

---

## Assessment of salivary pH in patients with oral leukoplakia

---

DR. MUKUNDH CHAITHANYA.V<sup>1</sup>, DR. UMA MAHESWARI.T. N<sup>2\*</sup>

<sup>1</sup>Department of Oral Medicine and Radiology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai -77

<sup>2</sup>Professor and Head of Admin, Department of Oral Medicine and Radiology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai -77

\*Corresponding Author

Email: umasamsi@gmail.com, umamaheswaritn@saveetha.com

---

**Abstract:** Saliva is an important and a complex secretion to be formed in the oral cavity. Salivary pH was done in patients with the presence of leukoplakia and a comparison is done to estimate the salivary pH in patients with leukoplakia and in healthy subjects, though saliva pH has an effect based on the usage of tobacco products and other substances, this study is done to prove that saliva can be used as a diagnostic tool in identifying the lesion which are pre-malignant based on changes in pH. The results of the study show that there is a significant decrease in the mean pH of Saliva in patients with tobacco consumption as compared with non-users, though there are other studies that show in contrast, this study is done to prove the significance of salivary pH change in users of smokeless and smoking in patients with oral leukoplakia. Association analysis of mean pH of patients with oral leukoplakia and Type of leukoplakia shows no statistical significance (Chi-square: 30.000, p value: 0.414 > 0.05).

**Keywords:** Leukoplakia ; Tobacco; Pre-malignancy; Salivary pH; smokeless tobacco

---

### INTRODUCTION

Saliva has varied functions such as lubrication, buffering, digestion etc., salivary pH was done in patients with the presence of leukoplakia and a comparison is done to estimate the salivary pH in patients with leukoplakia and in healthy subjects, the less buffering capacity of the saliva causes the decrease in Salivary pH (acidic pH)[1]. Variations in salivary pH and flow can be affected, reversibly or irreversibly, by numerous physiological and pathological factors including drugs such as antihypertensives, anti-histamines, anti-allergic, anti-diuretics which can adversely affect the salivary flow rate thereby the salivary pH[2]. Saliva is the most easily accessible fluid in the human body and in the future, it will also probably provide an easy tool for non-invasive measurements of various body parameters, pH of saliva has variation based on the periodontal status of the oral cavity [3]. Though saliva pH has an effect based on the usage of tobacco products and other substances, this study is done to prove that saliva can be used as a diagnostic tool in identifying the lesions which are pre-malignant based on its pH. The contents of Saliva can be altered by various drugs (anti-hypertensive, anticholinergic, anti-histamines, diuretics and psychoactive substances) and conditions such as metabolic, nutritional, post-surgery, neurological and psychological diseases. Studies have estimated a range of 5.4 to 7.8, the increase in pH is based on the increase in salivary flow rate (SFR)[4–6]. Clinical and epidemiological studies showed that, potentially malignant disorder associated with areca nut chewing has been associated with decrease in salivary pH (acidic pH)[7]. The increase in salivary flow, helps in the proper cleansing action of the oral cavity, thereby regulating the salivary pH in normal levels[8].

### MATERIALS AND METHODS

The study is done on patients who are provisionally diagnosed with oral leukoplakia and are treated in the department with oral medicine. There are two groups of oral leukoplakia in which patients with habits of tobacco either smoking or chewing (including 30 cases) and compared with patients who are healthy free from adverse habits (including 30 controls). The assessment of salivary pH is done by means of a pH measuring device called the Aquasol digital pH meter, (by Rakiro). Unstimulated saliva is collected from the patient in a sterile container by spitting method and the pH is measured using the digital pH meter. The time of collection of saliva is during the morning hours before 9 am, the patient is asked to refrain from eating, smoking and chewing before pH Assessment. The inferential statistics analysis such as pearson chi-square test was done between type of leukoplakia and levels of dysplasia, mean pH of cases with oral leukoplakia and levels of dysplasia, and mean of pH of cases and type of leukoplakia. Frequency analysis on gender, Type of leukoplakia, levels of dysplasia and tobacco habits was done.

