PALMAR DERMATOGLYPHICS IN ORAL LEUKOPLAKIA AND ORAL SQUAMOUS CELL CARCINOMA

Dissertation submitted to

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In partial fulfillment for the Degree of

MASTER OF DENTAL SURGERY

BRANCH IX
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CERTIFICATE

This is to certify that this dissertation titled **"Palmar Dermatoglyphics in Oral Leukoplakia and Oral Squamous Cell Carcinoma"** is a bonafide record of work done by **Dr.S.Ramasubramanian** under my guidance during his postgraduate study period **2009-2012.**

This dissertation is submitted to **THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY,** in partial fulfillment for the degree of **MASTER OF DENTAL SURGERY, BRANCH IX – Oral Medicine and Radiology.**

It has not been submitted (partial or full) for the award of any other degree or diploma.

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LIST OF ABBREVATIONS

S.NO	ABBREVIATION	EXPANSION
1.	OSCC	Oral Squamous Cell Carcinoma
2.	TFRC	Total Finger Ridge Count
3.	U,Lu	Ulnar loop
4.	R,Lr	Radial loop
5.	Wc	Concentric whorls
6.	Ws	Spiral whorl
7.	Wcp	Central pocket whorl
8.	WLP	Lateral pocket
9.	Wacc	Accidental pattern
10.	I_1	First inter digital area
11.	I_2	Second inter digital area
12.	I_3	Third inter digital area
13.	I_4	Fourth inter digital area
14.	t	Triradii
15.	Hy	Hypothenar area
16.	HPV	Human Papilloma Virus
17.	COE	Conventional Oral Examination

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Dermatoglyphics, coined by Cummins and Midlo in 1926, is a branch of genetics dealing with the skin ridge system. They have been studied for fortune telling by palmists and as a definitive and unalterable tool for identification by forensic experts. From cradle to grave until the body decomposes finger prints remain unchanged. Modern study of the hand has moved quite far from the popular image of the sooth saying hand reader uttering mysterious incantations in an arcane language.[1]

Rather, through decades of scientific research, the hand has come to be recognized as a very good measure in the diagnosis of psychological, medical and genetic conditions. The current state of medical dermatoglyphics is such that the diagnosis of some illness like Diabetes Mellitus, Schizophrenia, Hypertension and Epilepsy can now be aided by dermatoglyphic analysis. Currently, several dermatoglyphic research workers, claim a very high degree of accuracy, in their prognostic ability, from the hand's features.[2]

Dr. Theodore J. Berry[3] in his book "The hand as a mirror of systemic disease" has associated dermatoglyphics with 50 diseases or more, both congenital and acquired. Since most of the investigations needed to confirm the diagnosis in hereditary disorders are complex and expensive, dermatoglyphics can be efficiently employed with other clinical signs as a screening procedure to define indications for these laboratory procedures.

Many genes that take part in the control of finger and palmar dermatoglyphic development can also give indication to the development of potentially malignant disorders and malignant lesions, hence identifying

persons at high risk of developing oral leukoplakia and OSCC could be of greater value to decrease the incidence of the same.[4]

Taking these facts, into consideration, the present cross-sectional study aims to determine various dermatoglyphic features, among persons with the tobacco smoking and alcohol consuming habit without clinical evidence of premalignant and malignant lesion and compare them with the patients having oral leukoplakia and OSCC. By this, we can establish the importance of dermatoglyphics as an useful investigatory or screening procedure among persons with tobacco smoking and alcohol consuming habit, as this type of study has not been conducted in Chennai population.

AIM OF THE STUDY

Aim of the study is to determine whether specific dermatoglyphic patterns exists which help in predicting the occurrence of oral leukoplakia and oral squamous cell carcinoma

OBJECTIVE OF THE STUDY

1. To record and evaluate the finger and palm print pattern of patients diagnosed with oral leukoplakia and oral squamous cell carcinoma and control group.
2. To determine a degree of divergence and comparison of specific pattern among patients diagnosed with oral leukoplakia and oral squamous cell carcinoma and control group.

DERMATOGHLYPHICS

The study of epidermal ridges and their configurations in finger tips, palms and soles is called dermatoglyphics. The term was coined by Cummins and Midlo in 1961 from the Greek word derma means skin and glyphic means carve.[5]

HISTORY OF DERMATOGLYPHICS

In the early nineteenth century 1823, Joannes Evangelista Purkinje, Professor of Anatomy at Breslau University, drew attention in a Latin thesis to the diversity of fingerprinting patterns. He classified the finger print patterns into nine basic types.[6]

Sir Francis Galton in 1892, conducted extensive research on the significance of skin ridge patterns, not only to demonstrate their permanence, and consequently their use as a means of identification. He demonstrated the hereditary significance of fingerprints and the biological variations of different finger print patterns amongst different racial groups. He compared the finger print patterns of English, Jews, Negroes, Welsh and Basques. The frequency of pattern were same between the groups of same race and different race, however the Jews had larger proportion of whorled pattern than others. In 1892, he published the book 'Fingerprints" and in doing so, significantly advanced the science of fingerprint identification.[7]

Sir Edward Henry, during 1893 published the book 'The classification and uses of fingerprints" and with this classification system

commenced, the modern era of finger print identification, and is now the basis for most of the other classification systems.[8]

Cummins and Midlo, in 1926 were the first to coin the term 'Dermatoglyphics'. The main thrust of their research was into Down's syndrome and the characteristic hand formations. They showed that the hand with significant dermatoglyphic configurations would assist the identification of Mongolism in the newborn child. There is decrease frequency of whorls and increase in ulnar loops; a single transverse palmer crease; wide atd angle; significant deviation of axial triradii; increased frequency of patterns in hypothenar, second and third interdigital areas; and more common simian line as compared to non mongols.[9]

Charles Midlo M D, during 1929 together with others published one of the most widely referred book "Finger prints, Palms and soles", a bible in the field of dermatoglyphics.[10]

Penrose L S, in 1945 inspired by the works of Cummins and Midlo, conducted his own dermatoglyphic investigations as a further aspect of his research into Down's syndrome and other congenital medical disorders. He found that trisomy 13 is associated with distal axial triradius, 108 degrees 'atd' angle, and extra pattern in thenar region and the finger patterns have low ridge counts in Klienfilter's Syndrome. [11]

Kennedy–Galton Center, during 1965 contributed to the development of dermatoglyphics and formulated the measurement to

establish the position of displaced axial triradius in terms of atd angle, as well as establishing the inheritance of its position in the palm.[12]

Schaumann and Alter's, in 1976 published a book 'Dermatoglyphics in Medical disorders' which summarizes the findings of dermatoglyphic patterns in various disease conditions.[13]

Engler et al, in 1982 conducted a study on patients with breast cancer and concluded that the presence of six or more whorls on the fingertips of a person provided a high risk of obtaining breast cancer.[14]

EMBRYOGENESIS OF DERMATOGLYPHIC PATTERNS

William. J. Babler[6] on 1976, indicated that the epidermal ridges first appear in the form of localized cell proliferations around the 10th to 11th week of gestation. These proliferations form shallow corrugations that project into the superficial layer of the dermis. The number of ridges continue to increase, being formed either between or adjacent to existing ridges. It is during this period of primary ridge formation, that the characteristic patterns are formed. At about 14 weeks, the primary ridge formation ceases and secondary ridges begin to form as sweat gland anlagen, and develop along the apices of the primary ridges at uniform intervals. At this time, the epidermal ridges first begin to appear on the volar surfaces. The dermal papillae are reported to develop in the valleys between the ridges on the deep surface of the epidermis around the 24th week. Till then, the morphology of primary and secondary ridges appear as a smooth

ridge of tissue and thereafter peg like structures, the dermal Papillae, characteristic of the definitive dermal ridges progressively formed.

Babler in 1987, reports that there is a relationship between the volar pad shape and the epidermal ridge configuration, specifically narrow volar pads are related to whorl patterns. He also suggested the association between the shape of the distal phalanx and the pattern type. Significant correlations between the bony skeleton of the hand and the epidermal ridge dimensions and time of ossification may be a key factor in ridge patterning.[15]

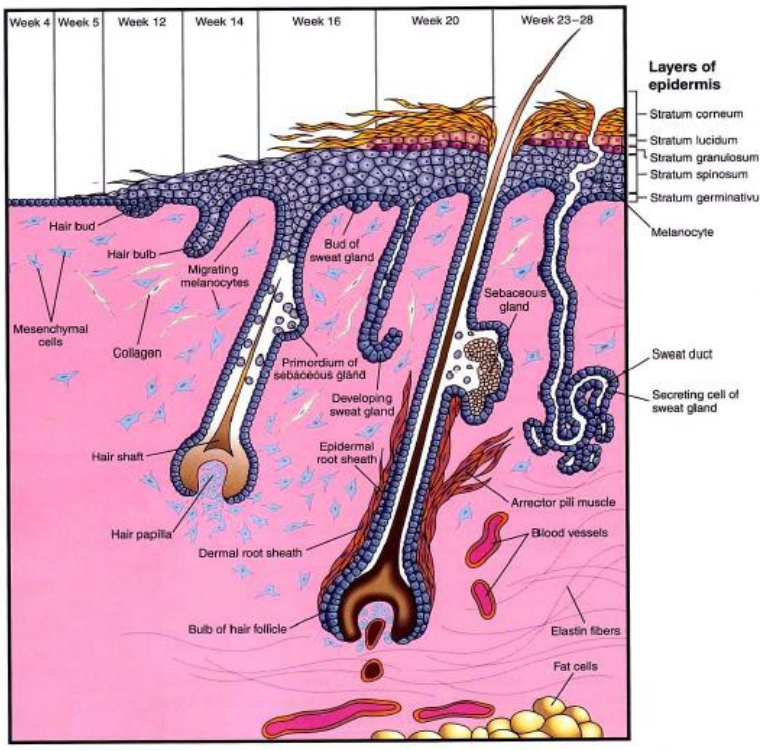


Fig. 1: Development of Epidermal Ridges

PATTERN CONFIGURATIONS

Fingertip pattern configurations

Galton (1892), divided the ridge patterns on the distal phalanges of the fingertips into three groups. [16]

1) Arches
2) Loops
3) Whorls.

Although numerous sub classifications have been subsequently offered, this simple classification is still recognized and used by majority of investigators today.

