HYPERGLYCEMIA AS A RISK FACTOR FOR ORAL CARCINOGENESIS

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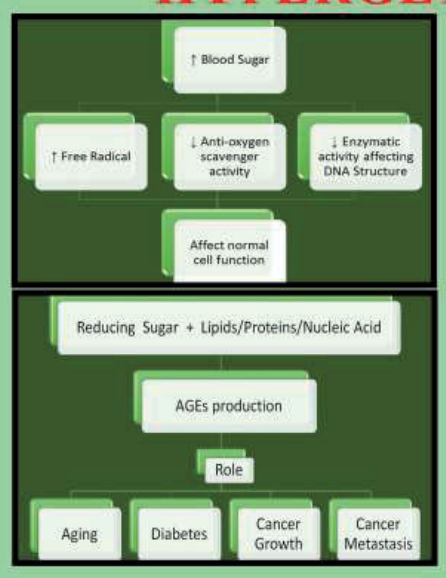
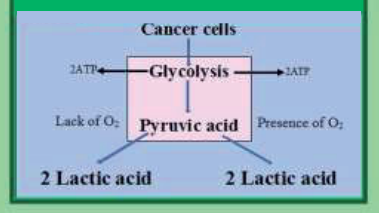


INTRODUCTION:

- Carcinogenesis is a complex, multistep process with alteration of metabolism at cellular level.
- Tobacco products are strongly associated with premalignant and malignant diseases.
- A wide range of parameters like protein, sugar, sialic acid, etc., are hampered during cancer progression. These can be termed as tumour markers.
- Advanced glycation end products (AGEs) is one such biochemical factor produced during hyperglycemia and can also be derived from tobacco products.
- AGEs play role in cancer growth and metastasis by affecting DNA structure and normal cell functioning.
- Tobacco smoke nicotine elevates plasmatic catecholamines, causing an increase in basal glycemia.
- Thus, a long-term tobacco habit may also be associated with increased random blood sugar (RBS) levels during carcinogenesis.
- This study aims to correlate RBS with the role of tobacco, oral PMDs and malignancy.

Warburg Effect

Otto Warburg, determined the strong association of anaerobic glycolysis in malignant tumours. Glucose acts as an anaerobic by-product, even in the presence of oxygen, by converting pyruvate to lactate and then sending it to oxidative phosphorylation; it is also known as **"glucose hunger",** and is considered a hallmark of cancer.



MATERIALS & METHOD:

- Study included 65 participants, divided into three age- and gender-matched groups.
- The oral mucosa was clinically examined and differentiated into normal, premalignant, and malignant diseases.