## RESULTS AND DISCUSSION

The salivary pH of the cases and controls varied based on the type of leukoplakia such as homogenous and non-homogenous, and based on the type of dysplasia such as mild, moderate and severe. Frequency analysis on the gender distribution has shown that the male patients (66.7%) were the majority when compared with females (33.3%) (Fig 1). The pH of cases and controls were recorded in patients with various tobacco consumption habits such as Cigarette(Smoking), Pan masala (Smokeless) and Gutka(Smokeless) (Table 1). Among the type of leukoplakia, non-homogenous is seen most commonly in patients with smokeless tobacco consumption (Table 2). Frequency analysis of type of leukoplakia has revealed that about 80% of cases are of homogenous type and the remaining 20% are non-homogenous (Fig.2). The percentage of levels of dysplasia in patients with oral leukoplakia has revealed that 30% of cases had mild dysplasia, 16.7% had moderate dysplasia, 6.7% had severe dysplasia and remaining 46.7% has no/absence of dysplasia (Fig.3). The frequency analysis of tobacco habits in cases with oral leukoplakia has revealed that about 50% of cases had smoking habit, 30% of cases had pan masala habit and remaining 20% had gutka chewing habit (Fig.4). Association analysis between the levels of dysplasia and type of leukoplakia has revealed that there is no statistical significance (Chi-square: 90.000, p value: 0.392>0.05). However, Severe dysplasia is more prevalent in patients with non-homogenous leukoplakia, and moderate dysplasia is seen in patients with homogenous leukoplakia (Fig 5). Association analysis between mean pH of cases with oral leukoplakia and levels of dysplasia has revealed that there is no statistical significance (Chi-square :30.000, p value : 0.392 > 0.05). However, patients with no dysplasia have shown more acidic pH of 6.48 and patients with mild dysplasia with a high pH of 6.95 (Fig 6). Association analysis between the type of leukoplakia and pH of cases with oral leukoplakia has shown that there is no statistical significance. (Chi-square: 30.000, p value: 0.414 > 0.05). However the mean salivary pH in is more acidic in non-homogenous leukoplakia(6.63) and 6.83 in homogenous leukoplakia patients. Studies done by Subha et al., (2018) have shown a slight decrease in salivary pH in patients with use of smokeless tobacco, there is also a mean value of 7.0 who is using smokeless tobacco in comparison with the smoking type which has a mean value of 6.9. In this study Schirmer tear strips for salivary flow rates and pH strips for assessment of pH whereas in the current study pH meter (Aquasol) was used, But the mean values were less significant when compared to my study where the mean value is 6.8, this[1]. Whereas studies done by Kanwar et al.,(2013) have shown a focus of decrease in the salivary flow rate as well as a decrease in pH in patient with habits of tobacco consumption, the mean value was 6.8 in smoking tobacco patients and 6.7 in smokeless tobacco patients where is healthy subjects it was 7.03, which is was significantly similar to my study where the mean value was 6.7 for smokeless consumers and 6.8 for smoking tobacco consumers and for healthy subjects it is 7.13, the reduction of salivary pH was yet again attributed to the decrease in salivary flow which was stated in this study, this is due to the fact that the taste receptors are blocked when there is chronic consumption of smokeless tobacco[2]. Comparing the studies done by Faisal et al.,(2016) shown that the number of patients who have been using smoking tobacco had more susceptibility for decreased pH when compared to the number of patients using smokeless tobacco, when related to other studies, this study proves that the smoking tobacco patients had the more number of patients with significant reduction in pH levels (pH level of 5)[5]. In studies done by Sangeetha et al., (2014) have shown that the salivary pH has less significance with the tobacco chewing habits (areca nut) when compared with patients having Oral potentially malignant disorders like oral submucous Fibrosis, and healthy individuals[6]. Studies done by Khader et al.,(2015) in contraindication have shown a rise in Salivary pH among a group of Areca nut chewers and oral submucous fibrosis group in which the mean value of was 6.76, 6.82 and 6.74 for areca nut chewers, OSMF and healthy subjects respectively, although the number of subjects in the study were about 135, the results of this study has proven that there is less significance of salivary pH among the tobacco consumers and non-consumers(Healthy subjects), but a significance was found only by mean salivary flow rate[7]. In other studies, done by Farsi NM.,(2007) have shown that the pH varies based on the flow rate of the saliva, but the mean difference were very insignificant as both the cases and controls had the same level of salivary pH about 7.38, but were done in normal patients only, but with only dry mouth and dry mucosa unlike the current study[9]. Alterations in salivary pH are observed in patients with areca nut chewing. The alteration in SFR and pH are vital in causation of various oral diseases.[10,11]. Certain studies done Donoghue et al., (2015) have concluded in their study that the long-term use of Areca nut (smokeless tobacco) has significant effect on salivary pH reduction, along with the presence of Oral premalignant disorders can promote the acidity of salivary pH[8]. Again, studies done by Groover et al., (2016) has shown that among the group of tobacco users, the smoking tobacco users had a pH of about 6.75 wherein the smokeless tobacco has pH of about 6.5, the reason quoted here is that the use of lime in Smokeless Tobacco reacts with bicarbonate buffering system, along with change in electrolytes and ions by which loss of bicarbonate occurs resulting the acidity nature of the saliva[12]