1) Arches:

It is the simplest pattern found on fingertips. It is formed by succession of more or less parallel ridges, which traverse the pattern area and form a curve that is concave proximally. Sometimes, the curve is gentle; at other times it swings more sharply so that it may also be designated as a low or high arch respectively.[2]

The arch pattern is subdivided into two types.

a) Simple arch or plain arch (A) composed of ridges that cross the fingertip from one side to the other without recurving.[17]

Fig. 2: Simple Arch

b) Tented arch (T or A1) composed of ridges that meet at a point so that their smooth sweep is interrupted. The point of confluence is called a triradius, because ridges usually radiate from this point in three different directions. In the tented arch, the triradius is located near the midline axis of the distal phalanx. The distal radiant of the triradius usually points vertically toward the apex of the fingertip. Ridges passing over this radiant are abruptly elevated and form a tent like pattern and are designated as 'tented arch'.[17]

Fig. 3: Tented Arch

(2) Loops

It is the most common pattern on the fingertip. A series of ridges enter the pattern area on one side of the digit, recurve abruptly, and leave the pattern area on the same side. If the ridge opens on the ulnar side, resulting loop is termed as ulnar loop (U,Lu) If the ridge opens toward the radial margin, it is called a radial loop. (R,Lr) A loop has a single triradius or confluence point of ridges. The triradius is usually located laterally on the fingertip and always on the side where the loop is closed.[18]

Loops may vary considerably in shape and size. They may be large or small, tall or short, vertically or horizontally oriented. Occasionally, 'Transitional' loops can be found which resemble whorls or complex patterns.[19]

Fig. 4: Loops

(3) Whorls

It is any ridge configuration with two or more triradii. One triradius is on the radial and the other on the ulnar side of the pattern.[20]

Henry on 1937, limited the designation of the term 'Whorl' to those configurations having ridges that actually encircle a core. He named more complex patterns as "Composites.".[3]

The ridges in a simple whorl are commonly arranged as a succession of concentric rings or ellipses. Such patterns are described as concentric whorls (Wc). Another configuration spirals around the core in either a clockwise or a counter clockwise direction. This pattern is called a spiral whorl (ws).[21]

Sometimes, both circles and ellipses or circles and spirals are present in the same pattern. The size of the whorl can vary considerably, and is determined by means of a ridge count.[22]

A central pocket whorl (Wcp) is a pattern containing a loop within which a smaller whorl is located. Central pockets are classified as ulnar or radial according to the side on which the outer loop opens. The significance of separating these two varieties of loop whorls for medical diagnosis remains unproved. Therefore, they are ordinarily grouped together as a double loop. Another type is composed of interlocking loops, which may form either a lateral pocket (WLP) twin or twinned loop (wt) pattern Each has two triradii and the two types of whorls are morphologically similar.[23]

Complex patterns, which cannot be classified as one of the above patterns, are called accidentals (Wacc). They represent a combination of two or more configurations such as a loop and a whorl, triple loops and other unusual formations.[24]

Fig. 5: Double Loop Whorl

Fig. 6: Target Whorl

DERMATOGLYPHIC LANDMARKS

The three basic Dermatoglyphic landmarks found on the fingertip patterns are

- ❖ Triradii
- ❖ Cores
- ❖ Radiant.

Triradius

It is formed by the confluence of three ridge systems. The geometric center of the triradius is designated as a triradial point. It is the meeting

point of three ridges that form angles of approximately 120° with one another. Around the core of a loop, the direction of the ridges turns through an angle of 180°. However, if the three ridges fail to meet, the triradial point can be represented by a very short, dot like ridge called an island or by a ridge ending or it may lie on a ridge at the point nearest to the center of the divergence of the three innermost ridges. Sometimes, the triradial point does not lie on a ridge and is determined as the point where three angles between the innermost ridges are each as near as possible to 120°. [24,25]

The triradial point forms one terminus of the line along which ridges are counted. Sometimes, large patterns are extralimital. These are commonly observed in the hypothenar areas of the palms and the hallucal areas of soles.[21]

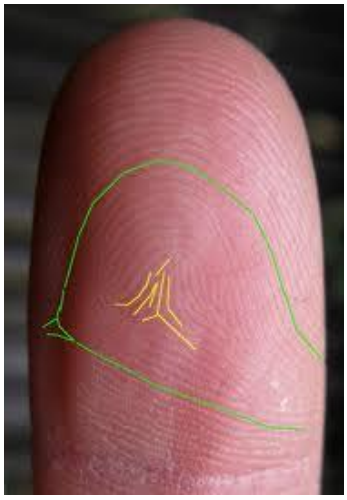


Fig. 7: Triradii

Core[7]

It is in the approximate center of the pattern. The core may be of different shapes.

A. In a loop pattern, the core is usually represented by a straight, rod like ridge or a series of two or more such parallel ridges, over which other recurving ridges pass. If a straight ridge is absent in the center of the loop, the innermost recurving ride is designated as a core.
B. In a whorl, the core can appear as a dot or a short ridge (either straight or bent) or it can be shaped as a circle or an ellipse in the center of the pattern.

Radiants[7]

These are the ridges that emanate from the triradius and enclose the pattern area. These ridges constitute the 'skeletal' framework of the pattern area.

PALMS

Palmar Pattern Configuration

In order to carry out dermatoglyphic analyses that can be compared in different individuals, the palm has been divided into several anatomically designed areas. It includes thenar area, four interdigital areas and hypothenar area.[26]

Thenar and first interdigital areas (Th / I1)

There is no pattern in the Th / I1 area, but the ridges follow a mild curve around the base of the thumb. Sometimes, the simple flow is disturbed by an area of abruptly disarranged ridges, which are oriented at an angle to the general direction of other ridges in the area. They do not form a true pattern. Hence, this configuration is called a vestige.[26]

Second, third and fourth interdigital areas

These areas are found in the distal palm in the region of the heads of the metacarpal bones. Each interdigital area is bordered laterally by digital triradii. Digital triradii are labelled a,b,c and d. The second interdigital area (I2) lies between triradii a & b, the third interdigital area (I3) between triradii b & c, and the fourth interdigital area (I4) between triradii c & d. If a digital triradius is absent, the midpoint of the base of the corresponding digit can be used to separate the interdigital areas.[26]

Configurations encountered in the interdigital regions are loops, whorls, vestiges and open fields.[9]

Hypothenar area

True patterns are commonly present in the hypothenar area (Hy). The patterns are whorls, loops, and tented arches. Simple arches, open fields, vestiges and ridge multiplications also occur. The triradius or triradii close to the palmar axis are termed axial triradii (t) symbols t, t' and t'' are used to designate the position of these triradii in the proximal – distal direction on the palm.[26]

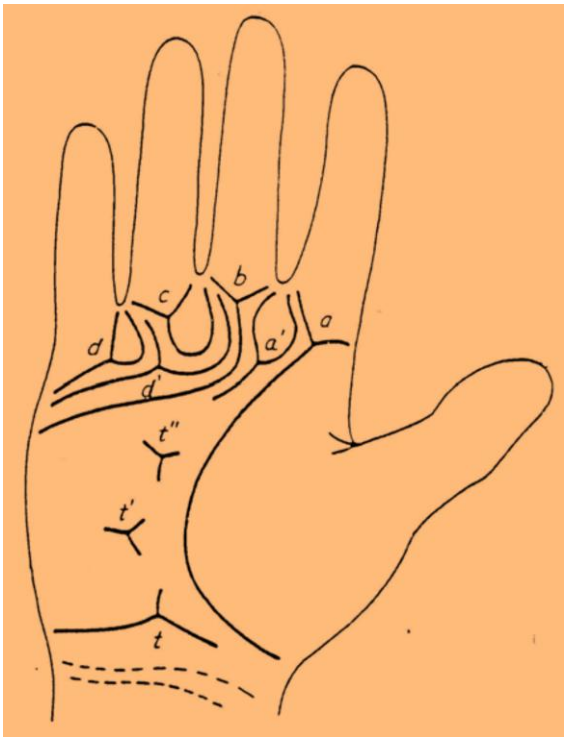


Fig. 8: Palmar Triradii

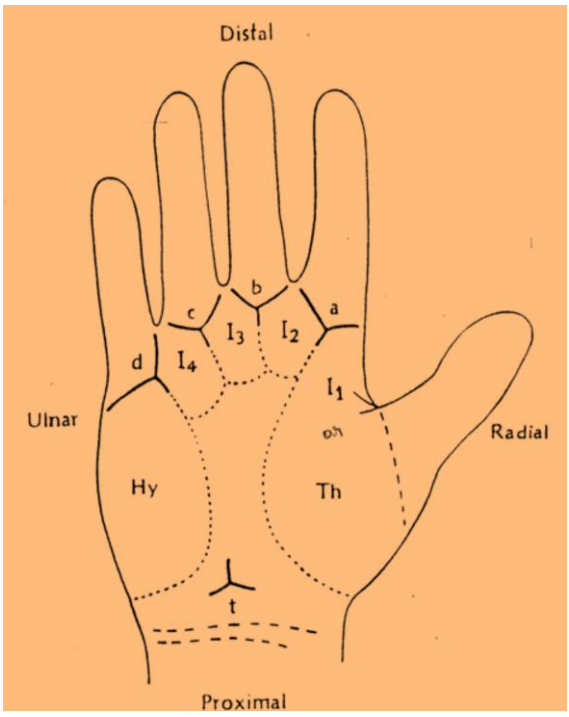


Fig. 9: Palmar Dermatoglyphic Pattern Areas

RIDGE COUNTING

It is used to indicate the pattern size. The counting is done along a straight line connecting the triradial point to the point of core. The ridges containing the point of core and triradial point are both excluded from the count. Whorls that possess two triradii and at least one point of core allow two different counts to be made, one from each triradius. Because the ridge counts are used to express the pattern size, only the largest count is scored in a pattern with more than one possible count. Both simple and tented arches have 0 counts.[3]

A total finger ridge count (TFRC) represents the sum of the ridge counts of all ten fingers, where only the larger count is used on those digits, with more than one ridge count.[27]

An absolute finger ridge count (AFRC) is the sum of the ridge counts from all the separate triradii on the fingers.[27]

The TFRC expresses the size of pattern, whereas the AFRC reflects the pattern size as well as the pattern intensity, which depends on the pattern type.[28]

Ridges are often counted between two digital triradii. The ridge count most frequently obtained is between triradii a and b, and is referred to as the a-b ridge count.[29]

atd ANGLE[30]

This angle is formed by lines drawn from the digital triradius (a) to the axial triradius (t) and from this triradius to the digital triradius (d). The more distal the position of‘t’, the larger the atd angle. Sometimes accessory a' or d' triradii are present on the palm.

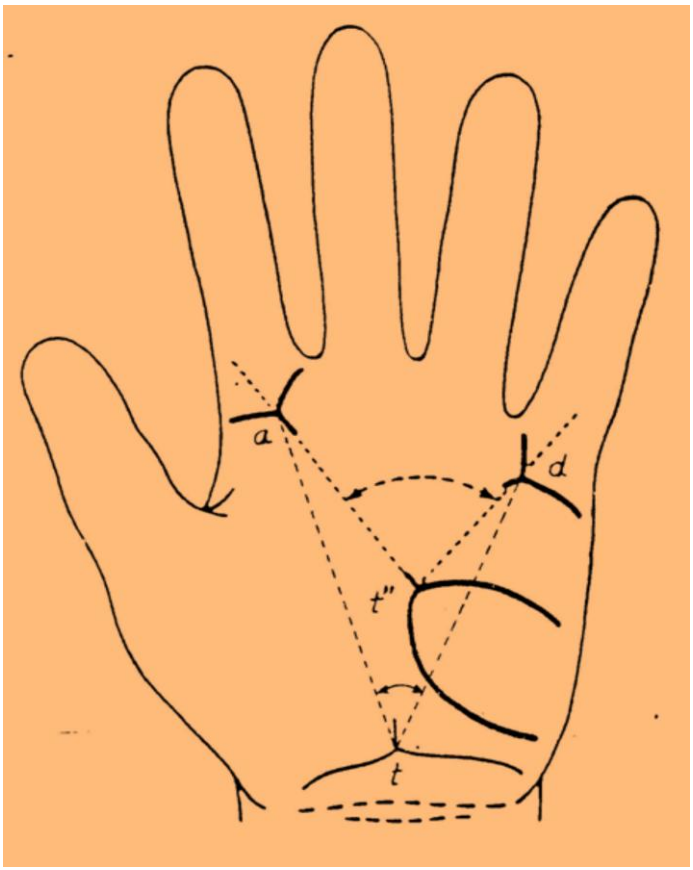


Fig. 10: Maximal and Minimal atd Angle

METHODS OF PRINTING

1. In most individuals other than newborn infants, the dermal patterning can be observed directly without magnification, or with the aid of a simple hand lens and good direct lighting. In infants,

direct observation by the use of an Otoscope without speculum, a simple lens attachment provides adequate magnification.[31]

