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Dermatoglyphics as an indicator in diagnosis of oral precancerous lesions among smoke and smokeless tobacco users

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Abstract

Introduction: Palmar prints or palmar dermatoglyphics has been useful in understanding the unanswered questions in biology, medicine, genetics and evolution. The role of dermatoglyphics in the study of chromosomal diseases and cardiac disease are explored in the past but their importance in dentistry is yet at infant stage. Oral cancer can be treated efficiently at an early stage by targeting the pre-cancerous lesion (Potentially malignant diseases (PMDs)) improving the survival rates.

Aim and objectives: The present study is conducted to co-relate the role of dermatoglyphics in determining the risk of developing potentially malignant diseases (PMDs) in patients with use of smoke tobacco and tobacco chewers.

Materials and methods: A cross sectional study was planned with the consent from institutional ethical committee and patient for taking the palmar prints. Five groups depending on the status of the habit (smoke tobacco with PMD, non smoke tobacco with PMD, combination of smoke and non smoke with PMD, Smoke or non smoke tobacco with NO PMD and control) were divided randomly each comprising of 20 patients. Following the inclusion and exclusion criteria the included patients were subjected for obtaining the palmar prints which were analysed quantitatively and qualitatively and data obtained was statistically evaluated.

Results: Qualitative analysis shows that simple arch patterns were common in Group 2 (the smoke tobacco users with PMD). Plain whorl patterns were common in Group 4 (the combination of smoke and non smoke tobacco users with PMD).

Quantitative analysis shows that left and right ATD angle were prominent in Group 5 (patient with tobacco habit with NO PMD). Right AB ridge count was prominent in Group 2 (the smoke tobacco users with PMD) and Group 4 (the combination of smoke and non smoke tobacco users with PMD). Right AB ridge count was in the range 29.8-30.50. ATD angles are in the range 40-42 degrees.

Conclusion: Dermatoglyphics has moved from obscurity to acceptability as a diagnostic tool which can be appreciated from this study.

Keywords: Dermatoglyphics, potentially malignant diseases, oral cancer, palmar prints

Introduction

Fingerprint analysis for personal identification is unique to all individuals and remains the same throughout the life. The hand become a powerful tool in the diagnosis of psychological, medical, genetic conditions. Dermatoglyphics has provided a firm empirical study for the modern study of chirolology.

The word "Dermatoglyphics" comes from Greek words *derma* = skin & *glyphe* means carve. It refers to study of epidermal ridges on the hands and feet^[1]. The term was coined by Harold Cummins in 1926, known as "Father of Dermatoglyphics"^[2]. Finger and palm prints are formed during the 6th-7th week of embryonic life and completed after 10-20 weeks of gestation. Since it is unique for each person & not even same in monozygotic twins, studying them can determine a number of parameters helpful in the diagnosis and treatment of examined individuals.

The ridge patterns are influenced by the blood vessel nerve pairs at the border between dermis and epidermis during prenatal development and factors such as inadequate oxygen supply, unusual distribution of sweat glands and alteration of epithelial growths.

The ridge pattern is considered sensitive because it originates from the fetal volar pads just like the teeth which also develop from the ectoderm at the 6th-7th week of intrauterine life. Dermatoglyphic patterns were previously used in the study of a chromosomal diseases, cardiac diseases and leukemia [3]. William Herschel was the first to experiment with fingerprints in India [4]. The purpose of this cross-sectional study is to correlate the dermatoglyphic patterns and risk of developing precancerous, cancerous lesions in smokeless and smoke tobacco users.

Aims & Objectives

The present study is conducted to co-relate the role of

dermatoglyphics in determining the risk of developing precancerous lesions in patients with tobacco and non-tobacco chewers.

Materials and Methods

Patients reporting to the Department of Oral Medicine And Radiology were selected for the study. Informed consent was taken from the patients prior to obtaining the fingerprints. It was explained to the patient purpose of recording their fingerprints and assured about confidentiality. individuals were divided into 5 groups depending on the habit.

PERFORMA	
PALMAR DERMATOGLYPHICS IN POTENTIALLY MALIGNANT DISEASES	
DEMOGRAPHIC DETAILS:	
AGE/SEX:	
HABIT:	
<ul style="list-style-type: none"> • SMOKE • SMOKELESS TOBACCO • COMBINATION • COMBINATION+OTHERS 	
FAMILY HISTORY AND/OR H/O PMD:	
GROUP 1: SMOKERS+PMD	
GROUP 2: SMOKELESS+PMD	
GROUP3: COMBINATION+PMD	
GROUP4: CONTROLS	
<small>CONSENT: I GIVE MY CONSENT TO USE THE INFORMATION OF FINGER/PALM PRINTS FOR RESEARCH PURPOSES ONLY.</small>	
<small>PT SIGN</small>	

Fig 1: Showing Performa Data sheet

Inclusion Criteria

- Patient's age group between 20 and 65 years.
- Patient who clinically diagnosed with oral malignant diseases but not associated with malignant changes.
- Patient who give history of using smokeless and smoke forms of tobacco.

Exclusion Criteria

- Patients with other causes of oral lesions like sharp tooth, dentures, aphthous ulcers, poor oral hygiene, and cavities.etc will be excluded.

Method

The finger and palm prints were obtained by the ink method on A4 size paper. The armamentarium comprises of Kore's Duplicating ink, white paper, magnifying lens, protractor, scale and pencil.



Fig 2: Showing Armamentarium

Hands were thoroughly washed with soap before taking prints to remove soil, oil, dirt. The required amount of ink is to be placed on the palms and fingers. The ink is to be spread evenly on palms and fingers. To take the palm print, the palm is to be kept on the white paper and firm pressure was given on the center of the dorsum hand and the interdigital areas. For fingerprints, they were placed on a white paper with one lateral edge and then rolled in the opposite direction. These Dermatoglyphic patterns are analyzed with a magnifying lens. The patterns are analyzed after blinding. Blinding means that the participants were not told about the groups they were placed into. This was done to avoid bias in the study. Patterns were analyzed both qualitatively and quantitatively.



Fig 3: Showing palmer prints

Qualitative Analysis

Fingerprints are classified into whorls, arches and loops. Patterns on all 10 fingers are analyzed. In every subject, the frequency of each pattern was recorded and the percentage

of pattern frequency was calculated for the entire group. Palmar patterns were observed in the hypothenar, thenar, interdigital areas. Comparison was made between study and control groups.

