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Original Research

Exploring Serum Uric Acid Levels in Individuals Affected by Oral Squamous Cell Carcinoma: A Cross-Sectional Investigation

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ABSTRACT:
Introduction- This cross-sectional study delves into the investigation of serum uric acid levels among individuals diagnosed with oral squamous cell carcinoma (OSCC). The research aims to shed light on the potential correlation between serum uric acid levels and the occurrence of OSCC. By employing a cross-sectional design, we aim to analyze and interpret the data to contribute valuable insights into the understanding of OSCC and its potential biochemical associations. **Materials and methods**- The study involved the recruitment of 70 subjects, with the study group consisting of patients clinically diagnosed with oral squamous cell carcinoma (OSCC) (n=45), alongside 25 individuals serving as healthy controls. **Results**-Oral squamous cell carcinoma (OSCC) exhibited a predominant diagnosis in males, comprising 53% of the cases. The occurrence of oral lesions was notably associated with tobacco habits and the chewing of betel quid/areca nut. **Conclusion**- The serum uric acid levels displayed a reduction in oral squamous cell carcinoma (OSCC) patients, when compared to healthy controls.
Keywords: Carcinoma, Uric acid, Alcohol

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INTRODUCTION

Oral squamous cell carcinoma (OSCC) stands out as the predominant form of oral malignancy, constituting approximately 80-90% of all malignant neoplasms affecting the oral cavity. While the incidence of oral cancer varies globally, the oral cavity typically ranks between the 6th and 9th most common location for cancer, influenced by geographical location and gender.[1] In specific regions, particularly in southeastern Asia, it can emerge as the primary site for cancer. The primary culprits behind OSCC are largely attributed to lifestyle choices, with smoking and alcohol consumption being a major etiological factors.[2] additionally, ultraviolet radiation, particularly linked to lip cancer, serves as a contributing factor. Other factors, such as infections from human papillomavirus (HPV) and Candida, nutritional deficiencies, and genetic predisposition, have also been associated with OSCC. This form of cancer predominantly affects adults and the elderly, presenting most commonly as an ulcerated lesion characterized by a necrotic central area surrounded by elevated rolled borders.

It is crucial to recognize the diverse factors contributing to OSCC to develop comprehensive prevention and treatment strategies. The primary drivers behind the development of oral squamous cell carcinoma (OSCC) are lifestyle factors, with the most prominent being tobacco use, alcohol consumption, and the habit of chewing areca nut or betel quid. These behaviours are widely recognized as major contributors to OSCC.[3,4]Additionally, the sexually acquired human papillomavirus (HPV) plays a role, albeit to a lesser extent, in contributing to the risk of developing OSCC. Its significance is highlighted by its responsibility for approximately 60% of free radical scavenging activity in humans .A notable attribute of uric acid is its ability to form a stable nitric oxide donor through interaction with peroxynitrite. [5]This interaction results in increased vasodilatation, contributing to a reduction in peroxynitrite-induced oxidative damage .In instances of heightened oxidative

stress, where cells are vulnerable to damage, antioxidants play a pivotal role in neutralizing oxidants to alleviate oxidative stress within the cell .

Uric acid, being a major antioxidant in human plasma, is implicated in both correlating with and predicting the development of conditions such as obesity, hypertension, and cardiovascular disease—conditions associated with oxidative stress. Consequently, uric acid may play a preventive role in cancer by mitigating cellular and genetic injuries. In the context of this assessment, uric acid was chosen deliberately due to its status as a primary endogenous antioxidant that can also be obtained exogenously.[6,7] Its involvement in redox reactions and protective action against oxidation make it a pertinent subject of study. Correlating uric acid levels with clinical and histopathological data provides valuable insights into its role. Moreover, the cost-effectiveness of uric acid testing makes it a viable option for mass screening, especially if its correlation with the disease process is established.

This study was designed to assess and correlate the uric acid levels in the serum of patients diagnosed with OSCC and to compare these to healthy controls.