## CONCLUSION

This study concludes that the mean pH among the smokeless, smoking tobacco and controls, is about 6.63, 6.83 and 7.01 respectively. Association analysis between types of dysplasia and leukoplakia has revealed that there is

no statistical significance (Chi-square: 90.000, p value: 0.392 > 0.05). Hence the study result proves that the salivary pH is low in both smoking and smokeless tobacco users when compared to non-users of tobacco. The mean pH in patients with Non-Homogenous leukoplakia (6.63) is less when compared with patients with homogenous leukoplakia (6.68), which shows that the decrease in pH corresponds to the high chances of malignancy. Saliva being a relatively easy to collect and non-invasive procedure, and need no specialised to collect it, with cheaper test kits available is one of the best diagnostic tools for analysis of salivary pH and other biomarkers by which the presence or absence of various diseases, and parameters in the human body can be elucidated, and therefore the results of this study prove the above statement.

### CONFLICT OF INTEREST

There is no conflict of interest in this study

### ACKNOWLEDGEMENT

We would like to thank the Saveetha Dental College and Hospitals, Chennai for granting us the clearance to conduct this study.

### REFERENCES

1. Shubha G, Fasalkar SS, Praveen BN, Patrick S, Subhashini AR, Keerthi G. Assessment of salivary flow rate and salivary pH in subjects with smoking and smokeless form of tobacco habits. *Journal of Medicine, Radiology, Pathology and Surgery* 2018;5:11–5.
2. Kanwar A, Sah K, Grover N, Chandra S, Singh RR. Long-term effect of tobacco on resting whole mouth salivary flow rate and pH: An institutional based comparative study. *European Journal of General Dentistry* 2013;2:296.
3. Baliga S, Muglikar S, Kale R. Salivary pH: A diagnostic biomarker. *J Indian Soc Periodontol* 2013;17:461–5.
4. Puy CL. The role of saliva in maintaining oral health and as an aid to diagnosis. *Med Oral Patol Oral Cir Bucal* 2006;11:449–55.
5. Rehan F, Khan RS, Memon MS, Naqvi S, Khan RS, Sultan ZK. Analysis of resting mouth salivary flow rate and salivary pH of tobacco chewers and smokers. *J Pak Dent Assoc* 2016;4:159–63.
6. Siddabasappa S, Ashok L, Sujatha GP. Estimation of unstimulated salivary flow rate, pH, copper and Iron in ghutka chewers with and without oral submucous fibrosis: a preliminary study. *Res J Pharm Biol Chem Sci* 2014;5:300–6.
7. Abdul Khader NF, Dyasanoor S. Assessment of Salivary Flow Rate and pH Among Areca Nut Chewers and Oral Submucous Fibrosis Subjects: A Comparative Study. *J Cancer Prev* 2015;20:208–15.
8. Donoghue M, Basandi PS, Adarsh H, Madhushankari GS, Selvamani M, Nayak P. Habit-associated salivary pH changes in oral submucous fibrosis-A controlled cross-sectional study. *J Oral Maxillofac Pathol* 2015;19:175–81.
9. Farsi NMA. Signs of oral dryness in relation to salivary flow rate, pH, buffering capacity and dry mouth complaints. *BMC Oral Health* 2007;7:15.
10. Rooban T, Mishra G, Elizabeth J, Ranganathan K, Saraswathi TR. Effect of habitual arecanut chewing on resting whole mouth salivary flow rate and pH. *Indian J Med Sci* 2006;60:95–105.
11. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. *Oral Oncol* 2009;45:309–16.
12. Grover N, Sharma J, Sengupta S, Singh S, Singh N, Kaur H. Long-term effect of tobacco on unstimulated salivary pH. *J Oral Maxillofac Pathol* 2016;20:16–9.

