cavity characterized by inflammation and preogressive fibrosis of the submucosal tissues (Lamina Propariaand Deepar Connective Tissues). Although nutritional deficiencies and immunological process may play an important role in the pathogenesis, this clinical study was conducted at Dental Department of Maharanilaxmi Bai Medical College in 20clinically diagnosed patients. No alteration was observed in serum uric acid, urea and creatinine levels in OSMF patients.

Keywords: Urea, Uric Acid, Creatinine.

INTRODUCTION

Oral sub-mucous fibrosis(OSMF) is a chronic, premalignant disease and characterized by epithelial inflammatory reaction. The main cause of the disease increase is the consumption of product such asgutkha, panmasala, khaini (made of areca nut). As the disease progresses, the jaws become inflexible to the point that the person is ineffective to open the mouth.

The role of areca nut in the pathogensis of OSMF has been studied in detail over last two decades. It apparent that fibrosis and hyalinization of subepithelial tissues account for most of the clinical features encountered in this condition. Moreover,

substantial amount of research appear to have been focused on changes in the extracellular matrix (ECM). It is logical to hypothesize that the increased collagen synthesis or reduced collagen degradation as possible mechanism in the development of the disease (Dyavanagoudar, 2009)¹.

It is a disease of unknown cause that occurs mainly in India (Singhet al, 2011; Sudarshanet al, 2012)² ³. The use of smokeless tobacco associated with oral cancer was pointed out as early as 1908. Subsequent Indian studies on tobacco have shown its association with major disease entities, both in smoking as well as in smokeless form. The habit of

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smokeless tobacco (also referred as tobacco chewing) is also very common. Association of smokeless tobacco has been observed with cancers of oral cavity, pharynx, larynex and oesophagus, and precancerous lesions of oral cavity (Niblock, 1902; Bhonsle et al, 1992)⁴⁵. The pathogenesis of the disease is still not clearly understood but it is usually considered to be an abnormal wound healing process and is often compared with fibrosis of other organs (Angadi et al, 2011)6. Fibrosis occurring in other organ like liver is associated with a number of biochemical changes which lead to structure and metabolic abnormalities. Impaired lipid metabolism abnormalities and consequent impaired or reduced cholesterol occurs in fibrosis and cirrhosis of liver (George and Chandrakasan, $2000)^{7}$.

On comparing OSMF with liver fibrosis, it can be hypothesized that biochemical parameters might also play a role in pathogenesis of the disease. However, to the best of our knowledge, no study has examined the role of urea, uric acid and creatinine in the aetiology of oral cancer. Very little information is available about the biochemical abnormalities and changes in metabolic parameters. Hence, in the present investigation, it was aimed to analyze the urea, uric acid and creatinine levels in oral cancer patients.

MATERIALS AND METHODS

The present study was carried out in the Department of Biochemistry, MLB Medical College, Jhansi, Utter Pradesh. Clinical details were retrieved from the Dental Department of MLB Medical College over a period of 5 months. After proper evaluation of the records, a total of 20 cases of clinically diagnosed OSMF were considered for the study. Clinical detail included name, age, gender and without any history of habit of chewing are canut and tobacco as well as any major illness in recent past was included as controls. The uric acid present in serum was determined by the method of uric acid test, creatinine present serum was determined by creatinine test kit and urea present serum was

determined by urea test kit and all data levels measuring by colorimeter.

RESULT

Half of patients (50%) were above 40 years followed by 30-40 (30%) and <30 (20%) years. The mean age of patients was a 38.65 \pm 10.24 year ranging from 22 to 60 years. Majority of patients were males (70%).