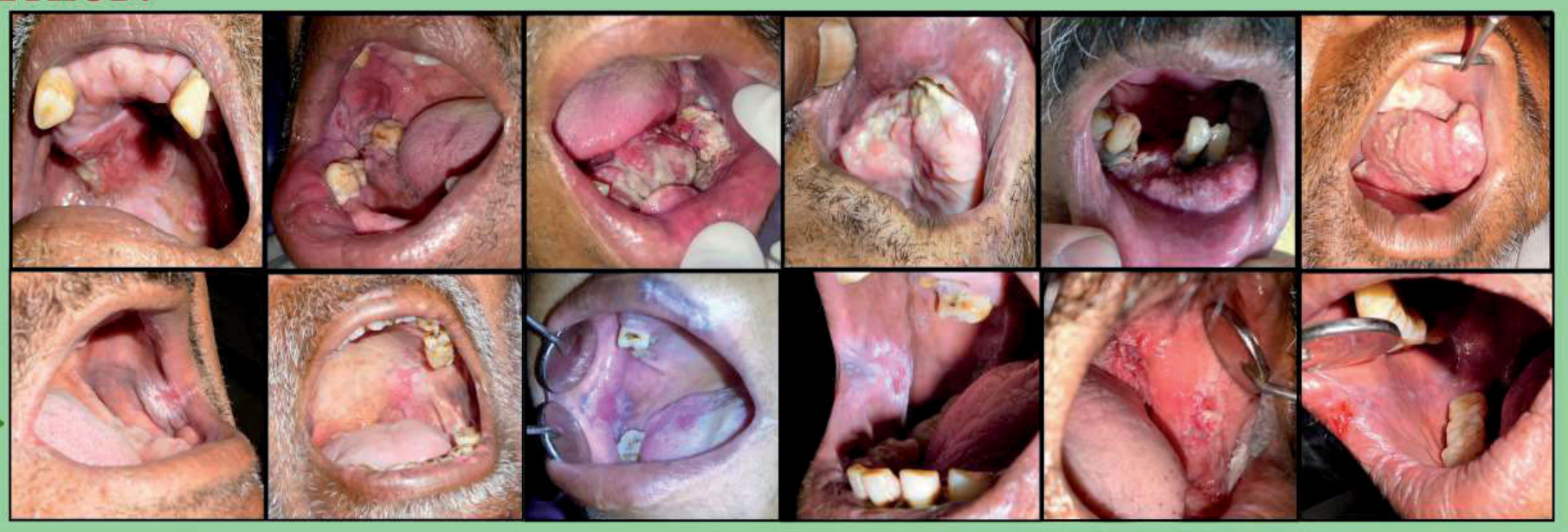


Fig: Clinical Pictures of group 3 participants

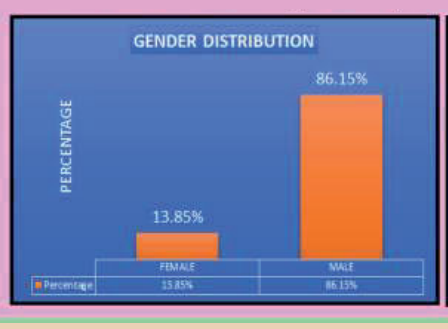
Study Groups	Participants
1. Subjects with clinically normal appearing oral mucosa and no history of smoking	21
2. Subjects with history of smoking but no evidence of PMD's	21
3. Subjects with oral PMD's and Malignancy (OM) with habit of tobacco chewing or smoking	23

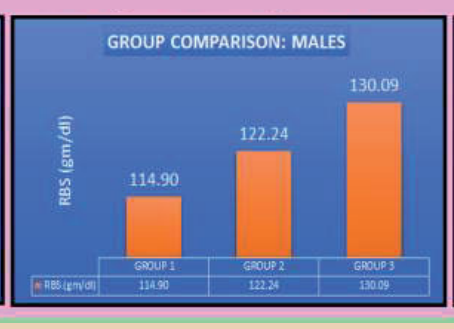
Method:

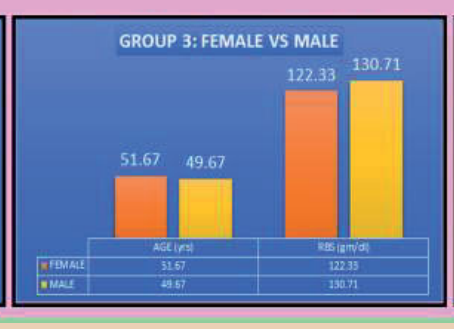
- After verbal consent, a detailed personal and family history was recorded, followed by a clinical oral examination of each participant.
- To investigate RBS, a glucometer along with a lancing device was used.
- Participant records were maintained and data were analysed.

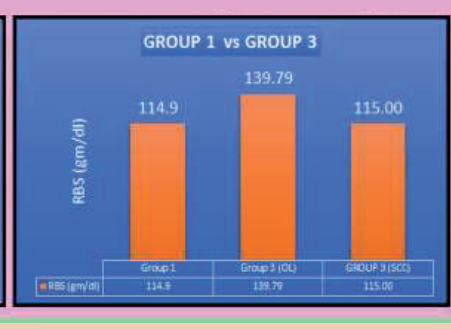
RESULTS:

All 65 participants were aged between 30-70yrs (mean age 47.4yrs). None of the subjects was a known diabetic or had a family history of diabetes. There was an overall increase in RBS level with a smoking habit and was even greater with the presence of clinical lesions. The male participants showed greater changes than females. Also among males, RBS was prominently increased with clinical lesion compared to controls. The following charts depict various aspects of our study:









DISCUSSION:

Browniee M quoted that hyperglycemia leads to the formation of AGEs that damage target cells by modifying intracellular proteins, extracellular matrix components, and plasma proteins, thereby inducing production of reactive oxygen species and causing pathological changes in gene expression. Shenoy et al. suggested that hyperglycemia increased the risk of oral cancer by two folds, while Meisel et al. said that diabetes is a potential risk factor for OL. Cerami et al. found that both aqueous extracts of tobacco and cigarette smoke contain glycotoxins that can rapidly induce AGE formation. In addition, tobacco-derived glycotoxins are far more potent than glucose as they can readily cross cell membranes and cause insertion and/or DNA mutations, possibly leading to cancer. More et al. studied RBS levels in OL, oral submucous fibrosis and OM subjects and found a significant relationship of gradually increasing RBS with premalignancy and malignant stages. In our study, we selected random patients fitting the study group criteria from the out-patient department of our tertiary hospital. As the patients were screened during their first visit, it was difficult to find them with an empty stomach. Hence only RBS was recorded for the current study. We found a positive relationship among study groups, gradually increasing with positive history and subsequently with oral lesions. However, our correlation of OM was inverse compared to More et al., as RBS for OM (115 gm/dl) was prominently less than OL (139.79 gm/dl) participants, being close to the control group (114.9 gm/dl). A gender-based comparison lacked comparable participant numbers, though overall results showed the male population had higher RBS levels than females.

The present pilot study suggests a possible association of RBS with OL. However, the relationship of OM was inverse. Given the limitations, such as only a clinical classification and missing biopsies for diagnosing the oral lesions, there is the possibility of error in diagnosis and its correlation with RBS. The cross-sectional study design precludes causal considerations. Hence, further studies with larger populations with an even distribution gender-wise accompanied by biopsy-based diagnosis shall provide more reliable results.

Note: This is only the 2nd attempt to correlate RBS as a risk factor with tobacco history, premalignant and malignant diseases.

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