2. Walker on 1957, described Faurot Inkless Method: This method makes use of a special fluid and sensitized paper. In this method, palm and sole are rubbed well with a cloth pad soaked in the fluid and then pressed lightly on the sensitized paper. It is advisable to place a sponge rubber pad beneath the paper when the print is being obtained. Care must be taken not to apply too much fluid or pressure, as the resulting points will be dark and smudged. Excellent descriptions of the above techniques were also presented by walker for children over 4 years. This method works well for adults.[32]
3. Hollister printer method: It is one of the most convenient methods, which gives satisfactory results in most instances. In this method, the hands and feet are placed on a pad covered with a special ink and then are pressed on a special paper which has a relatively hard and glossy surface. The baby's hand and foot and then pad must be warm, clean and dry or the prints will be blurred. Excess ink on the infants hands and feet can be removed easily with soap and warm water.[33]
4. Photographic method: Inked impression of the fingertip on paper is sensitive to environmental factors and the skin condition, and consequently many fingerprint images acquired this way are of poor quality. Photographic method was used by Achs, Harper and Seigal

during 1966. It may prove useful in dermatoglyphic analysis.[30] Palm print can be captured by widely used CCD based palmprint scanners, CIS based Digital Scanner, video cameras and Digital cameras. The digital scanner can acquire high resolution hand image but requires more time to scan. Digital and video cameras can also be used to collect palm print images and these images might cause recognition problem as their quality is low because they collect image in an uncontrolled environment with illumination variations and distortions due to hand movement.[34]

5. Andersen & D Kosz on 1993, in their study, used new numerical methods of fingerprints. Algorithm of synthesis of images of dermatoglyphics, and in particular all the possible arrangements of so-called minutiea is created. The model allows to look at digital coding of a fingerprint from a new point of view, not only as a set of pixels, but some two dimensional function of very interesting qualities. It also enables mathematical cataloguing of minutiae and types of patterns, and this means revolution in methods of analyzing, processing and compression of fingerprint images.[35]

ORAL LEUKOPLAKIA

Definition

WHO collaborating centre for oral precancerous lesions in 1978[36] defined oral leukoplakia as "A white patch or plaque that cannot be characterized clinically or pathologically as any other disease."

Axell T et al in 1996 [37] also defined oral leukoplakia as "A predominantly white lesion of oral mucosa that cannot be characterised as any other definable lesion;some leukoplakia will transform in to cancer.

Pindborg et al in 1997 [38] defined leukoplakia as " A predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion".

WHO in 2005[39] declared " Leukoplakia should be used to recognize white patch of questionable risk having excluded other known diseases or disorders that carry no increased risk for cancer.

Van der Waal I et al [40] reviewed Potentially malignant disorders of the oral and oropharyngeal mucosa; terminology, classification and present concepts of management and gave WHO workshop recommendations such as to abandon the distinction between potentially malignant lesions and potentially malignant conditions and to use the term potentially malignant disorders instead.

Epidemiology

Prevalence of leukoplakia was reported to be 3.6% and that of preleukoplakia was 6.4%. Idiopathic leukoplakia was reported to be 0.7% and tobacco specific leukoplakia was 2.9%.[41]

Age and Gender

The onset of lesions usually starts after 30 years, resulting in peak incidence of 50 years. Leukoplakia is seen most frequently in middle aged and older men, with an increasing prevalence with age. Oral leukoplakia can occur 5 years prior to oral cancer.[42,43]

It has a strong male preponderance. Leukoplakia is a commonly occurring lesion particularly in patients after 40 years of age. The male to female ratio is 2:1. The gender distribution in most studies varies, ranging from a strong male predominance in different parts of India, to almost 1:1 in Western world.[44]

Bánóczy J[45] made a follow-up study with 670 patients with oral leukoplakia during a 30-year-period showed cancer development in 40 cases. The age distribution revealed the prevalence of leukoplakia in the age-group 51-60 years; that of carcinoma in the age-group of 61-70 years. The sex distribution showed a male-female ratio of 3.2: 1 in the leukoplakia-group, and a 1.9: 1 ratio in the carcinoma-group.

Etiology

Causative Factors in Leukoplakia[46]

Local	Systemic
Local irritation Sharp ,malposed teeth Ill fitting denture Poor restorations	Heredity
Occlusal disharmony	Hormonal factors
Occlusal habit	Estrogen deficiency
Thermal factors	Nutritional deficiency
Smoking	Syphilis
Irritant foods,chemicals,mouthwashes, etc.	Atrophic glossitis

Dietrich T, [47] made an analytical study on Clinical risk factors of oral leukoplakia and found the results as, Tobacco smoking as the strongest independent risk factor. The Odds Ratio were 3.00 (0.77-11.8) for < for =10 cigarettes/day and up to 6.01 (2.4-15.0) for >20 cigarettes/day. Diabetes, age and socio-economic status were found as independent predictors of Oral leukoplakia. Alcohol consumption, race/ethnicity, years of education and Body Mass Index showed no independent association with Oral leukoplakia.

Females with a history of estrogen use were less likely to have Oral leukoplakia with an Odds ratio of 0.34 (0.11-1.07).

Prakash C.Guptha[48] made an Epidemiologic study of the association between alcohol habits and oral leukoplakia. The study included 10914 individuals for their tobacco and alcohol habits and examined for the presence of oral leukoplakia. Very few females (1.6%) were found to be alcohol users and they were excluded from further analysis. Among 7604 males, 30.4% used alcohol regularly, 25.4% occasionally and 44.2% were non-users. The prevalence of leukoplakia was significantly higher among regular (5.7%) and occasional (3.9%) users than among non-users (2.9%) of alcohol.

Clinical types[39]

Two main type exists:

- Homogeneous
- Non homogeneous

Distinction between these two forms is purely clinical, based on surface color and morphological characteristics like thickness which also has predilection for prognosis.

Homogeneous type

Homogeneous leukoplakia has been defined as a predominantly white lesion of uniform flat, thin appearance that may exhibit shallow cracks and has a smooth, wrinkled or corrugated surface with a constant texture throughout. The risk of malignant transformation is relatively low. The

lesion is predominantly white but can be grayish white. It constitutes for about 84% of the leukoplakia.

Non homogeneous type

- Ulcerative: Mixed white and red in color but retaining the predominant white character.
- Nodular (Speckled): Small polypoid outgrowths, rounded red or white excrescences.
- Verrucous: wrinkled or corrugated surface appearance.

The term "Erythro leukoplakia" is applied for predominantly red and white lesion that may be irregularly flat, nodular or exophytic. The nodular lesions are characterized by white patches or nodules on a erythematous base.

Clinical Features[44]

Most commonly involved sites are retro commissural area, buccal mucosa, edentulous alveolar ridge, hard palate, tongue, lips. The gingival, soft palate and floor of mouth are less commonly involved in an Indian population, where as it is not true for Western population.

Leukoplakia begins as thin, gray white plaques that may appear somewhat translucent, sometimes fissured or wrinkled and are soft and flat.

Leukoplakia Clinical Phases [46]

Phase	Descriptive terms	Risk of malignant transformation
I	Thin leukoplakia Preleukoplakia Homogeneous leukoplakia	+/-
II	Thick,smooth leukoplakia Fissured leukoplakia Homogeneous leukoplkia	++
III	Granular leukoplakia Verruciform leukoplakia Rough leukoplakia Candidal epithelial hyperplasia Homogeneous leukoplakia	+++
IV	Erythroleukoplakia Speckled leukoplakia Candidal leukoplakia Nonhomogeneous leukoplakia	++++

Classification and staging of Oral Leukoplakia

Pindborg et al in 1997[38] has given the classification and staging for leukoplakia as follows,

Provisional (Clinical Diagnosis)

L : Extent of leukoplakia

L0 : No evidence of lesion

L1 : ≤ 2 cm

L2 : 2-4 cm

L3 : ≥ 4cm

S : Site of leukoplakia

S1 : all sites excluding floor of mouth & tongue

S2 : floor of mouth &/ tongue

S3 : not specified

C : Clinical aspect

C1 : homogeneous

C2 : non homogeneous

C3 : not specified

Definitive diagnosis:(Histopathological diagnosis)

P : Histopathological features

P1 : no dysplasia

P2 : Mild dysplasia

P3 : Moderate dysplasia

P4 : severe dysplasia

Px : not specified

Staging:

1. any L,S1,C1,P1 or P2
2. any L,S1or S2,C2,P1 or P2
3. any L,S2,C2,P1 or P2
4. any L,any S,any C,P3 or P4.

Natural History

Leukoplakia can regress spontaneously without any intervention in habit or by any other means in about 40% of cases. Significantly higher rates of regression is seen who discontinue the tobacco habit. In one long term follow-up study among the Swedish population consisting 104 samples, they found that oral leukoplakia has disappeared in 43% of the people. About 70-80% of leukoplakia is associated with tobacco habits, also about 80% of the leukoplakia lesions disappear completely about 58% or regress within 12 months after smoking cessation.[49]

Malignant Transformation

It is generally accepted that dysplastic lesions carry a 5 fold greater risk than non dysplastic ones. It refers to the development of oral cancer from preexisting oral leukoplakia. So it is necessary to follow-up a case of leukoplakia for a period of 3 months to one year.[50]

In the period of follow-up, the lesion should be evaluated for development of thickened/nodular areas, ulcerations, rolled margins, growths or indurated areas. Since these changes represent early oral cancers. Lesions on the tongue, lip vermilion border, floor of the mouth

accounts for 93% of the leukoplakia with dysplastic changes or carcinoma. Globally 3-6% leukoplakia change to cancer.[51]

Non homogeneous leukoplakia accounted for the highest frequency of malignant transformation of 20%,whereas 3% of the homogeneous leukoplakia developed carcinoma. Proliferative verrucous leukoplakia has a malignant transformation rate as high as 70.3% with mean follow-up of 11.6%.[50]

ORAL SQUAMOUS CELL CARCINOMA

Oral cancer encompasses all cancers developing in the oral cavity and pharynx. Approximately 90% of all oral malignancies are squamous cell carcinomas that originate in the epithelial mucosa lining the oral cavity and its tissues. Oral squamous cell carcinoma (OSCC) is frequently the cancer-type referred to with the general term "oral cancer".[52]

Epidemiology and Etiology

Oral cancer occurs predominately in adult males than females, aged 50 years and older with a history of tobacco and alcohol use, the primary risk factors for oral cancer. These risk factors account for the high incidence rates found in populations where cultural and social use of tobacco and/or alcohol are common, such as Western Europe, Southeast Asia, and Melanesia. In many regions, men exhibit greater prevalence than women, with incidence rates of 7.9 per 100,000 males versus 3.3 per 100,000 females, due to higher proportion of smoking and drinking habits in men.[53-55]

Interestingly, these factors appear to act individually or synergistically, with up to 100 times higher risk in heavy smokers and heavy drinkers.[52]

Tobacco in all forms, including cigarettes, cigars, pipe tobacco, or smokeless tobacco such as chewing tobacco, snuff, and betel quid, increases the risk of oral cancer. Betel quid, common in India, Southeast Asia and the South Pacific islands, consists of a betel leaf that is wrapped around a mixture of areca nut and slaked lime with tobacco and sweeteners. In the past decade, there has been an alarming increase in the popularity of cheap, ready-packaged chewing tobacco that is often chocolate or mint candy flavored, among children in India over traditional betel quid. This trend has lead to an increase in malignant lesions and potentially malignant disorders of the buccal mucosa in younger Indian populations, <50 years old.[54,56,57]

In addition, recent studies have linked high-risk HPVs (human papiloma virus-16 and 18) to oral cancer development in up to 25 % of all OSCC cases.[58,59] HPV, one of the most common sexually transmitted diseases worldwide, may partially account for the increase in oral cancer among young adults 20-45, particularly those located on the tongue and tonsil.[60] HPV-associated OSCC may display distinct molecular, clinical, and pathological characteristics along with significantly improved prognosis (59% reduction in risk of death) versus non-HPV OSCC.[58,59] Additional

factors which may play a role in oral carcinogenesis include genetic susceptibility, diet, Epstein-Barr Virus infection and immunosuppression.[61]