MATERIALS AND METHODS

The study involved the recruitment of 70 subjects, with the study group consisting of patients clinically diagnosed with oral squamous cell carcinoma (n=45), alongside 25 individuals serving as healthy controls. A comprehensive medical history was obtained from each participant to validate their eligibility for enrollment in the study. The following criteria were applied for participant inclusion:

OSCC Patients (Study Group, n=45) - Clinical diagnosis of oral squamous cell carcinoma and willingness to participate in the study. Healthy Controls (n=25) - Absence of any clinically diagnosed oral squamous cell carcinoma or other significant oral health issues and willingness to participate in the study. For both groups: Age and Gender: - Inclusion criteria involved adults meeting the specified age range. Both genders were considered eligible for participation. These criteria were meticulously applied to ensure a representative and relevant study population, allowing for a meaningful investigation into the factors associated with oral squamous cell carcinoma.

A fasting blood sample of 10 mL was collected for standard clinical biochemistry, utilizing the uricase method to measure serum uric acid levels from non-hemolyzed blood samples, as previously described. In the course of this study, the subjects underwent comprehensive oral examinations subsequent to the acquisition of detailed patient histories, encompassing social habits such as tobacco, betel quid/areca nut, and alcohol use. As part of the overall patient management process, an incisional biopsy was performed, and subsequent histopathological evaluations were conducted. This multifaceted approach allowed for a thorough understanding of the clinical, social, and histological aspects, contributing to a comprehensive assessment of the factors associated with oral squamous cell carcinoma.

RESULTS

Oral squamous cell carcinoma (OSCC) exhibited a predominant diagnosis in males, comprising 53% of the cases. The occurrence of OSCC was notably associated with tobacco habits and the chewing of betel quid/areca nut. Additionally, alcohol consumption was significantly reported among patients with OSCC. Remarkably, the buccal mucosa emerged as the most frequently affected anatomical site. A substantial proportion of OSCC cases, majority were diagnosed at an advanced stage (stage IV).

TABLE 1 Sociodemographic details of the participants and the clinical and histopathological diagnosis and Mean ± SD serum uric acid levels in control, oral squamous cell carcinoma.

Characteristics	OSCC PATIENTS	HEALTHY CONTROL
Sample (size)	45	25
Gender(male)	37	16
Mean age	46	19
Tobacco chewing	9	-
Tobacco smoking	11	-
Non tobacco user	8	-
Beetle quid user	14	-
Alcohol consumer	10	-
Serum uric acid(mg/dl)	3.59±1.89	4.85± 1.12

DISCUSSION

It is crucial to emphasize that oxygen radicals can induce cell damage, contributing to oral carcinogenesis through various molecular mechanisms, including DNA damage, oxidation of essential enzymes, protein damage, and activation of specific cytokines.[13] Uric acid, functioning as an antioxidant, is influenced by factors such as alcohol consumption and dietary intake. In the OSCC cohort, the mean serum uric acid level was 3.59±1.89 mg/dL, significantly lower compared to the controls (4.85± 1.12). This difference was statistically significant and aligns with findings from various studies reporting a notable decrease in serum uric acid levels in oral cancer patients compared to healthy controls.[8,9] A study conducted at a tertiary institution in Nigeria observed a similar pattern, reporting a mean serum uric acid level of 5.18 ± 1.96 mg/dL in oral cancer patients, lower than the control group (7.09±1.84 mg/dL). The researchers concluded that this low serum uric acid level was associated with a 3.98 times increased risk of oral cancer development[7,12]. Additionally, Ara et al. found significantly lower serum uric acid levels in patients with OSCC (3.80±2.26 mg/dL) compared to the

control group (5.66±1.82 mg/dL) [8,10,11]. This observation highlights a discernible decrease in serum uric acid levels corresponding to an escalation in clinical staging and grading. Specifically, as the disease progresses, there is a trend of diminishing serum uric acid levels.