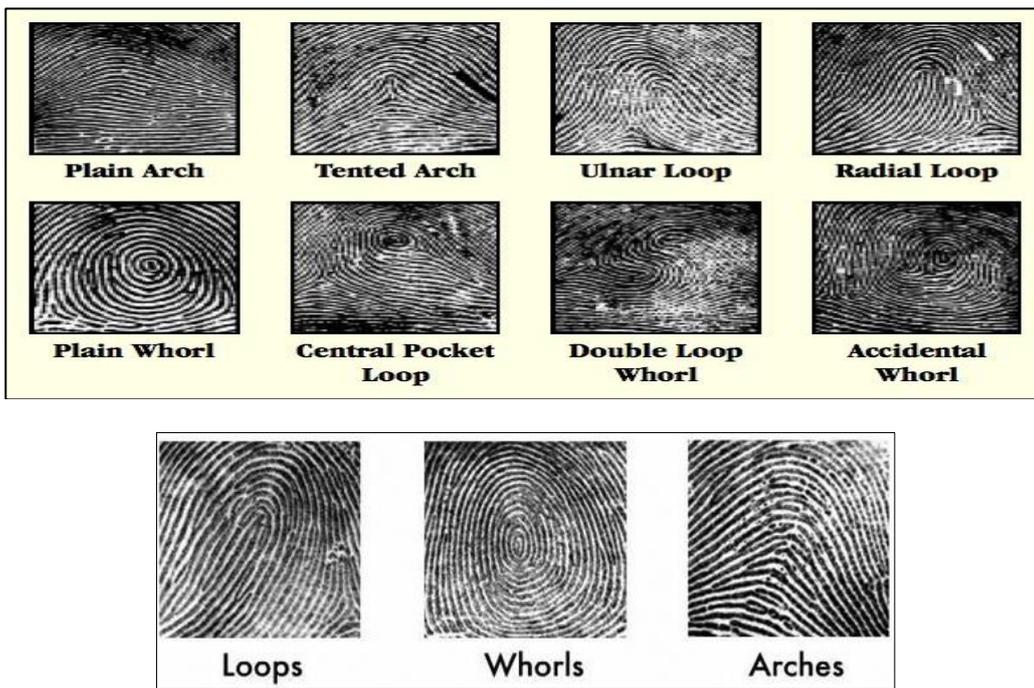


Fig 4: Patterns of fingerprints

Quantitative Analysis

The following parameters are to be analyzed

- ATD angle: It is a dermatoglyphic trait formed by drawing lines between the triradii below the first and last digits and the most proximal triradius on the hypothenar region of the palm. ATD angles are measured in both hands.
- AB ridge count: The number of ridges present between triradii A and triradii B.

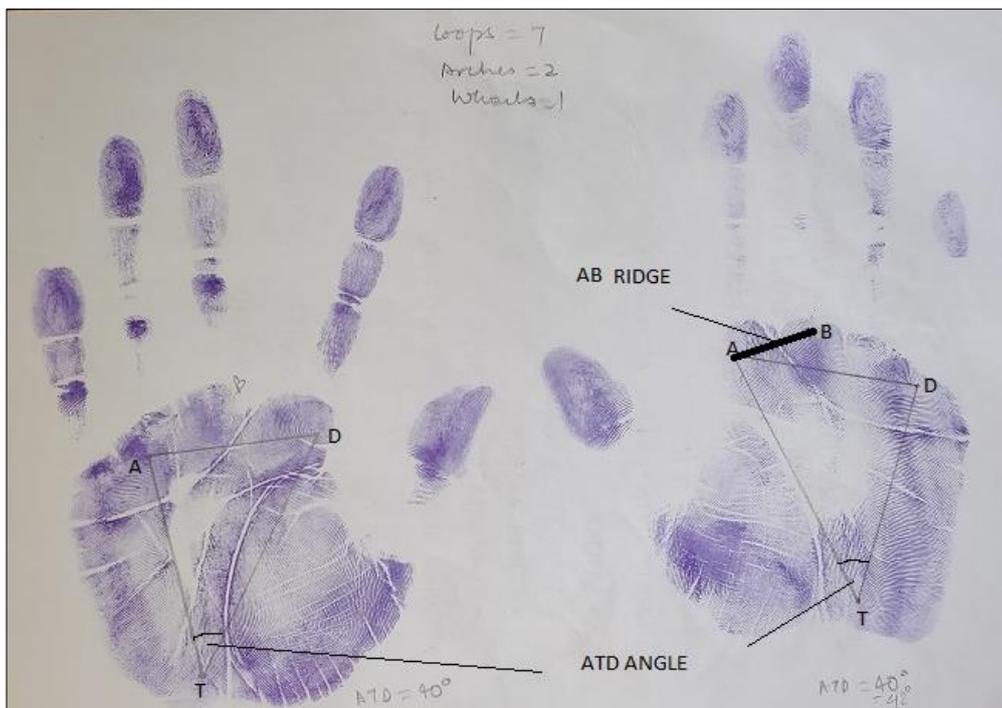


Fig 5: Showing different parameters like ATD angle AB Bridge

These parameters were analyzed statistically using SPSS software. For qualitative and quantitative analysis chi square test was performed. Inter and intra observer agreement is to

be assessed by Kappa statistic. The statistical tests applied were ANOVA, Kruskal Wallis and Mann Whitney test.

Results

(Qualitative – Ridge pattern)

Table 3: Comparison of whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	3.30	2.886	2.318	0.677
Smoking with lesion	20	3.15	2.925		
Smokeless with lesion	20	3.40	2.303		
Combination with lesion	20	4.10	2.426		
Habit with no lesion	20	3.85	2.621		
Total	100	3.56	2.614		

Non significant with P value 0.677

Table 4: Comparison of loops in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	5.40	2.761	0.441	0.979
Smoking with lesion	20	5.25	2.489		
Smokeless with lesion	20	5.80	2.067		
Combination with lesion	20	5.55	2.395		
Habit with no lesion	20	5.65	2.519		
Total	100	5.53	2.414		

Non significant with p value 0.979

Table 5: Comparison of arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	1.30	2.250	10.887	0.028*
Smoking with lesion	20	1.60	1.875		
Smokeless with lesion	20	0.80	1.436		
Combination with lesion	20	0.30	0.733		
Habit with no lesion	20	0.50	1.051		
Total	100	0.90	1.611		

($p < 0.05$ - Significant*, $p < 0.001$ - Highly significant**)

Statistically significant p value of 0.028 which shows that the arches show significant changes in the groups.

(Individual comparison using Mann Whitney U test)

	Control	Smoking with lesion	Smokeless with lesion	Combination with lesion	Habit with no lesion
Control	-	0.349	0.576	0.045*	0.166
Smoking with lesion	0.349	-	0.116	0.004*	0.020*
Smokeless with lesion	0.576	0.116	-	0.117	0.361
Combination with lesion	0.045*	0.004*	0.117	-	0.473
Habit with no lesion	0.166	0.020*	0.361	0.473	-

Table 6: Comparison of left ATD angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi square value	P value
Control	20	40.45	4.850	1.534	0.821
Smoking with lesion	20	39.95	5.073		
Smokeless with lesion	20	40.35	4.977		
Combination with lesion	20	40.65	3.617		
Habit with no lesion	20	41.70	5.488		
Total	100	40.62	4.780		

Non significant results with p value 0.821 and left ATD angle meagerly increased in case of group 5.