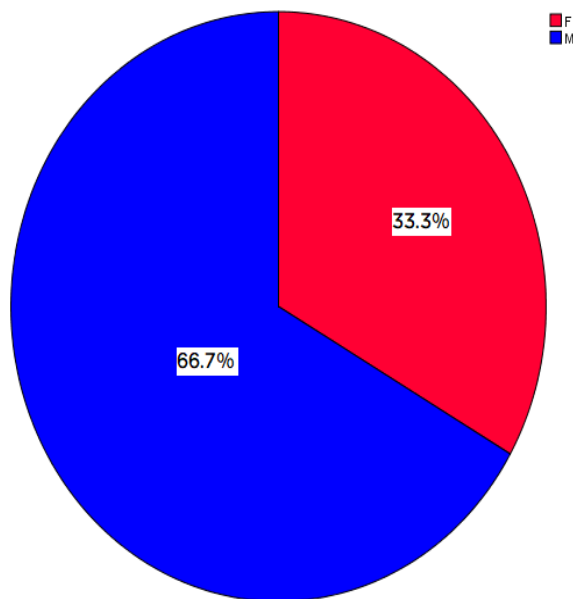
**Table 1: Salivary pH in control and tobacco users**

S.No	pH of Controls	Habits associated with cases	pH of Cases
1	6.6	Cigarette (Smoking)	6.8
2	7.2	Pan Masala (Smokeless)	7.0
3	6.8	Pan Masala (Smokeless)	7.2
4	7.2	Cigarette (Smoking)	6.9
5	7.8	Pan Masala (Smokeless)	6.7
6	7.8	Pan Masala (Smokeless)	7.4
7	7.4	Cigarette (Smoking)	6.7
8	7.2	Gutkha (Smokeless)	5.0
9	7.0	Cigarette (Smoking)	7.0
10	6.8	Cigarette (Smoking)	6.7
11	6.9	Gutkha (Smokeless)	6.5
12	6.7	Gutkha (Smokeless)	6.7
13	7.0	Pan Masala (Smokeless)	6.7

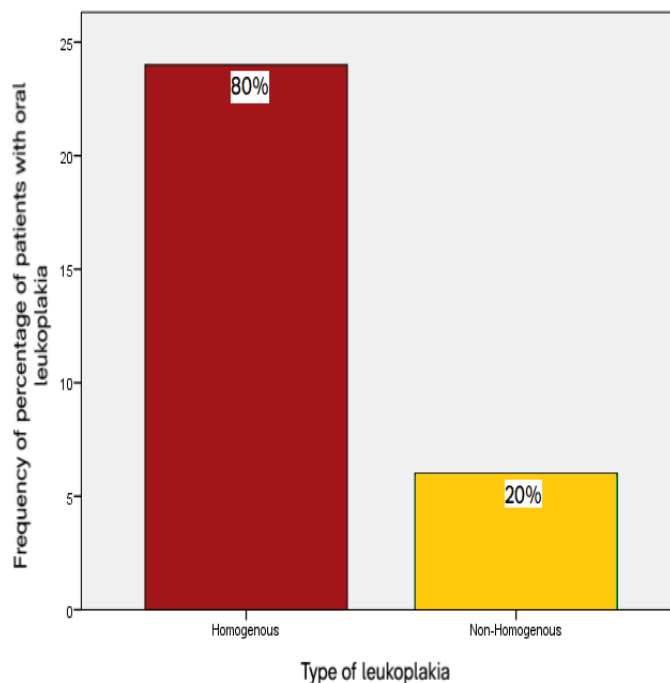
14	7.2	Cigarette (Smoking)	6.9
15	7.0	Pan Masala (Smokeless)	6.6
16	6.8	Cigarette (Smoking)	6.8
17	6.7	Cigarette (Smoking)	6.7
18	6.9	Pan Masala (Smokeless)	6.6
19	7.0	Cigarette (Smoking)	6.8
20	7.2	Cigarette (Smoking)	6.8
21	6.7	Gutkha (Smokeless)	6.5
22	6.6	Cigarette (Smoking)	6.8
23	6.8	Cigarette (Smoking)	6.9
24	6.9	Pan Masala (Smokeless)	6.5
25	7.0	Cigarette (Smoking)	6.8
26	6.8	Cigarette (Smoking)	6.9
27	7.5	Gutkha (Smokeless)	6.6
28	7.2	Cigarette (Smoking)	7.0
29	6.8	Pan Masala (Smokeless)	6.9
30	6.9	Gutkha (Smokeless)	6.6