Oral cancer incidences for men and women according to geographic regions as reported in GLOBOCAN 2002 statistics.[62]

Region/Country	Age-Standardized Incidence Rate of Oral Cancer (per 100,000)[32]	
	Male	Female
North America	7.8	3.3
United States	7.9	3.3
Canada	6.9	2.9
Southern Africa	11.1	3.1
Botswana	23.1	9.5
Namibia	16.1	7.2
Lesotho	2.9	1.6
South African Republic	11.2	2.9
Swaziland	2.4	1.4
South Central Asia	12.7	8.3
Afghanistan	6.8	5.9
Bangladesh	13.4	16.8
Bhutan	12.8	8.4
India	12.8	7.5
Iran	2.9	1.7

Kazakhstan	14.9	2.7
Kyrgyzstan	8.1	1.7
Nepal	12.8	8.4
Pakistan	14.7	14.7
Sri Lanka	24.5	9.2
Tajikistan	2.6	1.3
Turkmenistan	12.9	3.3
Uzbekistan	9.3	2.3
Western Europe	11.3	2.7
Austria	11.3	1.7
Belgium	7.7	2.5
France	14.8	2.7
Germany	11.1	2.8
Luxembourg	9.0	2.7
The Netherlands	5.6	3.3
Switzerland	9.0	2.5
Australia/New Zealand	10.2	4.5
Melanesia	31.5	20.2
Fiji	1.9	1.4
Papua New Guinea	40.9	26.3
Solomon Islands	34.1	21.7
Vanuatu	3.7	2.0

Clinical Features

OSCC is found most frequently in the lateral tongue, representing approximately 40% of all cases, and the floor of the mouth.[52] The high-risk of malignancy at these sites is attributed to the pooling of saliva containing carcinogens in these areas, as well as the lack of protection afforded by the thin, non-keratinized epithelium present.[63] A large number of squamous cell carcinomas also develop in the lower lip vermilion border due to excessive sun exposure, but typically possess low risk of metastasis.[52] Asian population usually suffer from cancer of the buccal mucosa due to betel quid/tobacco chewing habits; Buccal mucosa SCC constitute 40% of OSCC in indian population.

The most common symptom is a non-healing sore or ulcer. Other potential signs and symptoms include pain, numbness, a persistent lump or thickened area, a persistent red or white patch, dysphagia, sore throat or the sensation of something "caught" in the throat.[64]

The clinical appearance of OSCC is variable. It can be exophytic (growing outward) or endophytic (growing inward), and may have an ulcerated surface. OSCCs are characteristically firm on palpation, which can be a helpful diagnostic clue. The color of OSCC can be white, red or, in many cases, speckled red and white.[64]

Advanced metastatic spread of OSCC regularly encompasses multiple oral sites and/or cervical lymph nodes with greater than 50% of all

OSCC cases showing regional lymph node involvement at initial diagnosis.[57]

Genetic Alterations in OSCC

In a study by Rosin *et al.*[65] risk of cancer development from potentially malignant disorders was low in the absence of genetic alterations, increased moderately in the presence of genetic mutations on chromosomes 3p and 9p, and high when 3p and 9p mutations were accompanied by additional loss in one or more chromosomal regions (including 4q, 8p, 11q, 13q and 17p). The continued accumulation in genetic mutations as a result of exposure to carcinogens, such as tobacco and alcohol, ultimately leads to wide-spread genomic instability associated with advanced cancer progression and metastasis.

Current Detection of Oral Cancer and Pre-malignant Lesions

Currently, detection of oral cancer and potentially malignant disorders relies upon visual inspection of the oral cavity for mucosal abnormalities in a process known as conventional oral examination (COE). Dental professionals and primary care physicians who see patients regularly are more likely to identify early-stage lesions through yearly cancer related check-ups, as recommended by the American Cancer Society.[66]

In a recent systematic review of seven studies evaluating COE as a method for detecting early cancerous lesions, sensitivity ranging from 60% - 97% and specificity ranging from 75% - 99% were reported, which are

comparable to rates found in other cancer screening programs.[67] This suggests that COE may be an adequate screening method to identify oral lesions. Shortcomings of this method include the inability to detect sub-clinical abnormalities or discriminate between benign lesions and those with a high-risk of malignancy which may require the use of adjunctive diagnostic techniques.[68] Further, the effectiveness of COE screening to reduce disease-related mortality remains to be determined.[69,70]

Clinical Diagnosis and Staging

Tumors are most often classified according to the TNM, tumor-node-metastasis system updated by the American Joint Committee on Cancer in 2002, where (T) represents the primary tumor size, (N) indicates the status and extent of regional lymph node involvement, and (M) denotes the presence or absence of distant metastasis.[71,72]

TNM classification of carcinomas of the oral cavity

T – Primary tumor

TX- Primary tumour cannot be assessed

T0 - No evidence of primary tumor

Tis - Carcinoma in situ

T1- Tumour 2 cm or less in greatest dimension

T2 - Tumour more than 2 cm but not more than 4 cm in greatest dimension

T3 - Tumour more than 4 cm in greatest dimension

T4a - Tumor invades through cortical bone, into deep/extrinsic Muscle of tongue (genioglossus, hyoglossus, palatoglossus, and styloglossus), maxillary sinus, or skin of face

T4b - Tumor invades masticator space, pterygoid plates, or skull base; or encases internal carotid artery

Note: Superficial erosion alone of bone/tooth socket by gingival primary is not sufficient to classify a tumour as T4.

N – Regional lymph nodes##

NX - Regional lymph nodes cannot be assessed

N0 - No regional lymph node metastasis

N1 - Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension

N2 - Metastasis as specified in N2a, 2b, 2c below

N2a - Metastasis in a single ipsilateral lymph node, more than 3 cm but not more than 6 cm in greatest dimension

N2b - Metastasis in multiple ipsilateral lymph nodes, none more than 6 cm in greatest dimension

N2c – Metastasis in bilateral or contralateral lymph nodes, none more than 6 cm in greatest dimension

N3 - Metastasis in a lymph node more than 6 cm in greatest dimension

Note: Midline nodes are considered ipsilateral nodes.

M – Distant metastasis

MX - Distant metastasis cannot be assessed

M0 - No distant metastasis

M1 - Distant metastasis

Stage grouping

Stage 0	Tis N0 M0
Stage I	T1 N0 M0
Stage II	T2 N0 M0
Stage III	T1, T2 N1 M0
	T3 N0, N1 M0
Stage IVA	T1, T2, T3 N2 M0
	T4a N0, N1, N2 M0
Stage IVB	Any T N3 M0
	T4b Any N M0
Stage IVC	Any T Any N M1

The regional lymph nodes are the cervical nodes.

The TNM stage grouping establishes an overall clinical stage (I-IV) that is closely related to survival according to an inverse relationship where the five-year survival rate for advanced stage disease (stage III-IV) is at or below 41%, whereas in early stage disease (stage I-II) five-year survival approaches 85%.[42,72]

Five-year Survival rates of Oral Cancer according to tumor stage[71,72]

Clinical Stage	TNM Classification	Five-year survival rate	Disease Status	% Cases at Diagnosis
0	T_{is} N_0 M_0	NA	Localized *in situ*	30-40%
I	T_1 N_0 M_0	68-85%	Localized	
II	T_2 N_0 M_0	53-66%		
III	T_{1-3} N_{0-1} M_0	41%	Advanced Regional Lymph Nodes	>50%
IV A/B	Any T_4,or T_{1-3} N_{2-3} M_0	9-27%		
IV C	Any T Any N M_1		Distant Metastasis	10%

Lymph node status appears to be the most significant prognostic factor for OSCC with survival approximately cut in half when metastases are found in local or regional lymph nodes.[73] In these patients, the number of positive nodes and the presence of extracapsular spread contribute to a negative prognosis.[74] Other classic clinicopathological features including anatomical site, tumor size, grade, and maximal thickness have been shown to possess limited predictive value for the identification of patients with a high risk of disease relapse and death.[73]

DERMATOGLYPHIC STUDIES IN ORAL LEUKOPLAKIA AND ORAL CANCER:

Hakan polat et al[75] conducted a study in Istanbul university on 2004 in patients with oral cancer to evaluate the dermatoglyphic pattern. 29 patients with oral cancer and 80 healthy individuals as controls were included in the study. Qualitative analysis was done by studying the finger tip pattern like arches , loops and whorls also the palmar pattern studied in hypothenar area, thenar areas I1,I2,I3,I4. Quantitative analysis was done by

estimating the a-b ridge count, finger ridge count, total finger ridge count and atd angle.

The study results of finger print pattern distribution showed 7.2% arches, 57.2% ulnar loops, 2.4% radial loops and 33.1% whorls pattern in oral cancer patients and 3.9% arches, 56.0% ulnar loops, 4.9% radial loops and 35.3% whorls pattern in control group. The frequency of various finger print pattern is compared among the two groups and the result showed increased frequency of arches oral cancer patients. The P value was <0.05.

The percentage frequency of palmar dermatoglyphic pattern of patients with oral cancer showed less loops on thenar I_4 when compared with controls. The percentage was 24.1% in oral cancer patients and 45.6% in controls and the p value was < 0.05 which is statistically significant. All other parameters like thenar I_1, I_2, I_3 and hypothenar pattern were statistically insignificant.

There is no significant difference observed in TFRC. In oral cancer the mean ± S.D. was 117±46.45 in males and 126.95±34.68 in females. In control group the mean±S.D. was 131.47±34.15 in males and 108.53±42.79 in females. The results were statistically insignificant.

There is no significant difference observed in ab count. In oral cancer the mean ± S.D. was 69.51±14.03 in males and 66.91±.9.64 in females. In control group the mean±S.D. was 76.07±11.89 in males and 74.07±7.28 in females. The results were statistically insignificant.

There is significant difference observed in atd angle. In oral cancer the mean ± S.D. was 87.5±25.42 in males and 85.18±10.02 in females. In control group the mean±S.D. was 97.79±22.82 in males and 104.5±20.87 in females. The p value was <0.05 in males which is statistically significant and the value was < 0.01 in females which is statistically highly significant.

Venkatesh et al[5] conducted a study in KLE institute, Belgam on 2009 in patients with oral leukoplakia and oral squamous cell carcinoma. 30 patients with oral leukoplakia, 30 patients with OSCC and 30 controls with habits but no oral lesions were included in the study. Qualitative analysis was done by studying the finger tip pattern like arches, loops and whorls also the palmar pattern studied in hypothenar area, thenar areas I1,I2,I3,I4. Quantitative analysis was done by estimating the a-b ridge count, finger ridge count, total finger ridge count and atd angle.

The study results of finger print pattern distribution showed 6.3% arches, 63% loops and 30% whorls pattern in oral leukoplakia patients, 7% arches, 60.7% loops and 32.3% whorls pattern in OSCC patients and 2% arches, 30% loops and 68% whorls pattern in control group. The frequency of various finger print pattern is compared among the three groups and the result showed increased frequency of arches and loops in oral leukoplakia and OSCC patients whereas in control group there is an increased frequency of whorls. The x^2 was 109.493 and the P value was 0.000

The distribution of pattern in hypothenar area among the three groups was statistically insignificant. In oral leukoplakia, 80% in right hand

and 90% in left hand had pattern. In OSCC, 76.67% in right hand and 73.3% in left hand had pattern. In control group 80% in right hand and 83.3% in left hand had pattern. The X^2 value was 1.986 and the P value was 0.370.