Table 7: Comparison of right ATD angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi square value	P value
Control	20	40.80	4.150	3.570	0.467
Smoking with lesion	20	40.40	3.515		
Smokeless with lesion	20	40.15	5.631		
Combination with lesion	20	40.30	3.813		
Habit with no lesion	20	42.55	5.246		
Total	100	40.84	4.541		

Non significant with p value 0.467. Right ATD angle meagerly increased in case of group 5

Table 8: Comparison of left AB angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi square value	P value
Control	20	28.05	4.071	2.018	0.732
Smoking with lesion	20	30.00	4.746		
Smokeless with lesion	20	29.50	4.752		
Combination with lesion	20	30.00	4.425		
Habit with no lesion	20	28.50	4.407		
Total	100	29.21	4.468		

Not significant with p value 0.732

Table 9: Comparison of right AB angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi square value	P value
Control	20	29.80	4.549	4.085	0.395
Smoking with lesion	20	31.80	5.033		
Smokeless with lesion	20	32.20	4.991		
Combination with lesion	20	30.50	4.212		
Habit with no lesion	20	30.80	4.324		
Total	100	31.02	4.623		

Non significant with p value 0.395. Very minor increase found in group 2 and 4. (Arches – Simple & tented)

Table 10: Comparison of simple arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi square value	P value
Control	20	1.45	2.235	12.180	0.016*
Smoking with lesion	20	1.55	1.877		
Smokeless with lesion	20	0.95	1.468		
Combination with lesion	20	0.55	1.432		
Habit with no lesion	20	0.35	0.988		
Total	100	0.97	1.690		

($p < 0.05$ - Significant*, $p < 0.001$ - Highly significant**)

Significant with p value 0.016 simple arches are prominent among group 2. (Individual comparison using Mann Whitney U test)

	Control	Smoking with lesion	Smokeless with lesion	Combination with lesion	Habit with no lesion
Control	-	0.657	0.545	0.05*	0.020*
Smoking with lesion	0.657	-	0.292	0.016*	0.005*
Smokeless with lesion	0.545	0.292	-	0.125	0.05*
Combination with lesion	0.05*	0.016*	0.125	-	0.668
Habit with no lesion	0.020*	0.005*	0.05*	0.668	-

Table 11: Comparison of tented arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	0.05	0.224	0.002	1.000
Smoking with lesion	20	0.05	0.224		
Smokeless with lesion	20	0.05	0.224		
Combination with lesion	20	0.10	0.447		
Habit with no lesion	20	0.05	0.224		
Total	100	0.06	0.278		

Non significant results with tented arches with p value 1.000 (Whorls –Plain, spiral, central, lateral & complex)

Table 12: Comparison of plain whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	1.05	1.234	16.116	0.003*
Smoking with lesion	20	0.85	1.226		
Smokeless with lesion	20	2.00	1.589		
Combination with lesion	20	2.70	2.342		
Habit with no lesion	20	1.95	1.395		
Total	100	1.71	1.719		

($p < 0.05$ - Significant*, $p < 0.001$ - Highly significant**)

Plain whorls have significant with p value 0.003 prominent among group 4.

Table 13: Comparison of spiral whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	0.70	1.174	1.094	0.895
Smoking with lesion	20	1.10	1.553		
Smokeless with lesion	20	0.85	1.268		
Combination with lesion	20	0.70	0.923		
Habit with no lesion	20	1.40	2.349		
Total	100	0.95	1.527		

Table 14: Comparison of central whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	0.95	2.064	3.126	0.537
Smoking with lesion	20	0.95	1.468		
Smokeless with lesion	20	0.35	0.988		
Combination with lesion	20	0.70	1.218		
Habit with no lesion	20	0.65	1.785		
Total	100	0.72	1.538		

Table 15: Comparison of lateral whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	0.00	0.000	0.0	1.0
Smoking with lesion	20	0.00	0.000		
Smokeless with lesion	20	0.00	0.000		
Combination with lesion	20	0.00	0.000		
Habit with no lesion	20	0.00	0.000		
Total	100	0.00	0.000		

Table 16: Comparison of complex whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	0.00	0.000	0.0	1.0
Smoking with lesion	20	0.00	0.000		
Smokeless with lesion	20	0.00	0.000		
Combination with lesion	20	0.00	0.000		
Habit with no lesion	20	0.00	0.000		
Total	100	0.00	0.000		

(Loops – Ulnar & radial)

Table 17: Comparison of ulnar loop in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	4.25	1.970	1.485	0.829
Smoking with lesion	20	4.35	2.183		
Smokeless with lesion	20	4.10	1.619		
Combination with lesion	20	3.75	1.713		
Habit with no lesion	20	3.85	2.254		
Total	100	4.06	1.938		

Table 18: Comparison of radial loop in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Group	N	Mean	Std. Deviation	Chi squarevalue	P value
Control	20	1.55	0.945	4.766	0.312
Smoking with lesion	20	1.20	0.951		
Smokeless with lesion	20	1.60	0.883		
Combination with lesion	20	1.60	0.883		
Habit with no lesion	20	1.75	0.786		
Total	100	1.54	0.892		

(Individual comparison using Mann Whitney U test)

	Control	Smoking with lesion	Smokeless with lesion	Combination with lesion	Habit with no lesion
Control	-	0.489	0.05*	0.005*	0.037*
Smoking with lesion	0.489	-	0.015*	<0.001**	0.010*
Smokeless with lesion	0.05*	0.015*	-	0.491	0.923
Combination with lesion	0.005*	<0.001**	0.491	-	0.421
Habit with no lesion	0.037*	0.010*	0.923	0.421	-

(Qualitative – Ridge pattern)