Table-1: Age and sex distribution of patients

| | No. | % |
|-----------------|---------------------|------|
| | (n=20) | |
| Age in years | | |
| <30 | 4 | 20.0 |
| 30-40 | 6 | 30.0 |
| >40 | 10 | 50.0 |
| Mean±SD (Range) | 38.65±10.24 (22-60) | |
| Sex | | |
| Male | 14 | 70.0 |
| Female | 6 | 30.0 |

The mean uric acid, creatinine and urea were 5.10 ± 1.31 , 1.17 ± 0.61 and 37.40 ± 7.93 respectively

Table-2: Distribution of patients according to biochemical parameters

| Biochemical parameters | Mean±SD |
|------------------------|------------|
| Uric acid (mg/dl) | 5.10±1.31 |
| Creatinine (mg/dl) | 1.17±0.61 |
| Urea (mg/dl) | 37.40±7.93 |

DISCUSSION

The harmful habits such as use of tobacco intake both in smoking and smokeless form, pan masala and gutkha chewing as well as product which contain areca nut are the main causative agent for premalignant disorders. In the literature, from Indian subcontinent purview, use of areca nut is the most etiologic common agent (Joseph and George, 2015)8. Hence, we evaluated the serum levels of urea, uric acid and creatinine in OSMF patients. This study showed that serum uric acid, urea & creatinine were altered in OSMF patients. This shows that changes in biochemical values do occur in the premalignant state of the body. Joseph et al (2015)⁸ also found non-significant alteration in the creatinine, phosphokinase levels in oral OSMF patients in fibrosis cases.

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Due to reduced nutrient flow, there is reduction in blood supply in the region to the region just as in systemic sclerosis as demonstrated by Partovi et al (2012)⁹, over a period of time there can be complete fibrosis and loss of activity of the muscle leading to improper nutrition intake, malnutrition and therefore decrease in standard and quality of life. Serum uric acid was lowered in oral cancer patients when compared with healthy controls and this low serum uric acid was associated with increased risk of oral cancer development. The lowering of serum uric acid in oral cancer patient may be due to tumour necrosis factor (TNF) and interleukin-6 produced in patient which result in loss of appetite and malnutrition (Singh et al, 2011; Sudarshan et al, 2012)10 11.

CONCLUSION

From this study, it is suggested that the biochemical analysis can be helpful in masses required to find out the exact role which these parameters play in the pathogenesis of OSMF.

REFERENCES

- Dyavanagoudar SN .Oral Submucous Fibrosis: Review On Etiopathogenesis. J Cancer SciTher: 2009; (2):072-071.
- Singh L,Bharti SS, Sudhapallis, Chopra D, Srivastava V.International Journal Of Clinical And Dental Science, 2011; 2(4), 29-35.
- 3. Sudarshan R, Annigeri RG, Sree VG. Pathogenesis of Oral Submucous Fibrosis: The Past And Current Concept, International Journal Of Oral And Maxillofacial Pathology, 2012; 3(2), 27-36.
- 4. Niblock WJ. Cancer InIndia.Indian Medical Gazette, 1902; 37,161-165.
- Bhonsle RB,Murti PR And Gupta PC.Tabacco HabitsIn India.In:Gupta PC &Hamner JE III (Eds.), ControlOf Tobacco Related Cancer And Other Disease. International Symposium, 1990. Oxford University Press, Bombay,P25-46,1992.

- 6. Angadi PY, Kale AD ,Hallikerimath S. Evaluation Of Myofibroblasts In Oral Submucous Fibrosis :Correlation With Disease Severity .J Oral Pathol Med. 2011; 40(3): 208-13
- 7. George J, ChandrakasanG. Biochemical Abnormalities During The Progression Of Hepatic Fibrosis Induced By Dimethylnitrosamine. Clinic Biochem 2000; 33(7):563-570.
- 8. Joseph BB, George S. Level of Serum Creatine Phosphokinase in Oral Submucous Fibrosis, A Biochemical Study .Int J Cur Rev.2015; 7(13):74-78.
- Partovi Sasan, Anja Carina Schulte, Impaired SkeletAl., Muscle Microcirculation Ic Systemic Sclerosis, Arthritis Research & Therapy 2012,14:R209
- Singh L,Bharti SS, Sudhapallis, Chopra D, SrivastavaV. International Journal Of Clinical And Dental Science, 2011; 2(4), 29-35.
- 11. Sudarshan R, Annigeri RG, Sree VG. Pathogenesis Of Oral Submucous Fibrosis: The Past And Current Concept, International Journal Of Oral And Maxillofacial Pathology, 2012; 3(2), 27-36.