**Table 2: Type of smoking and smokeless tobacco in clinical types of leukoplakia**

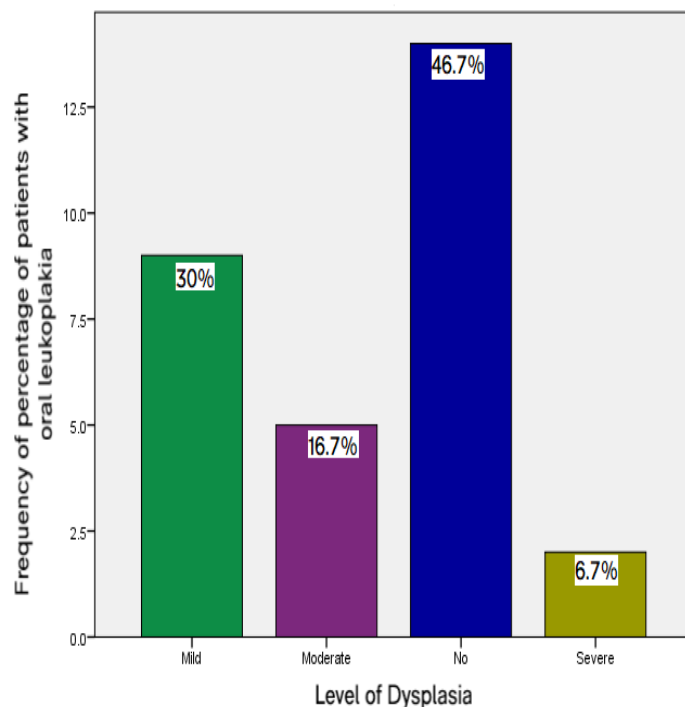
S.No	Type of Tobacco used	Type of leukoplakia
1	Cigarette (Smoking)	Homogenous
2	Pan Masala (Smokeless)	Non-Homogenous
3	Pan Masala (Smokeless)	Homogenous
4	Cigarette (Smoking)	Non-Homogenous
5	Pan Masala (Smokeless)	Homogenous
6	Pan Masala (Smokeless)	Homogenous
7	Cigarette (Smoking)	Homogenous
8	Gutkha (Smokeless)	Homogenous
9	Cigarette (Smoking)	Homogenous
10	Cigarette (Smoking)	Homogenous
11	Gutkha (Smokeless)	Non-Homogenous
12	Gutkha (Smokeless)	Homogenous
13	Pan Masala (Smokeless)	Homogenous
14	Cigarette (Smoking)	Homogenous
15	Pan Masala (Smokeless)	Non-Homogenous
16	Cigarette (Smoking)	Homogenous
17	Cigarette (Smoking)	Homogenous
18	Pan Masala (Smokeless)	Homogenous
19	Cigarette (Smoking)	Homogenous
20	Cigarette (Smoking)	Homogenous
21	Gutkha (Smokeless)	Non-Homogenous
22	Cigarette (Smoking)	Homogenous
23	Cigarette (Smoking)	Homogenous
24	Pan Masala (Smokeless)	Homogenous
25	Cigarette (Smoking)	Homogenous
26	Cigarette (Smoking)	Homogenous
27	Gutkha (Smokeless)	Homogenous
28	Cigarette (Smoking)	Homogenous
29	Pan Masala (Smokeless)	Homogenous
30	Gutkha (Smokeless)	Non-Homogenous