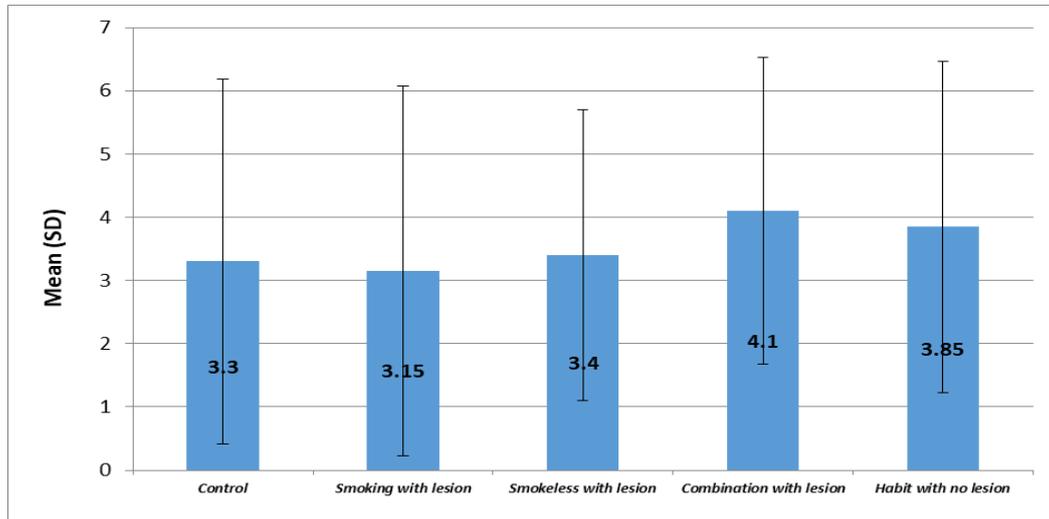


Fig 3: Comparison of whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

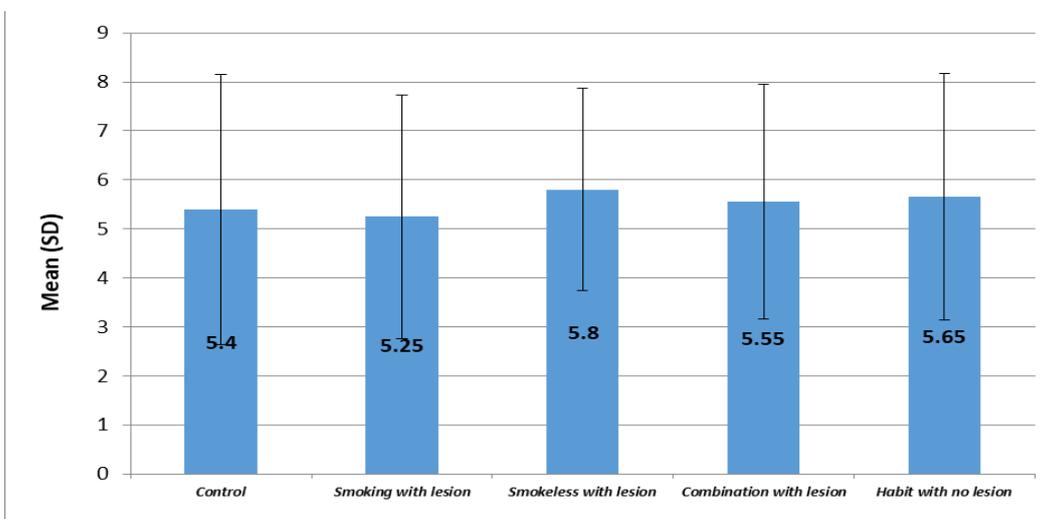


Fig 4: Comparison of loops in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

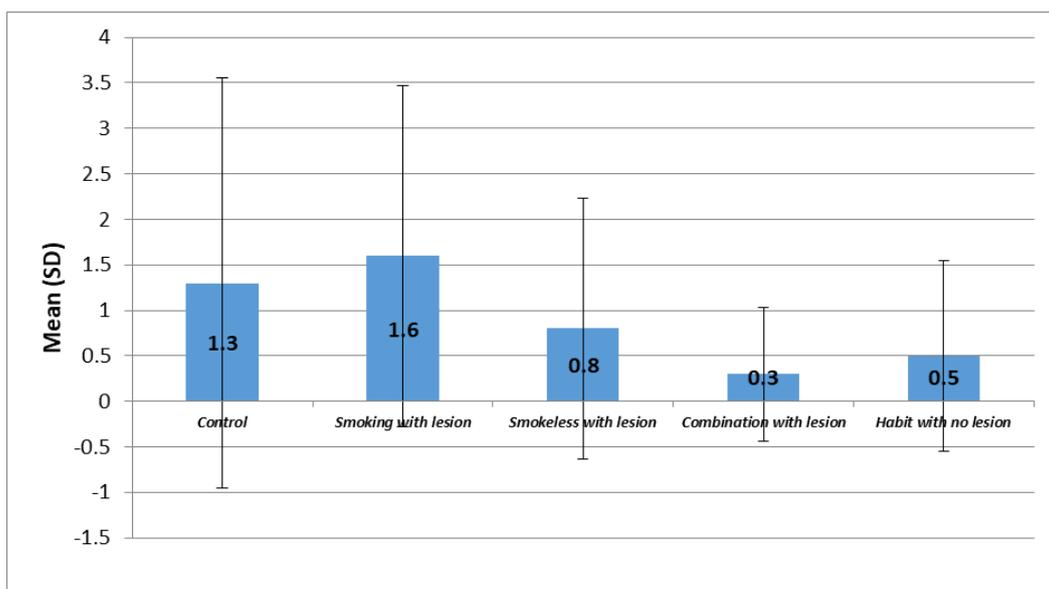
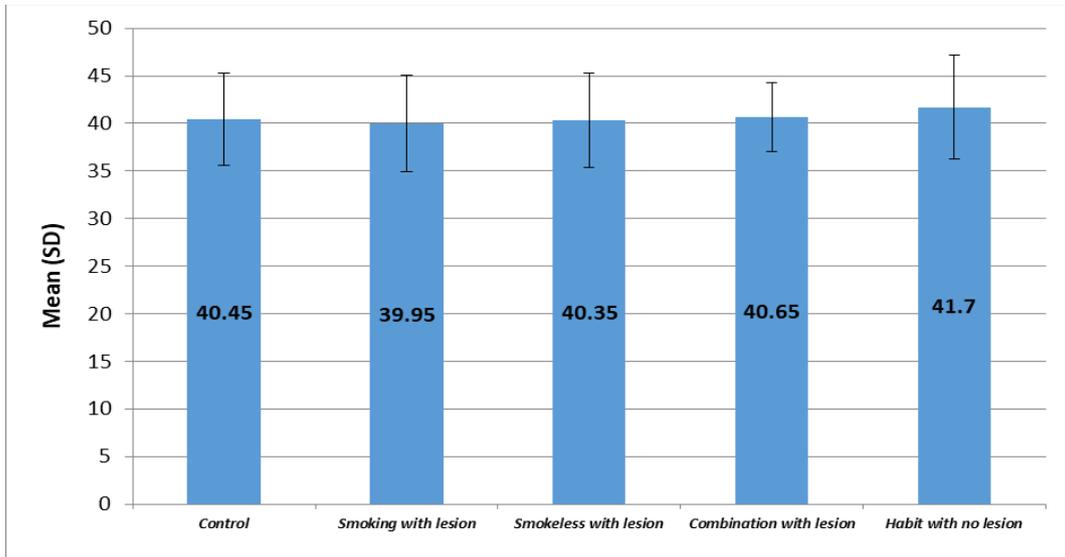


Fig 5: Comparison of arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test



(Quantitative)

Fig 6: Comparison of left ATD angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