The distribution of pattern in thenar area I1 among the three groups was statistically insignificant. In oral leukoplakia, 86.67% in right hand and 76.67% in left hand had pattern. In OSCC, 90% in right hand and 80% in left hand had pattern. In control group 83.3% in right hand and 73.3% in left hand had pattern. The X^2 value was 0.891 and the P value was 0.64.

The distribution of pattern in I2, I3 and I4 area showed increased freaquency of loops in control group as compared to oral leukoplakia and OSCC patients. In oral leukoplakia, 33.33 in right hand and 21.11% in left hand had pattern. In OSCC, 18.88% in right hand and 21.11% in left hand had pattern. In control group 34.44% in right hand and 35.5% in left hand had pattern. The X^2 value was 13.109 and the P value was 0.011.

There is no significant difference observed in TFRC. In oral leukoplakia the mean value was 148.1 and the standard deviation was 42.58. In OSCC the mean value was 168.13% and the standard deviation was 43.56. In control group the mean value was 168.43 and the standard deviation was 40.67. Frequency was 1.866 and the P value was 0.061.

There is no significant difference observed in ab count. In oral leukoplakia the mean value was 38.77 in right hand and 38.87 in left hand. In OSCC the mean value is 38.57 in right hand and 40.17 in left hand . In

control group the mean value in right hand was 40.67 and 41.47 in left hand. The P value was 0.339 for right hand and 0.309 for left hand.

There is no significant difference observed in atd angle. In oral leukoplakia the mean value was 40.33 in right hand and 39.93 in left hand. In OSCC the mean value is 39.93 in right hand and 38.50 in left hand. In control group the mean value in right hand was 40.93 and 39.96 in left hand. The P value was 0.609 for right hand and 0.206 for left hand.

Study Topic: "Palmar dermatoglyphics in Oral Leukoplakia and Oral squamous cell carcinoma patients"

Study Design: The present study is a Randomized Control Study.

Study Duration: This study was conducted between April 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Dr. Rai Memorial Medical and Cancer Centre, Chennai.

Study Population

A total number of 90 patients were involved in the study.

Obtaining approval from the authorities

Permission from the ethical committee of **Ragas Dental College and Hospital**, Chennai was obtained before starting the study.

Due consent to participate in the study was obtained from the Subjects in letter format both in Tamil and English.

STUDY GROUP

The study group consists of a total number of 90 patients. Out of the 90 patients, 30 were controls with tobacco smoking habit but no evident lesions,30 patients were suffering from Oral Leukoplakia and 30 patients were suffering from Oral cancer.

Group I – Control

The control group comprises of 30 healthy individuals with the habit of smoking but no evident lesion who visited the outpatient department of Oral Medicine and Radiology.

Inclusion Criteria

1. Individuals with habit of smoking tobacco of any form ; more than 10 numbers for more than 10 years.
2. Individuals with no mucosal lesions.

Exclusion Criteria

1. Individuals with the habit of chewing and with the habit of both smoking and chewing.
2. Individuals with dermatological diseases or disorders or syndromes which affects the palmar region.

Group II - Oral Leukoplakia

This study group comprised of 30 patients visited the Department of Oral Medicine and Radiology.

Inclusion Criteria

1. Patients with positive history of smoking.
2. During soft tissue examination ,subjects with well-defined white patch, localized or extensive, that is slightly elevated and that has a fissured, wrinkled or corrugated surface or a mixed red – white lesion in which keratotic white nodules or patches are distributed over an atropic erythematous background or presence of thick white lesions with papillary surfaces in the oral cavity and on palpation which reveals leathery consistency and which is in consistent with the diagnosis of leukoplakia.

Exclusion Criteria

1. Lesions belonging to other entities such as Lichen planus, lupus erythematosus, leukedema and white sponge nevus and lesions for which etiology can be established, such as frictional keratosis, cheek/lip/tongue biting, contact lesions and stomatis nicotina palatine.
2. Patients with dermatological diseases or disorders or syndromes which affects the palmar region.
3. Patients with the habit of chewing and with the habit of both smoking and chewing.

Group III Oral Cancer

This study group consists of 30 patients suffering from oral cancer diagnosed clinically. These patients were selected from the Department of Oral Medicine and Radiology and Dr.Rai Memorial Medical and Cancer center institute.

Inclusion Criteria

1. Patients with positive history of smoking.
2. During soft tissue examination, presence of a non – healing ulceroproliferative growth with pain, tenderness, limitation / loss of function, bleeding, indurated margins and presence of regional lymphadenopathy and which is in consistent with the diagnosis of oral cancer.

Exclusion Criteria

1. Oral cancer patients with the habit of chewing and with the habit of both smoking and chewing.
2. Patients with dermatological diseases or disorders or syndromes which affects the palmar region.

MATERIALS

Examination of the Patient

- Conventional Dental chair with halogen lamp
- A pair of sterile gloves and disposable mouth mask
- Stainless steel Kidney trays
- Plain mouth mirror, straight probe, tweezer
- Sterile gauze pieces and cotton
- Glass tumbler with water
- 0.2% chlorhexidine gluconate
- Sterilizer, cheatel forceps.
- Sterile plastic containers for collection of saliva.

Sample Collection

- Canon flat bed scanner- Canoscan lide 25
- Laptop for data storage.
- ScanGear starter software.

Sample Analysis

- Photoshop version 8.0

METHODOLOGY

Examination of the Subjects

The experimental subjects were made to sit comfortably on a dental chair. Sterile hand gloves were used during examination of the patients. Patients were examined under halogen lamp in the dental chair under aseptic conditions and relevant demographic data were collected. Clinical diagnosis was made and patients who showed characteristic features of Leukoplakia, Oral Cancer and control group were prepared for sample collection.

Sample Collection

Subjects were asked to wash their hands with soap water, so as to remove any oil or dirt. The glass platen of the scanner is cleaned thoroughly to remove the dust. Then the patient was asked to place the right hand on the top of the glass platen and instruction given to the patient not to move the hand or not to press the hand hardly against the glass platen. The image is previewed in the laptop screen using the scanGear starter software and then the image of the hand was scanned at 300dpi. The same procedure was repeated for the left hand and the thumb fingers then the images were stored in the laptop.

Sample Analysis

The finger and palm prints were analysed qualitatively and quantitatively using Photoshop 8.0 software. The qualitative analysis done to analyze, finger print patterns and palmar patterns. The quantitative analysis done to analyze, total finger ridge count, ab count and atd angles.

Qualitative Analysis

To analyse finger pattern frequency, the finger tip pattern configurations were classified as arches (A), Loops (L) and whorls (W).

To study palmar pattern configurations parameters chosen were patterns in Thenar / I_1, I_2, I_3 and I_4 interdigital areas and hypothenar area.

Quantitative Analysis

The counting was done along a straight line connecting the triradii point to the point of core. Ridge counts were recorded in order, beginning from first digit of right hand to the fifth digit and from first digit of left hand to fifth digit of same hand. The total finger ridge count was derived by adding the ridge counts on all ten fingers. Only the larger count was used on those digits with more than one ridge count. In a loop there is one triradius and so one ridge count; in a whorl with 2 triradii there are two counts and higher is used.

The ab ridge count was done along the straight line connecting the triradii point a and b in the palm.

The atd angle was recorded by drawing lines from the digital triradius 'a' to the axial triradius 't' and from this to the digital triradius 'd'. The angle was measured using the measuring tool in Photoshop 8.0 software.

Data management and Statistical Analysis

All the datas were entered in Microsoft excel sheets. Statistical analysis was done using SPSS software SYSTAT version 7.0.

For qualitative analysis chi square test was used to find the significance.

For quantitative analysis mean and standard deviation were estimated in the sample for each study group. Mean values were compared by using one-way ANOVA followed by multiple range tests by Tukey-HSD procedure.

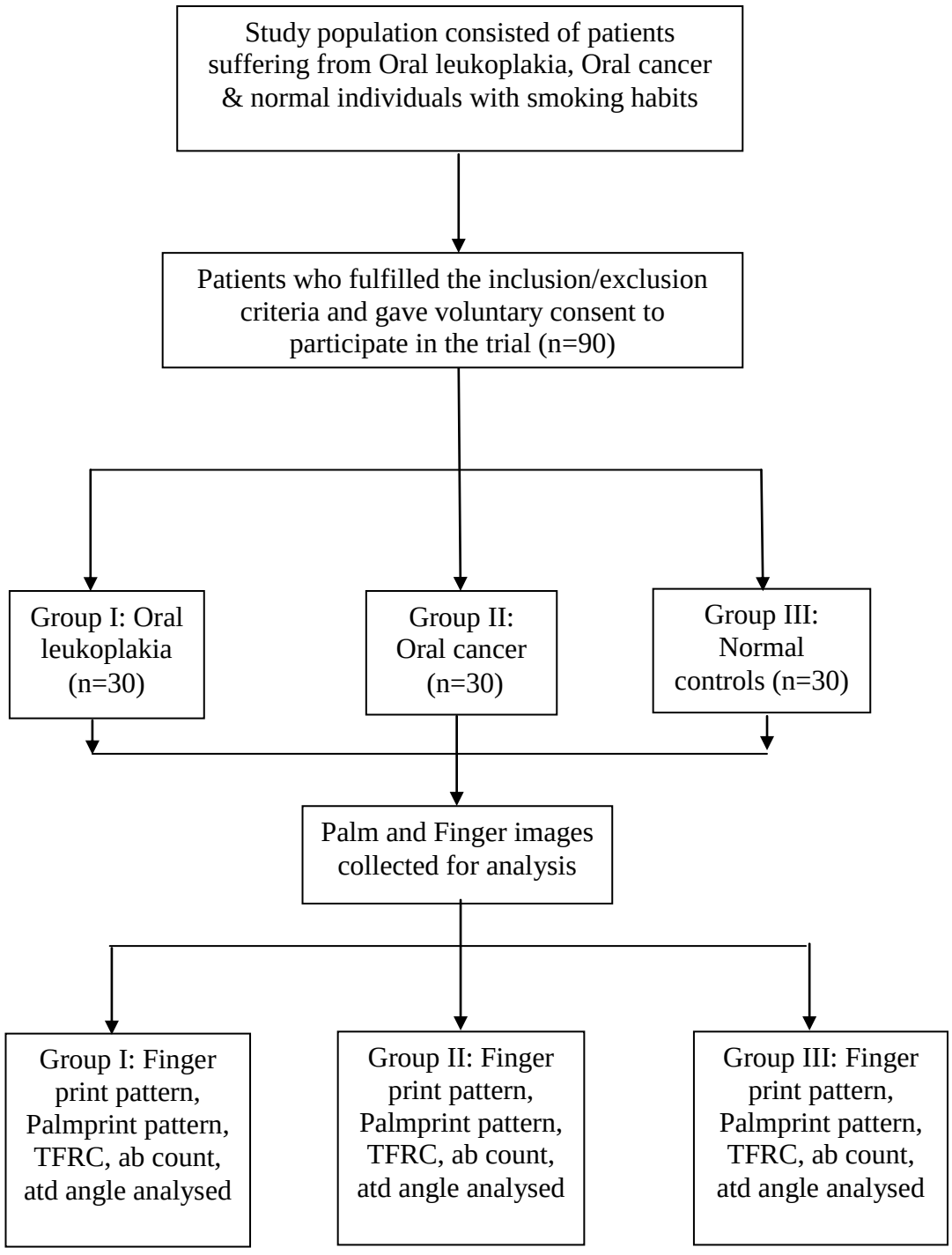
In the present study P <0.05 was considered as the level of significance.

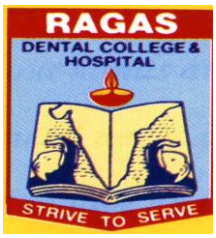
$$\text{Mean } (X) = \frac{\sum \overline{Xi}}{n}$$

$$\text{Standard Deviation} = \sqrt{\frac{\sum (Xi - X)^2}{n-1}}$$

Where Xi is the individual observation and n is the sample size.