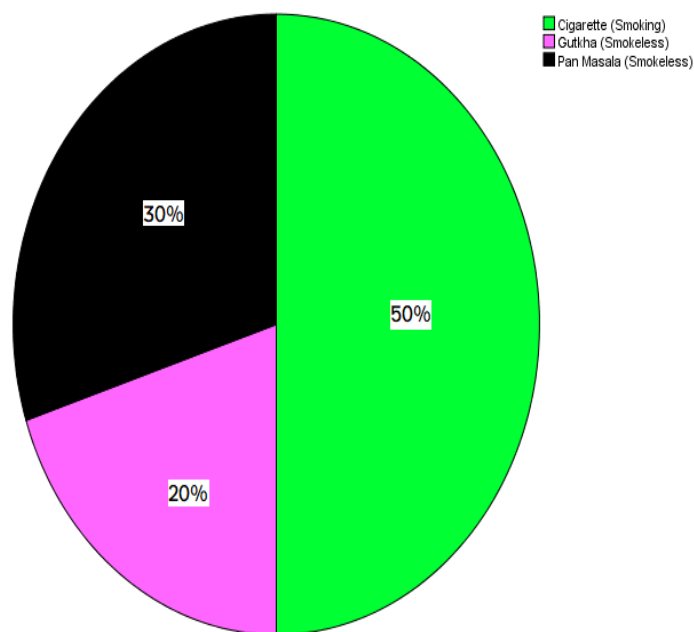
**Fig.1: Pie chart representing gender distribution in patients with oral leukoplakia. Percentage prevalence has revealed that 66.7% of patients are male, and 33.3% of patients are females. Hence , patients with oral leukoplakia are more common in male patients**



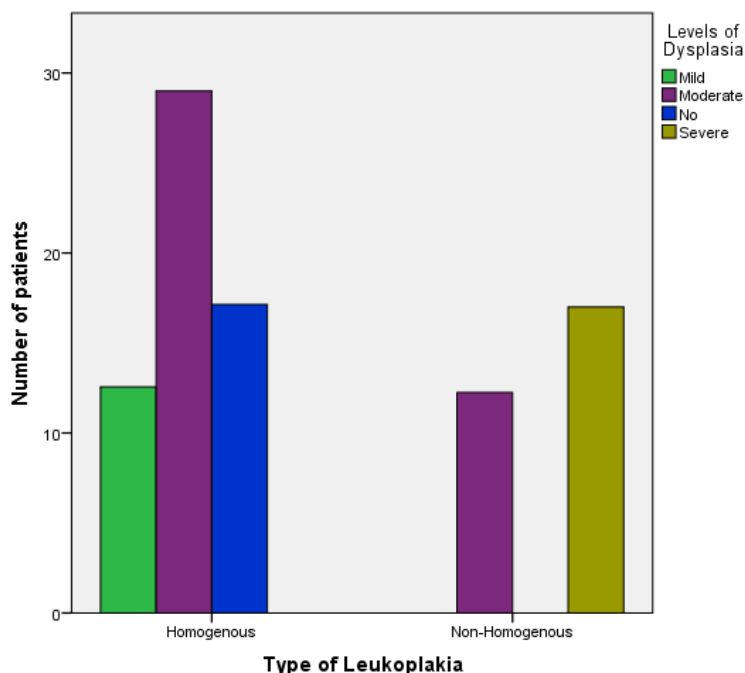
**Fig.2: Bar chart representing distribution of the type of leukoplakia in dental patients. X-axis represents the type of leukoplakia, Y-axis represents the percentage of oral leukoplakia patients. Frequency distribution shows that homogeneous leukoplakia (80%) is the most prevalent , followed by non-homogenous leukoplakia (20%)**