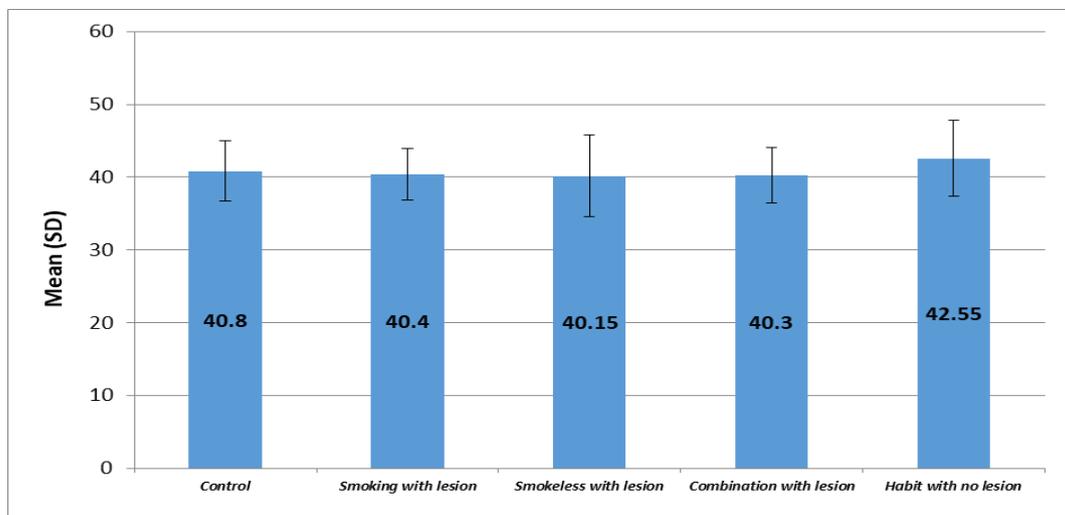


Fig 7: Comparison of right ATD angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

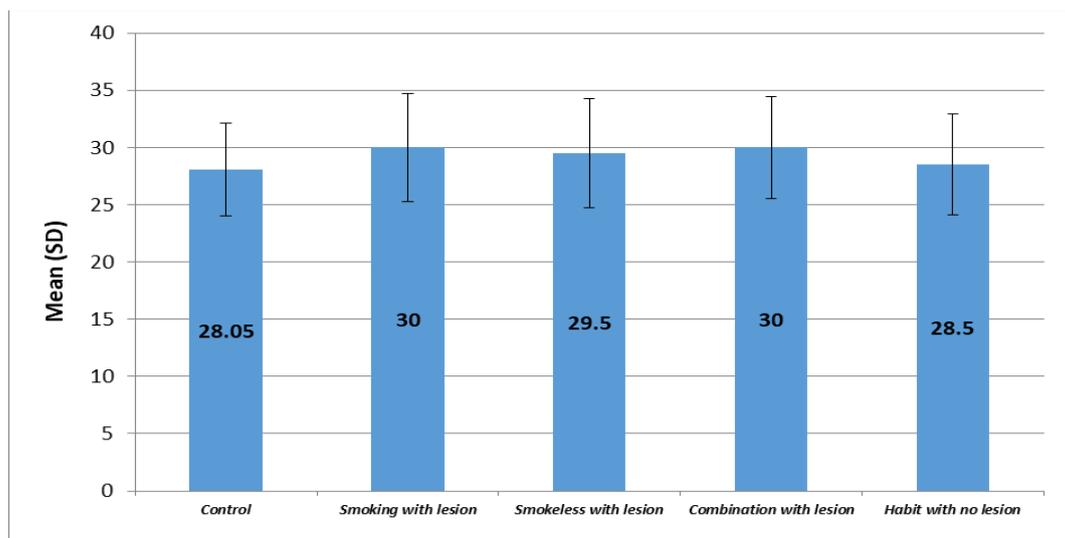


Fig 8: Comparison of left AB angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

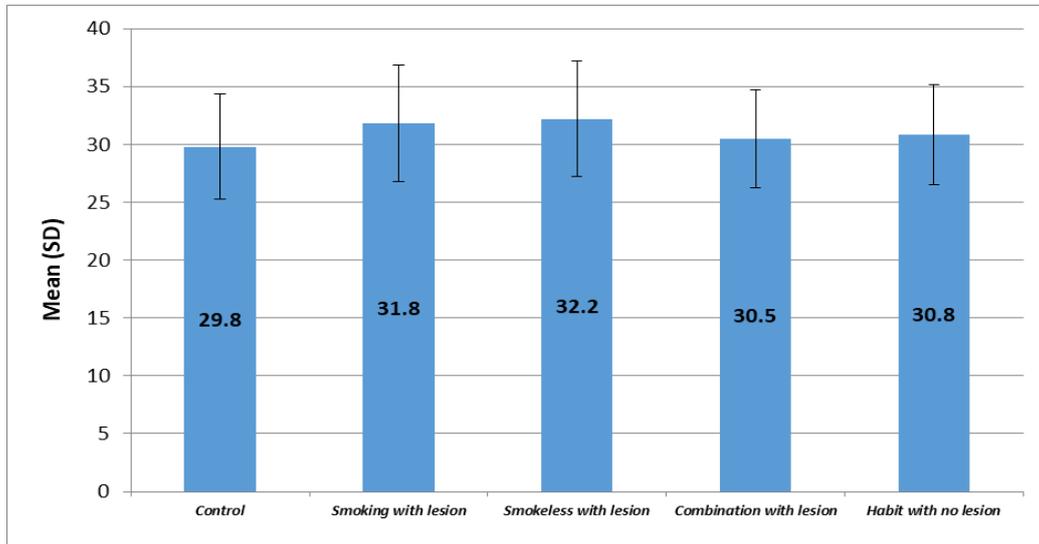
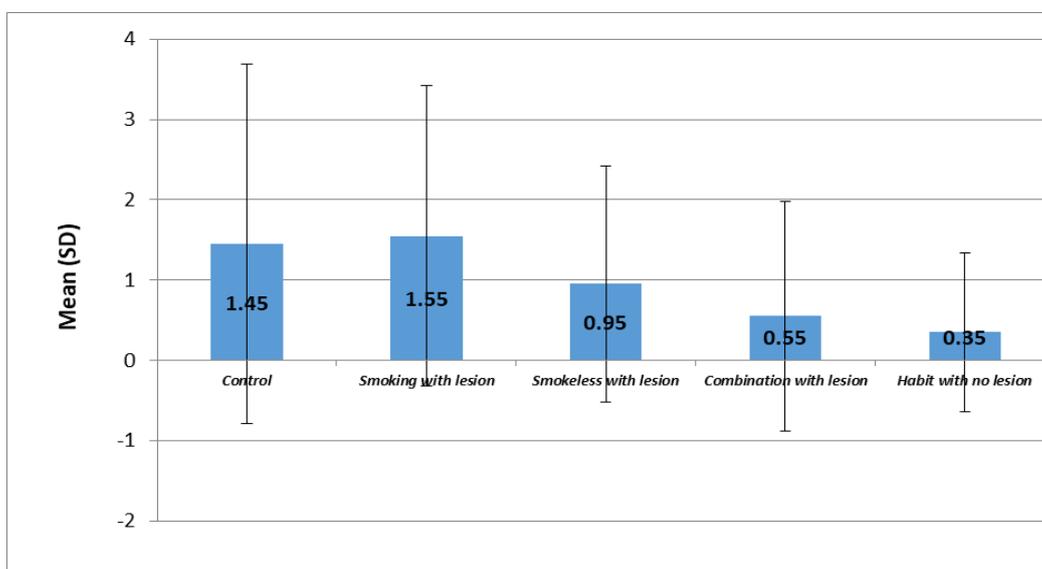