A Furthermore, an examination of salivary superoxide dismutase levels revealed a progressive increase from well-differentiated to poorly differentiated OSCC cases, although this trend did not reach statistical significance.[14,15] These findings shed light on potential associations between serum uric acid levels, TNM staging, and grading in OSCC, as well as provide insights into the variation of salivary superoxide dismutase levels across different degrees of OSCC differentiation.

CONCLUSION

The serum uric acid levels displayed a reduction in oral squamous cell carcinoma (OSCC) patients, when compared to healthy controls. Despite the simplicity and cost-effectiveness of measuring serum uric acid levels, the data from this cross-sectional cohort does not provide substantial support for the clinical utility of evaluating uric acid levels in patients with potentially malignant disorders of the oral cavity and OSCC. However, recognizing the limitations of this study, future investigations with larger sample sizes may offer a more comprehensive understanding of the potential contributory role of uric acid.

REFERENCES

1. Johnson NW, Jayasekara P, Amarasinghe AA. Squamous cell carcinoma and precursor lesions of the oral cavity: epidemiology and etiology. Periodontol 2000. 2011;57:19–37.
2. Liu L, Kumar SK, Sedghizadeh PP, Jayakar AN, Shuler CF. Oral squamous cell carcinoma incidence sectioned by sublocations among diverse racial and ethnic populations in California. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2008;105:470–480.
3. Neville BW, Day TA. Oral cancer and precancerous lesions. CA Cancer J Clin. 2002;52:195–215.
4. Carvalho AL, Singh B, Spiro RH, Kowalski LP, Shah JP. Cancer of the oral cavity: a comparison between institutions in a developing and a developed nation. Head Neck. 2004;26:31–38.
5. Rastogi T, Devesa S, Mangtani P, Mathew A, Cooper N, Kao R, et al. Cancer incidence rates among South Asians in four geographic regions: India, Singapore, UK and US. Int J Epidemiol. 2008;37:147–160.
6. Skak-Nielsen H, Torp-Pedersen C, Finer N, et al. Uric acid as a risk factor for cardiovascular disease and mortality in overweight/obese individuals. PLoS One 2013;8:e59121. 10.1371/journal.pone.0059121.
7. Hanna BE, Hamed JM, Touhala LM. Serum uric Acid in smokers. Oman Med J 2008;23:269-74.
8. Lawal AO, Kolude B, Adeyemi BF. Serum uric Acid levels in oral cancer patients seen at tertiary institution in Nigeria. Ann Ib Postgrad Med 2012;10:9-12
9. Ara SA, Ashraf S, Patil BM. Evaluation of serum uric acid levels in patients with oral squamous cell carcinoma. Indian J Dent Res 2016;27:178-83. 10.4103/0970-9290.183128.
10. Gervásio OL, Dutra RA, Tartaglia SM, Vasconcellos WA, Barbosa AA, Aguiar MC. Oral squamous cell carcinoma: a retrospective study of 740 cases in a Brazilian population. Braz Dent J. 2001;12:57–61.
11. Losi-Guembarovski R, Menezes RP, Poliseli F, Chaves VN, Kuasne H, Leichsenring A, et al. Oral carcinoma epidemiology in Paraná State, Southern Brazil. Cad Saude Publica. 2009;25:393–400.
12. Chi AC, Day TA, Neville BW. Oral cavity and oropharyngeal squamous cell carcinoma--an update. CA Cancer J Clin 2015;65:401-21. 10.3322/caac.21293.
13. Farah CS, Shearston K, Nguyen AP, et al. Oral Carcinogenesis and Malignant Transformation. Premalignant Conditions of the Oral Cavity. Singapore: Springer Singapore, 2019:27-66
14. Warnakulasuriya S, Johnson NW, van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. J Oral Pathol Med 2007;36:575-80. 10.1111/j.1600-0714.2007.00582.
15. Porter S, Gueiros LA, Leao JC, et al. Risk factors and etiopathogenesis of potentially premalignant oral epithelial lesions. Oral Surg Oral Med Oral Pathol Oral Radiol 2018;125:603-11. 10.1016/j.oooo.2018.03.008.