STUDY OUTLINE





RAGAS DENTAL COLLEGE & HOSPITAL

2/102, East Coast Road, Uthandi, Chennai – 600119

DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

Date:

Serial No. Op. No.

Name: Age/ Sex:

Religion:

Occupation: Income:

Address: Phone no**:**

Study group : Group I / Group II / Group III

Smoking:

- Duration of smoking (<10 yrs ,10-20 yrs / 20-30 yrs / >30 yrs)
- Frequency of smoking per day (<5 times / 6-10times / 11-20times / >20 times)

Alcohol Consumption:

- Duration of alcohol consumption (<10 yrs ,10-20 yrs / 20-30 yrs / >30 yrs)
- Frequency of alcohol consumption per month (<5 times / 6-10times / >11times)

Leukoplakia :

Site :

Size :

Type :

Oral Squamous Cell Carcinoma :

Site :

Staging :

RIGHT HAND

FINGERS	THUMB	INDEX	MIDDLE	RING	LITTLE
DISTAL PHALANX RIDGE PATTERN					
RIDGE COUNT					
INTER DIGITAL SPACE	I1	I2	I3	I4	HYPOTHENAR
RIDGE PATTERN					

AFRC -
TFRC -
a-b count -
atd angle -

LEFT HAND

FINGERS	THUMB	INDEX	MIDDLE	RING	LITTLE
DISTAL PHALANX RIDGE PATTERN					
RIDGE COUNT					
INTER DIGITAL SPACE	I1	I2	I3	I4	HYPOTHENAR
RIDGE PATTERN					