Fig 9: Comparison of right AB angle in terms of {Mean (SD)} among all the groups using Kruskal Wallis test



(Arches – Simple & tented)

Fig 10: Comparison of simple arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

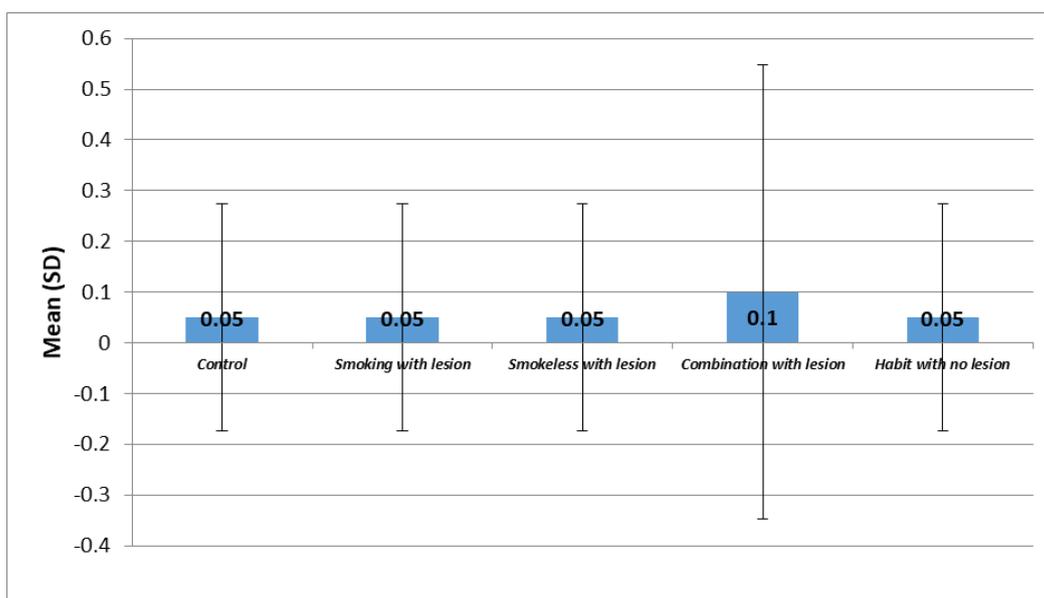
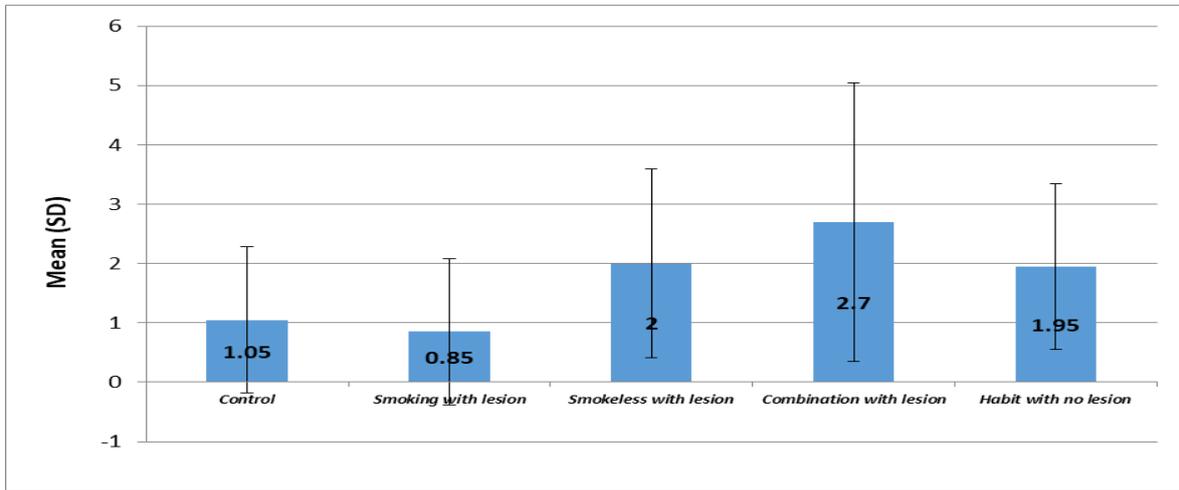


Fig 11: Comparison of tented arches in terms of {Mean (SD)} among all the groups using Kruskal Wallis test



(Whorls –Plain, spiral, central, lateral & complex)

Fig 12: Comparison of plain whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

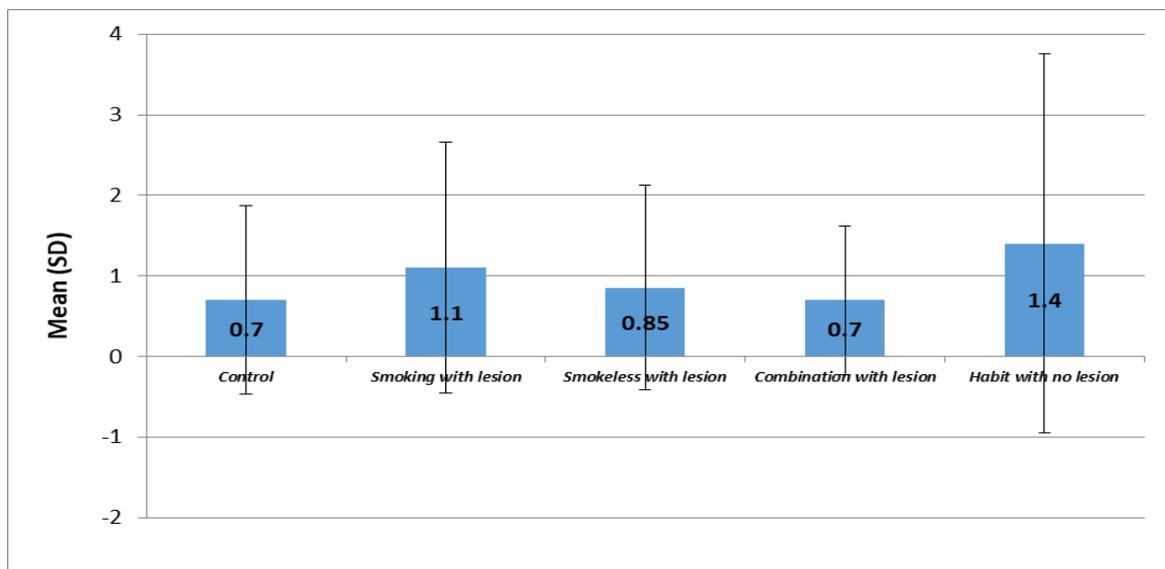


Fig 13: Comparison of spiral whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