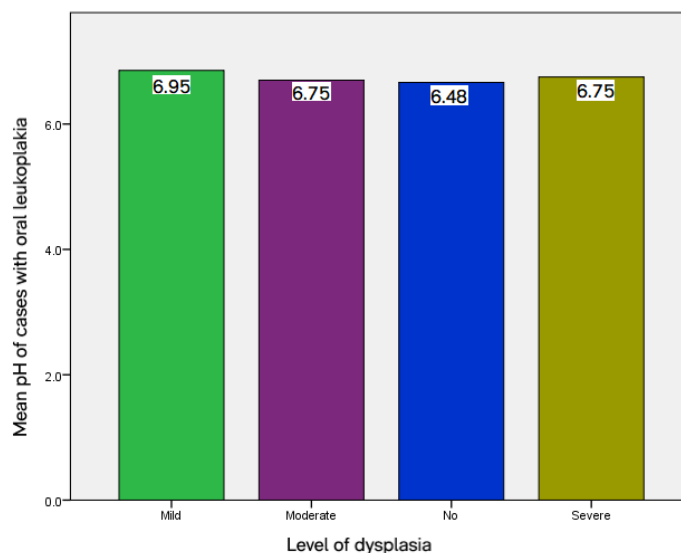
**Fig.3:** Bar graph represents the level of dysplasia in patients with oral leukoplakia. X-axis represents the level of dysplasia. Y-axis presents the number of patients with oral leukoplakia. Frequency distribution reveals that Mild dysplasia (30%) is the most common, while severe dysplasia (6.7%) is the least common.



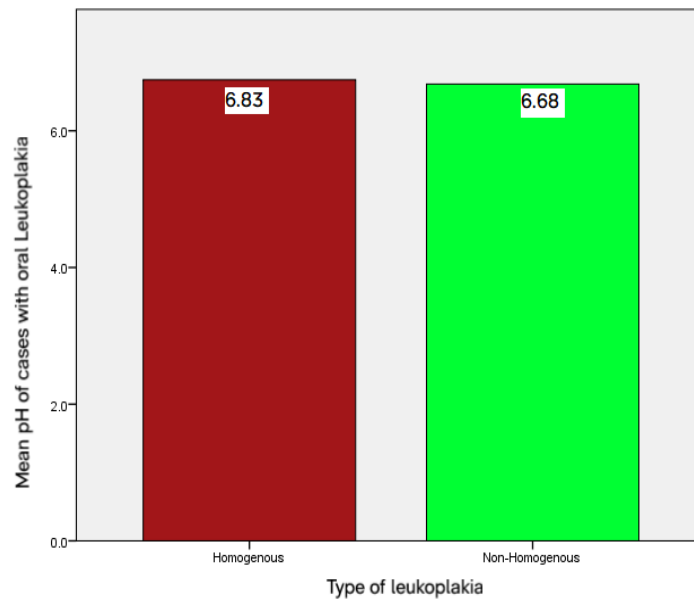
**Fig.4:** Pie chart showing the percentage of oral leukoplakia with various tobacco habits , Percentage distribution shows that Cigarette (Smoking-50%) is the most common habit followed by Pan masala (Smokeless-30%), Gutkha (Smokeless-20%)



**Fig.5: Bar graph representing the association between Type of leukoplakia and levels of dysplasia. X-axis represents the type of leukoplakia. Y-axis represents the number of patients. Association analysis using pearson- chi square test that there is no statistical significance (Chi-square: 90.000, p value: 0.392>0.05). However, Severe dysplasia is more prevalent in patients with non-homogenous leukoplakia, and moderate dysplasia is seen in patients with homogenous leukoplakia**



**Fig.6: Bar graph represents the association between levels of dysplasia and mean pH of cases with oral leukoplakia. X-axis represents the level of dysplasia and Y-axis represents the Mean pH of cases with oral leukoplakia. Association analysis using pearson-chi analysis test reveals that there is no statistical significance (Chi-square :30.000, p value : 0.392 > 0.05). However, patients with no dysplasia have shown more acidic pH of 6.48 and patients with mild dysplasia with a high pH of 6.95.**



**Fig. 7: Bar graph represents the association between type of leukoplakia and pH of cases with oral leukoplakia. X-axis shows the type of leukoplakia. Y-axis shows the mean pH of cases. Association analysis using Pearson chi-square test revealed that there is no statistical significance (Chi-square: 30.000, p value: 0.414 > 0.05). However the mean salivary pH in is more acidic in non-homogenous leukoplakia(6.63) and 6.83 in homogenous leukoplakia patients**