AFRC -

TFRC -

a-b count -

atd angle -

Figure 11: Armamentarium for Clinical Examination

Figure 12: Normal Mucosa

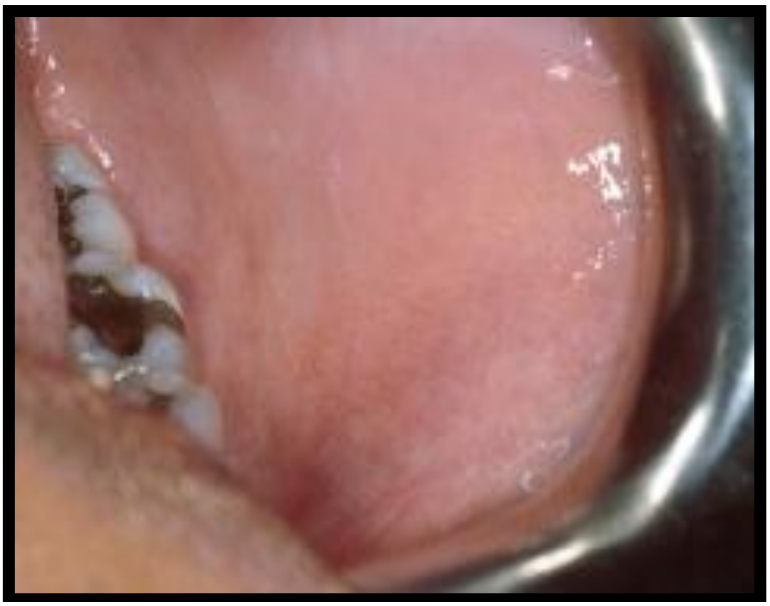


Figure 13: Clinical Lesion - Leukoplakia

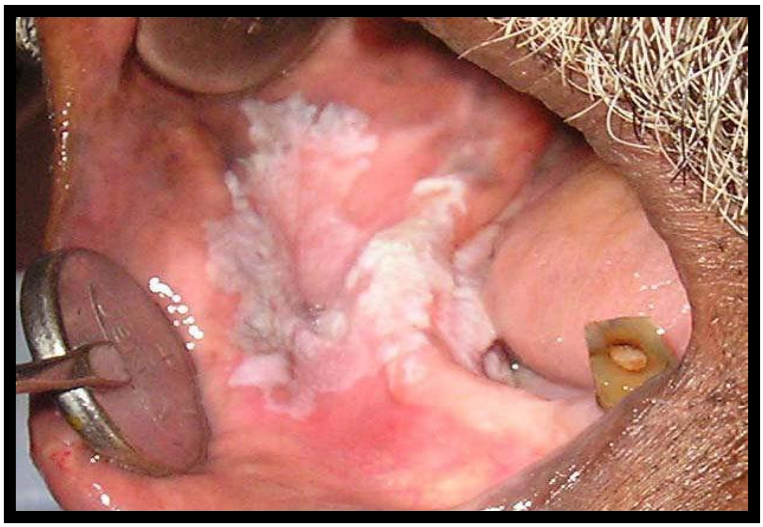


Figure 14: Clinical Lesion – Oral Cancer

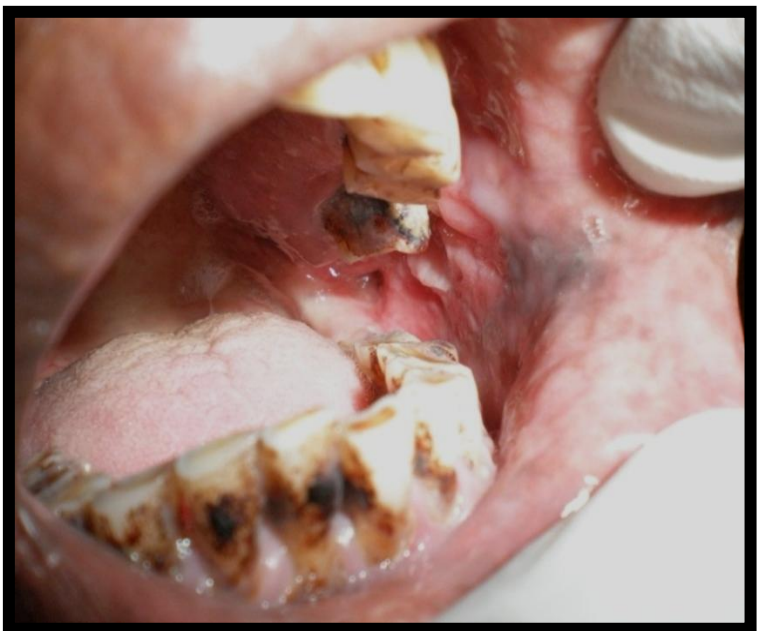


Figure 15: Clinical Lesion – Oral Cancer

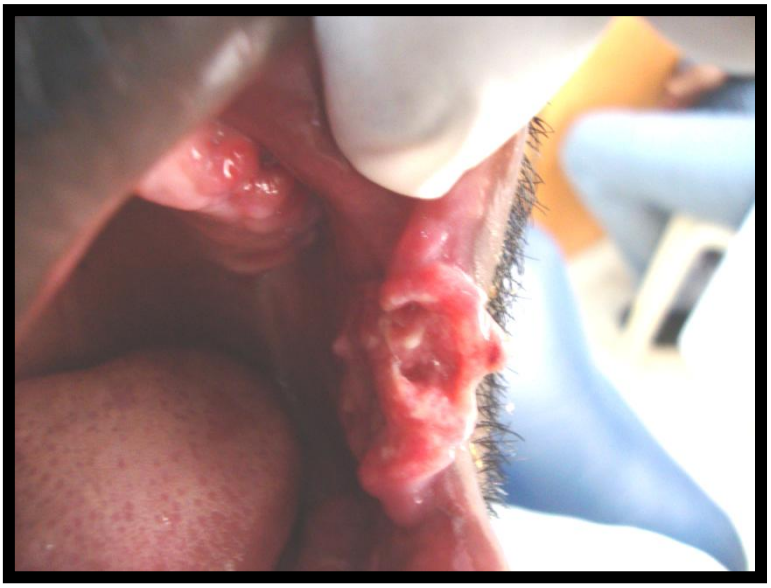


Figure 16: Armentarium used for Sample Collection

Figure 17: Procedure of Scanning Palmar Region of a Patient

Figure 18: Preview of Scanned Image

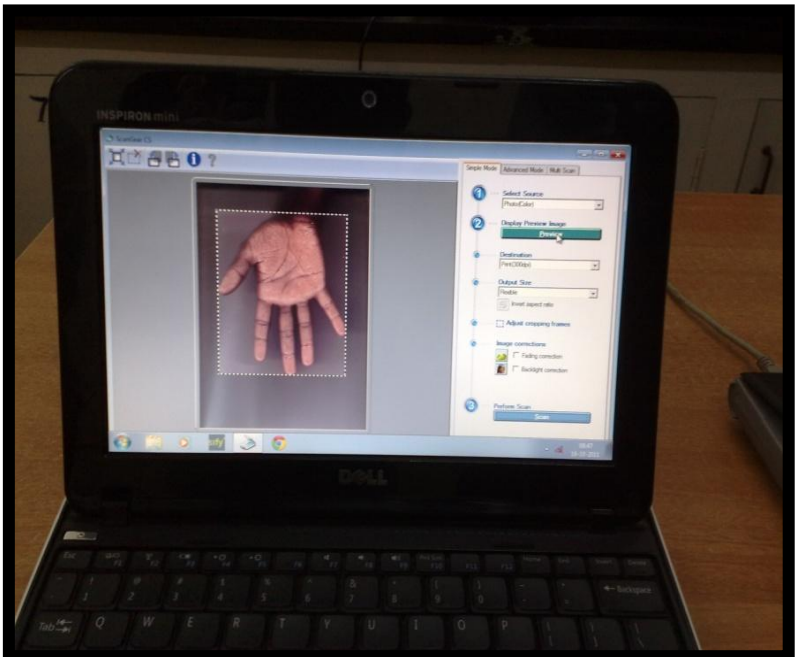


Figure 19: Image of Arches

Figure 20: Image of Loops

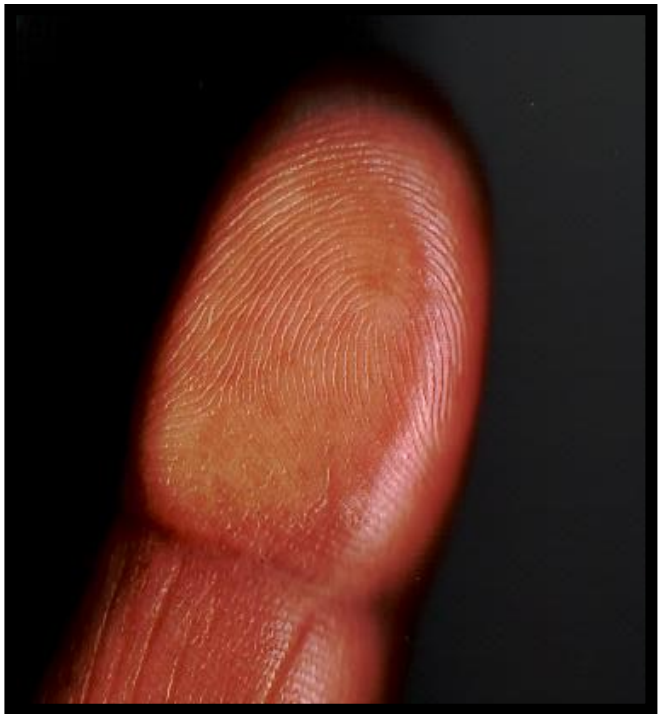


Figure 21: Image of Whorls

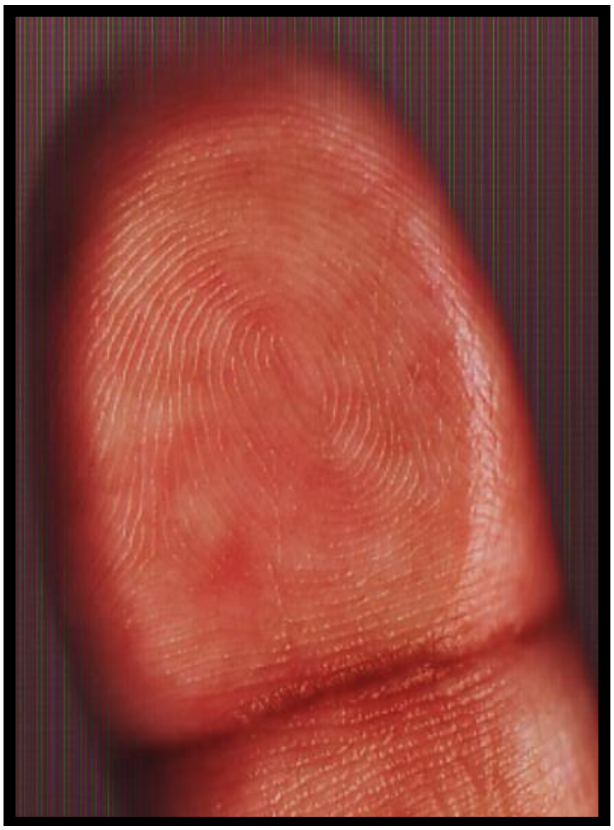


Figure 22: Finger Ridge Count Calculation

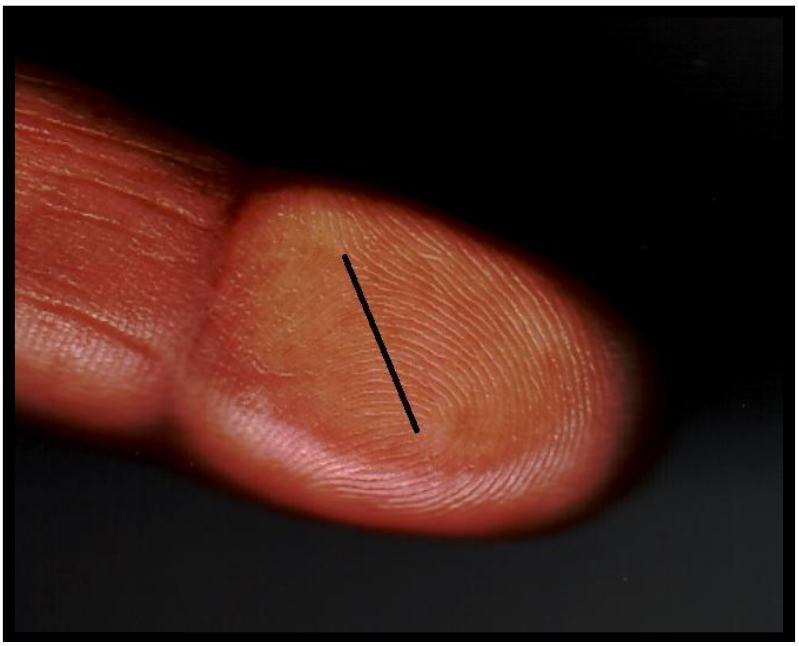


Figure 23: ab Count Calculation

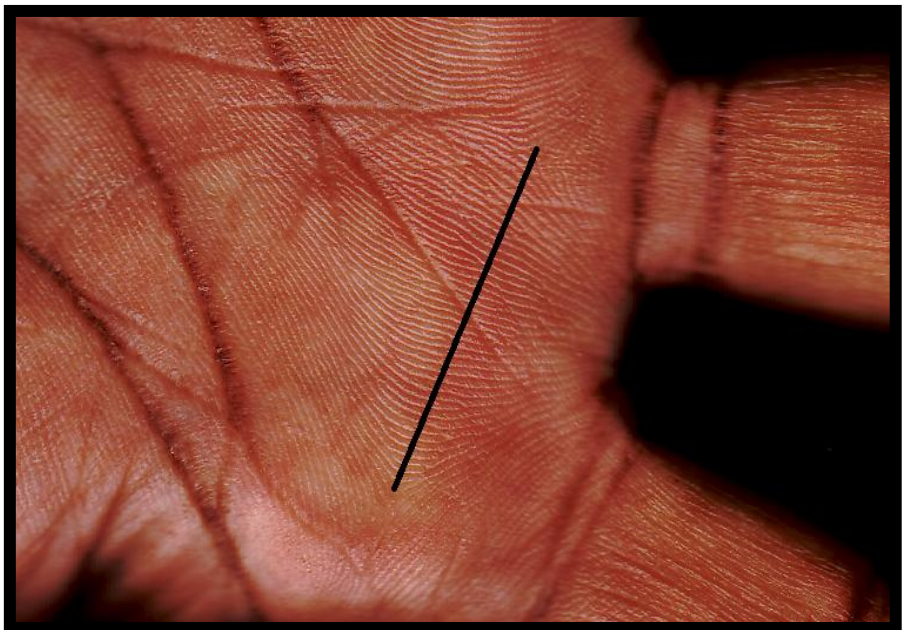
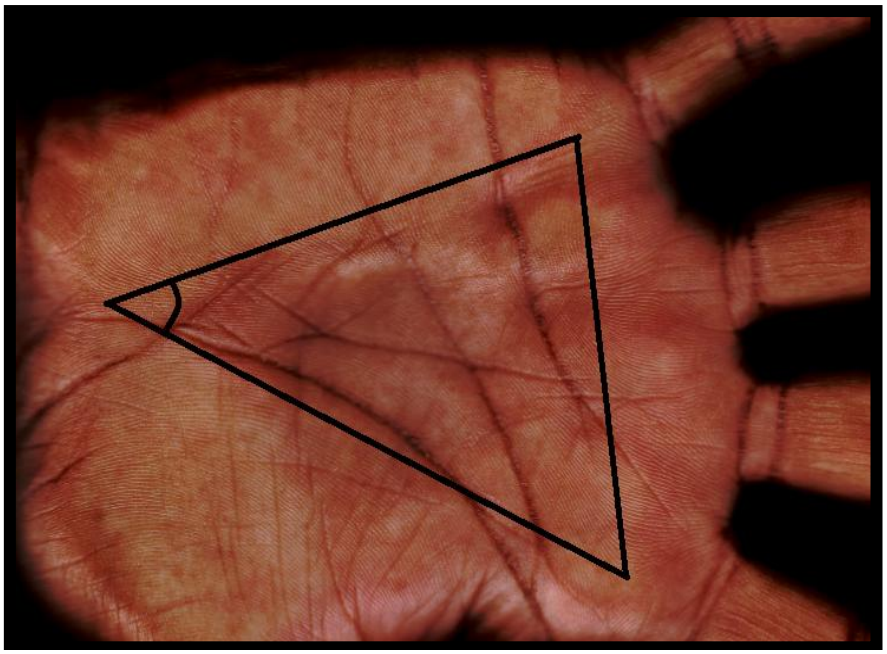


Figure 24: atd Angle Measurement



The present study is a randomized case control study which was conducted in the Department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Uthandi, Chennai. It was devised to estimate the Palmar dermatoglyphic variation in Oral leukoplakia patients, Oral squamous cell carcinoma patients and healthy controls. The study was conducted between April 2010 – May 2011 on a total of 90 subjects with 30 subjects in each group. The data obtained from the study were statistically analysed. The results extracted are compared with various variables included in the study and are presented here.

Table 1. Distribution of subjects according to Sex

The study group consisted of a total number of 90 subjects. Out of the 90 patients, 30 subjects were included in control (Group I) and among them all the 30(100%) were males and 0 (0%) were females, 30 were included in Oral leukoplakia (Group II) and among them 30 (100%) were males and 0(0%) females and 30 subjects were included in OSCC (Group III) and among them 30 (100%) were males and 0 (0%) were females. As the study groups include only the individuals with smoking habits the female percentage was 0 in all the three groups.

Table 2. Distribution of subjects according to Age

The age of the subjects included in the study ranges between 35-70 years. So the subjects were divided into four age groups which are as follows: less than 40 years, 41-50 years, 51-60 years and above 61years. Among the 30 in group I, 2(6.7%) were less than 40 years, 8(26.6%%) were

between 41-50 yrs , 14(46.6%) were between 51-60 years and 6(20.1%) were above 61 years. Among the 30 in group II, 4(13.3%) were between 41-50 years, 18(60%) were between 51-60, and 8(26.7%) were above 60 years. Among the 30 in group III, 1(3.3%) was between 41-50 years, 20(66.7%) were between 51-60 years, 9(30%) were above 60 years. The p value is ≤0.926 which is insignificant.

Table 3. Distribution of subjects based on habits

The distribution of habits was grouped as smoking only and smoking plus alcohol consumption.

In Group I, 9(30%) had the habit of only smoking and 21(70%) had the habit of smoking plus alcohol consumption. In Group II, 3(10%) had the habit of only smoking and 27(90%) had the habit of smoking plus alcohol consumption. In Group III, 2(6.6%) had the habit of only smoking and 28(93.4%) had the habit of smoking plus alcohol consumption. The p value is ≤0.026 which is significant.

Table 4. Distribution of subjects according to the duration of habits

The distribution of habits was grouped as 10-20 years, 21-30 years and more than 30 years.

In group I, 9(30%) were with the duration of habit for 10-20 years, 16(53.3%) were with the duration of habits for 21-30 years and 5(16.7%) were with the duration of habits for more than 30 years. In group II, 5(16.7%) were with the duration of habit for 10-20 years, 14(46.7%) were with the duration of habits for 21-30 years and 11(36.7%) were with the

duration of habits for more than 30 years. In group III, 2(6.7%) were with the duration of habit for 10-20 years, 14(46.7%) were with the duration of habits for 21-30 years and 14(46.7%) were with the duration of habits for more than 30 years. The p value is ≤0.061 which is insignificant.

Table 5. Frequency of finger print pattern in group I (Control)

This table shows the finger print pattern in control group, 7(2.3%) have arches, 82(27.3%) have loops and 211(70.4%) have whorls.

Table 6. Frequency of finger print pattern in group II (Oral Leukoplakia)

This table shows the finger print pattern in group II patients, 18(6.0%) have arches, 176(58.6%) have loops and 106(35.4%) have whorls.

Table 7. Frequency of finger print pattern in group III (Oral OSCC)

This table shows the finger print pattern in group III patients, 17(5.6%) have arches, 185(61.7%) have loops and 98(32.7%) have whorls.

Table 8. Frequency of finger print pattern in all three groups

This table compares the frequency of finger print in all the three groups. When arches were compares between the three groups, group I had less frequency of arches (2.3%) when compared to group II (6.0%) and group III (5.6%). When loops were compared group I had less frequency of loops(27.3%) when compared to group II(58.6%) and group III(61.7%). When whorls were compared group I had increased frequency of whorls(27.3%) when compared to group II(58.6%) and group III(61.7%). The p value was ≤0.001 which is highly significant.

Table 9. Frequency of Hypothenar pattern in all three groups

This table comparers the hypothenar pattern in all the three groups. When the pattern in the right hand was compared 24(80%) in group I, 23(76.6%) in group II and 22(73.3%) in group II and had pattern. When pattern in the left hand was compared 23 (76.6%) in group I 25(83.3%) in group II and 25(83.3%) in group III and had pattern. The p value is ≤0.912 which is insignificant.

Table 10. Frequency of Thenar I_1 pattern in all three groups

This table comparers the thenar I_1 pattern in all the three groups. When the pattern in the right hand was compared 25(83.3%) in group I, 23(76.6%) in group II and 25(83.3%) in group III had pattern. When pattern in the left hand was compared 26(86.6%) in group I, 27(90%) in group II and 26 (86.6%) in group III had pattern. The p value is ≤0.993 which is insignificant.