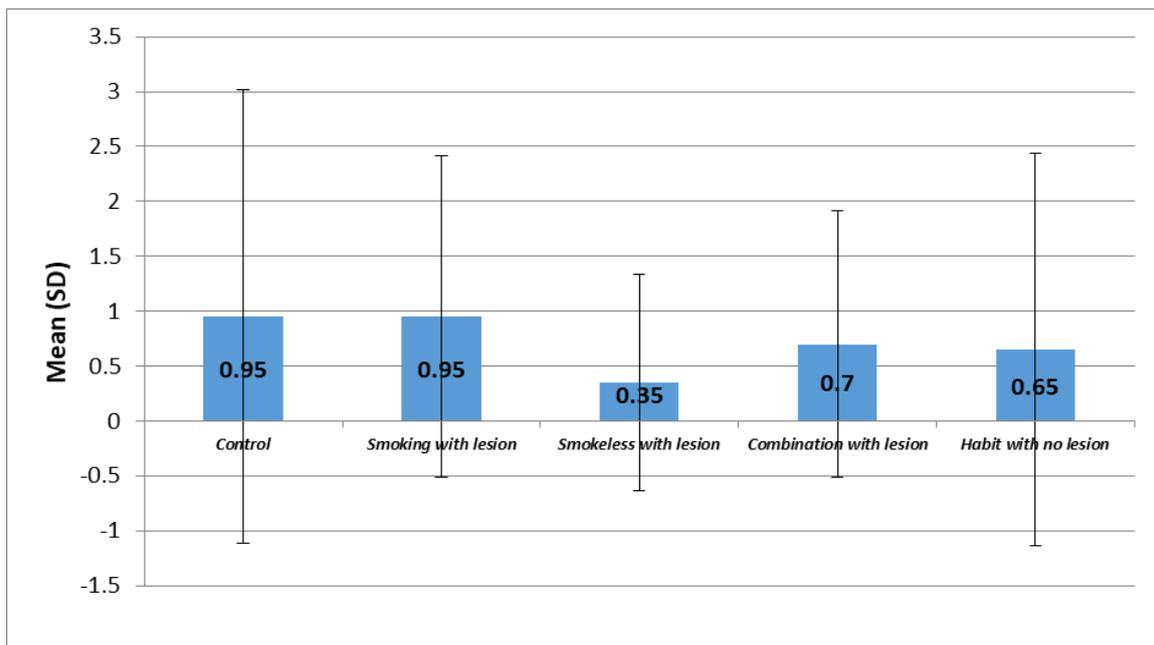


Fig14: Comparison of central whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

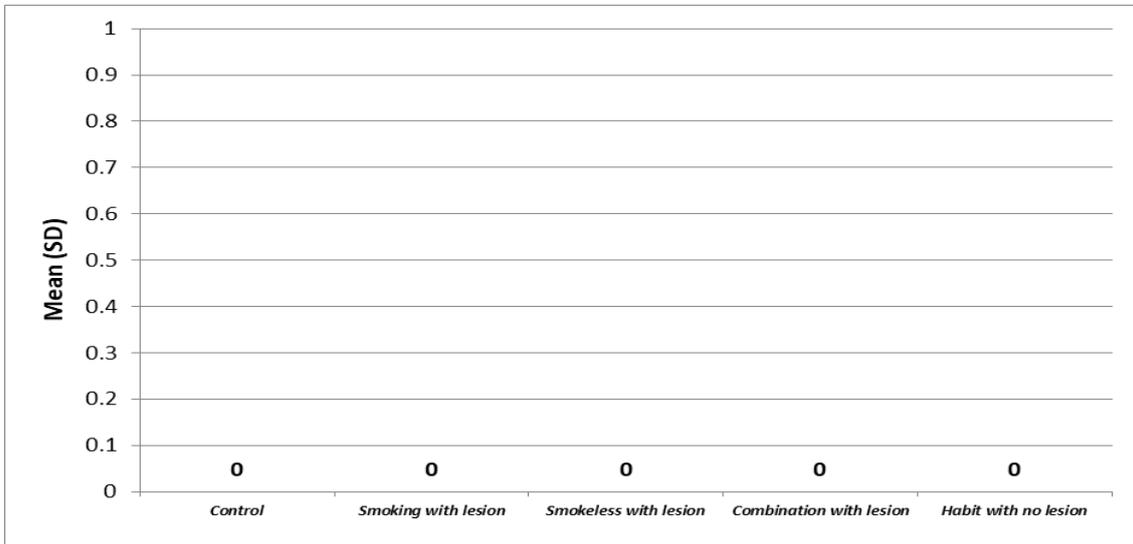


Fig 15: Comparison of lateral whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

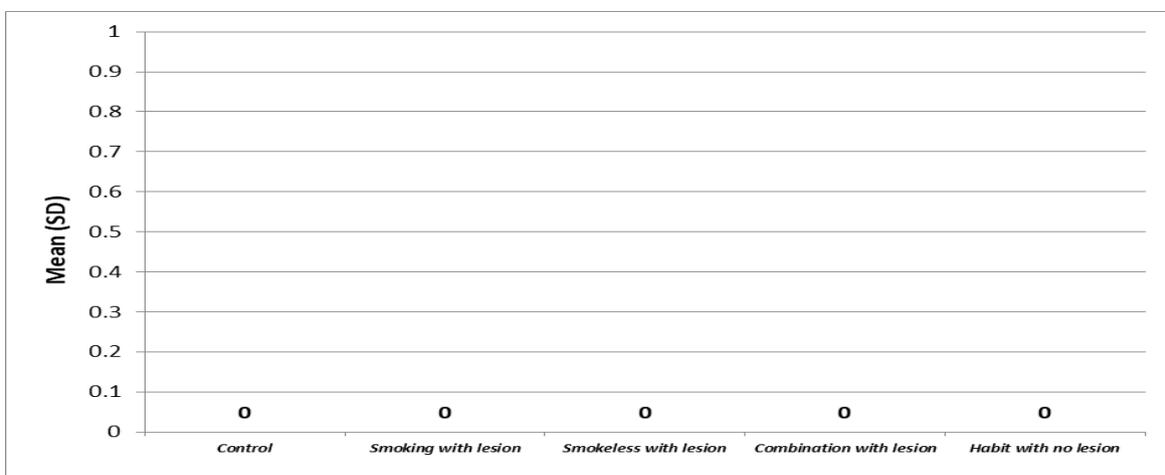
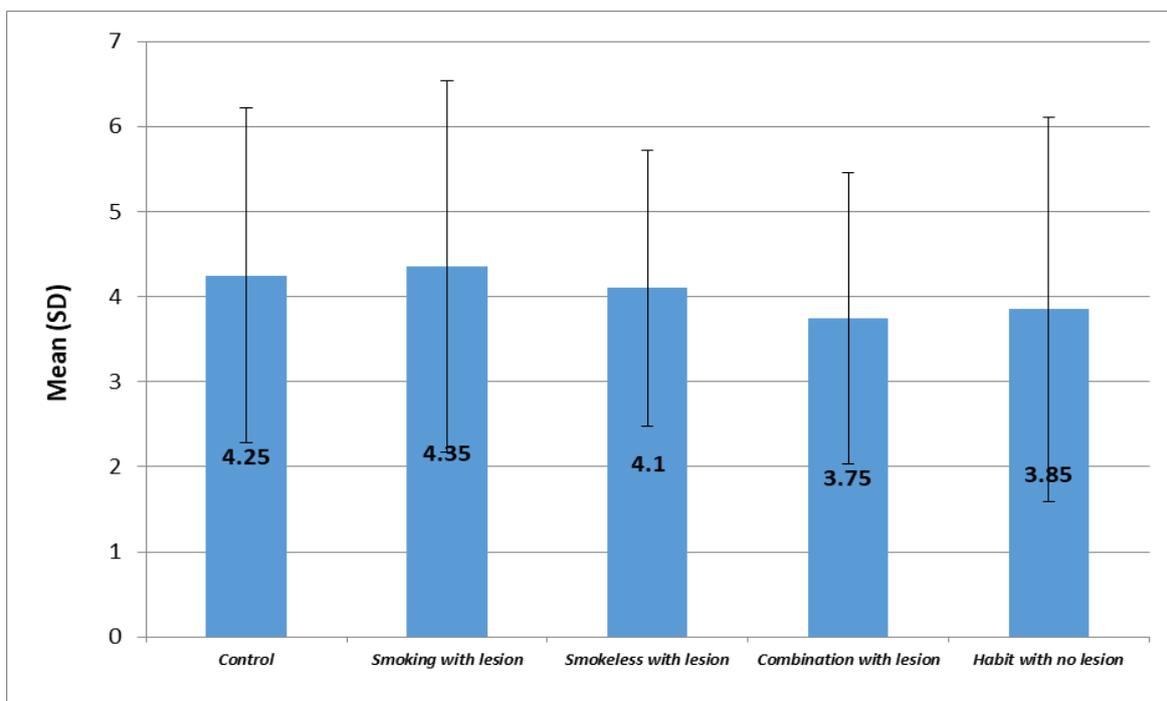


Fig 16: Comparison of complex whorls in terms of {Mean (SD)} among all the groups using Kruskal Wallis test



(Loops – Ulnar & radial)

Fig 17: Comparison of ulnar loop in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

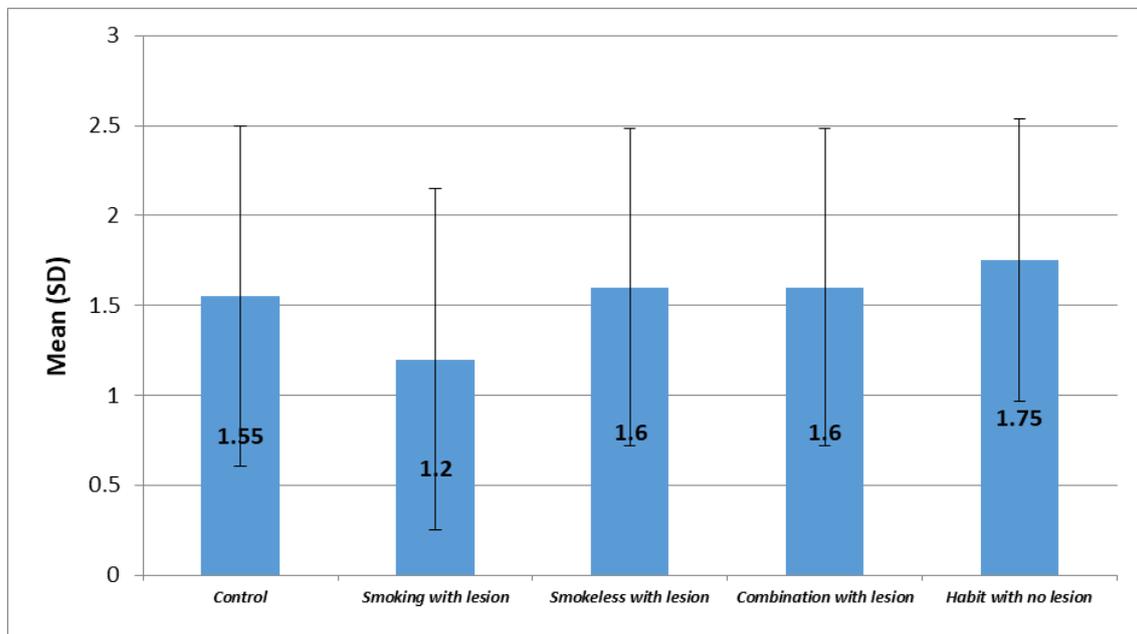


Fig 18: Comparison of radial loop in terms of {Mean (SD)} among all the groups using Kruskal Wallis test

Discussions

Dermatoglyphic analysis is beginning to prove itself a very valuable tool in diagnosis of certain precancerous conditions and lesions. Epidemiological studies show that not individuals develop precancerous lesions due to tobacco related habit. Patient without any risk factors also develop such lesions. Genetic predisposition can explain such individual variability. This genetic predisposition can be studied through palmar dermatoglyphics^[8,9]. These genetic alterations probably alter oral epithelium making it more susceptible to oral precancerous lesions. Sir Francis Galton with his extensive research demonstrated the hereditary significance of fingerprints and biological variations of different racial groups^[5]. Cummins and Midlo coined the term dermatoglyphics^[6].

This study aims at studying the importance of palmar dermatoglyphics in diagnosis of precancerous lesions and their susceptibility. Qualitative analysis shows that simple arch patterns were common in Group 2. Plain whorl patterns were common in Group 4.

Quantitative analysis shows that left and right ATD angle were prominent in Group 5. Right AB ridge count was prominent in Group 2 and Group 4. Right AB ridge count was in the range 29.8-30.50. ATD angles are in the range 40-42 degrees.

There is a paucity in literature on this subject but it has a lot of scope. Studies conducted in this field will help universalize this finding of dermatoglyphics in oral precancerous lesions.

Conclusion

To conclude, our study confirms that there is a qualitative and quantitative variation in the palmar dermatoglyphics in patients with habit of tobacco smoke/smokeless form. With our study, we concluded that there is a predominance of arches and ATD angle. Plain whorls were prominent in group 4 and simple arches in group 2 in comparison with control individuals. Therefore, we recommend use of dermatoglyphics to identify individuals who prone to develop OSMF. We suggest use of same in educational tool in genetics.

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