Table 11. Frequency of Thenar I_2 pattern in all three groups

This table comparers the thenar I_2 pattern in all the three groups. When the pattern in the right hand was compared 13(43.3%) in group I, 14(46.6%) in group II and 13(43.3%) in group III had pattern. When pattern in the left hand was compared 10(33.3%) in group I, 11(36.6%) in group II and 10(36.6%) in group III had pattern. The p value is ≤0.985 which is insignificant.

Table 12. Frequency of Thenar I_3 pattern in all three groups

This table comparers the thenar I_3 pattern in all the three groups. When the pattern in the right hand was compared 11(36.6%) in group I, 8(26.6%) in group II and 8(26.6%) in group III had pattern. When pattern in the left hand was compared 10(33.3%) in group I, 9(30%) in group II and 7(23.3%) in group III had pattern. The p value is ≤0.926 which is insignificant.

Table 13. Frequency of Thenar I_4 pattern in all three groups

This table comparers the thenar I_4 pattern in all the three groups. When the pattern in the right hand was compared 9(30%) in group I, 13(43.3%) in group II and 14(46.6%) in group III had pattern. When pattern in the left hand was compared 13(43.3%) in group I, 13(43.3%) in group II and 13 (43.3%) in group III had pattern. The p value is ≤0.724 which is insignificant.

Table 14. Comparison of total finger ridge count in all the three groups

This table compares the TFRC in all the three groups. In group I the mean value was 168.7 and standard deviation was 35.36. In group II the mean value was 158.87 and standard deviation was 39.18. In group III the mean value was 165.7 and standard deviation was 37.95. The p value is ≤0.457 which is insignificant.

Table 15. Comparison of ab count of right hand in all the three groups

This table compares the ab count of right hand in all the three groups. In group I the mean value was 39.27 and standard deviation was

6.198. In group II the mean value was 39.10 and standard deviation was 5.195. In group III the mean value was 37.43 and standard deviation was 5.811. The p value is ≤0.397 which is insignificant.

Table 16. Comparison of ab count of left hand in all the three groups

This table compares the ab count of left hand in all the three groups. In group I the mean value was 40.37 and standard deviation was 6.76. In group II the mean value was 39.67 and standard deviation was 4.97. In group III the mean value was 37.47 and standard deviation was 5.12. The p value is ≤0.121 which is insignificant.

Table 17. Comparison of atd angle of right hand in all the three groups

This table compares the atd angle of right hand in all the three groups. In group I the mean value was 40.53 and standard deviation was 3.026. In group II the mean value was 35.73 and standard deviation was 4.093. In group III the mean value was 34.53 and standard deviation was 2.063. The p value is ≤0.001 which is highly significant.

Table 18. Multiple Comparison of atd angle of right hand in all the three groups

This table compares the atd angle of right hand in all the three group. The p value between group I and group II is ≤0.001 which is highly significant. The p value between group I and group III is ≤0.001 which is highly significant. The p value between group II and group III is ≤0.312 which is insignificant.

Table 19. Comparison of atd angle of left hand in all the three groups

This table compares the atd angle of left hand in all the three groups. In group I the mean value was 41.03 and standard deviation was 3.079. In group II the mean value was 36.57 and standard deviation was 3.971. In group III the mean value was 34.40 and standard deviation was 2.111. The p value is ≤0.001 which is highly significant.

Table 20. Multiple Comparison of atd angle of right hand in all the three groups

This table compares the atd angle of left hand in all the three group. The p value between group I and group II is ≤0.001 which is highly significant. The p value between group I and group III is ≤0.001 which is highly significant. The p value between group II and group III is ≤0.025 which is significant.

Table 1. Distribution of subject according to Sex

Sex	Group I (Control)		Group II (Oral Leukoplakia)		Group III (OSCC)		Total	
Male	30	100%	30	100%	30	100%	90	100%
Female	0	0%	0	0%	0	0%	0	0%
Total	30	100%	30	100%	30	100%	90	100%

Table 2. Distribution of Subjects according to Age

Sex	Group I (Control)		Group II (Oral Leukoplakia)		Group III (OSCC)		Total	
<40	2	6.7%	0	0%	0	0%	2	2.2%
41-50	8	26.6%	4	13.3%	1	3.3%	13	14.3%
51-60	14	46.6%	18	60.0%	20	66.7%	52	57.8%
>61	6	20.1%	8	26.7%	9	30%	23	25.7%
Total	30	100%	30	100%	30	100%	90	100%

P value ≤0.018

Table 3. Distribution of Subjects according to Habits

Habits / Group	Smoking Only		Smoking + Alcohol		Total	
Group I Control	9	30%	21	70%	30	10%
Group II (Oral Leukoplakia)	3	10%	27	90%	30	10%
Group III (OSCC)	2	6.6%	28	93.4%	30	10%
Total	14	13.6%	76	84.4%	90	100%

P value ≤0.026

Table 4. Distribution of Subjects according to duration of Habits

Sex	Group I (Control)		Group II (Oral Leukoplakia)		Group III (OSCC)		Total	
10-20	9	30%	5	16.7%	2	6.7%	16	17.8%
21-30	16	53.3%	14	46.7%	14	46.7%	44	48.9%
>31	5	16.1%	11	36.7%	14	46.7%	30	33.3%
Total	30	100%	30	100%	30	100%	90	100%

P value ≤0.061

Table 5. Frequency of finger print pattern in Group I (Controls)

Pattern	Group I (Control)	Percentage
Arches	7	2.3%
Loops	82	27.3%
Whorls	211	70.4%
Total	300	100%

Table 6. Frequency of finger print pattern in Group II (Oral Leukoplakia)

Pattern	Group II (Oral Leukoplakia)	Percentage
Arches	18	6.0%
Loops	176	58.6%
Whorls	106	35.4%
Total	300	100%

Table 7. Frequency of finger print pattern in Group III (OSCC)

Pattern	Group III (OSCC)	Percentage
Arches	17	5.6%
Loops	185	61.7%
Whorls	98	32.7%
Total	300	100%

Table 8. Frequency of finger print pattern in all three study Groups

Sex	Group I (Control)		Group II (Oral Leukoplakia)		Group III (OSCC)		X^2	P
Arches	7	2.3%	18	6%	17	5.6%	110.226	≤0.001
Loops	82	27.3%	176	58.6%	185	61.7%		
Whorls	211	70.4%	106	35.4%	98	32.7%		
Total	300	100%	300	100%	300	100%		

Table 9. Frequency of Hypothenar Pattern in all three Groups

Hypothenar Pattern	**Group I (Control)**		**Group II (Oral Leukoplakia)**		**Group III (OSCC)**		X^2	**P**
Right	24	80%	23	76.6%	22	73.3%	0.184	≤0.912
Left	23	76.6%	25	83.3%	25	83.3%		

Table 10. Frequency of Thenar I_1 Pattern in all three Groups

Thenar Pattern	**Group I (Control)**		**Group II (Oral Leukoplakia)**		**Group III (OSCC)**		X^2	**P**
Right	25	83.3%	23	76.6%	25	83.3%	0.014	≤0.993
Left	27	90%	25	83.3%	26	86.6%		

Table 11. Frequency of Thenar I_2 Pattern in all three Groups

Thenar Pattern	**Group I (Control)**		**Group II (Oral Leukoplakia)**		**Group III (OSCC)**		X^2	**P**
Right	13	43.3%	14	46.6%	13	43.3%	0.029	≤0.985
Left	10	33.3%	11	36.6%	11	36.6%		

Table 12. Frequency of Thenar I_3 Pattern in all three Groups

Thenar Pattern	**Group I (Control)**		**Group II (Oral Leukoplakia)**		**Group III (OSCC)**		X^2	**P**
Right	11	36.6%	8	26.6%	8	26.6%	0.154	≤0.926
Left	10	33.3%	9	30%	7	23.3%		

Table 13. Frequency of Thenar I_4 Pattern in all three Groups

Thenar Pattern	**Group I (Control)**		**Group II (Oral Leukoplakia)**		**Group III (OSCC)**		X^2	**P**
Right	9	30%	13	43.3%	14	46.6%	0.645	≤0.724
Left	13	43.3%	13	43.3%	13	43.3%		

Table 14. Comparison of total finger ridge court in all three Groups

TFRC	Mean	S.D	F	P
Group I (Oral Leukoplakia)	168.7	35.36	0.789	≤0.457
Group II (OSCC)	158.87	39.18		
Group III (Control)	165.27	37.95		

Table 15. Comparison of ab court of right hand in all three Groups

ab court Right	Mean	S.D	F	P
Group I (Oral Leukoplakia)	39.27	6.198	0.933	≤0.397
Group II (OSCC)	39.10	5.195		
Group III (Control)	37.43	5.811		

Table 16. Comparison of ab court of left hand in all three Groups

ab court left	Mean	S.D	F	P
Group I (Oral Leukoplakia)	40.37	6.67	2.162	≤0.121
Group II (OSCC)	39.67	4.97		
Group III (Control)	37.47	5.12		

Table 17. Comparison of atd angle of right hand in all three Groups

atd angle right	Mean	S.D	F	P
Group I	40.53	3.026	30.074	≤0.001
Group II	35.73	4.093		
Group III	34.53	2.063		

Table 18. Multiple Comparison of atd angel of right hand in all three Groups

		Sig
Group I (Control)	**Group II (Oral Leukoplakia)**	≤0.001
	Group III (OSCC)	≤0.001
Group II (Oral Leukoplakia)	**Group I (Control)**	≤0.001
	Group III (OSCC)	≤0.312
Group III (OSCC)	**Group I (Control)**	≤0.001
	Group II (Oral Leukoplakia)	≤0.312

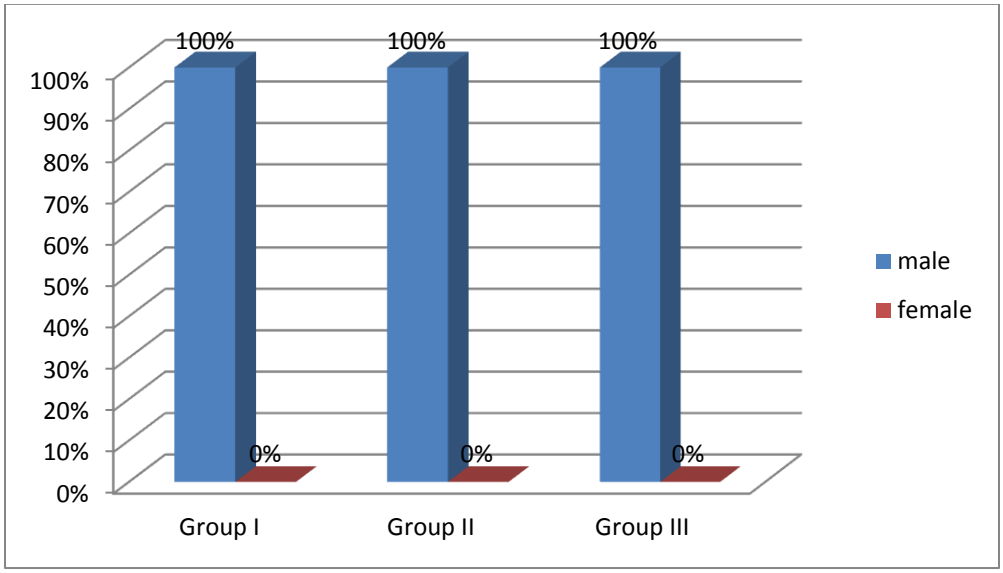
Table 19. Comparison of atd angle of left hand in all three Groups

atd angle left	Mean	S.D	F	P
Group I	41.03	3.079	34.661	≤0.001
Group II	36.57	3.971		
Group III	34.40	2.111		

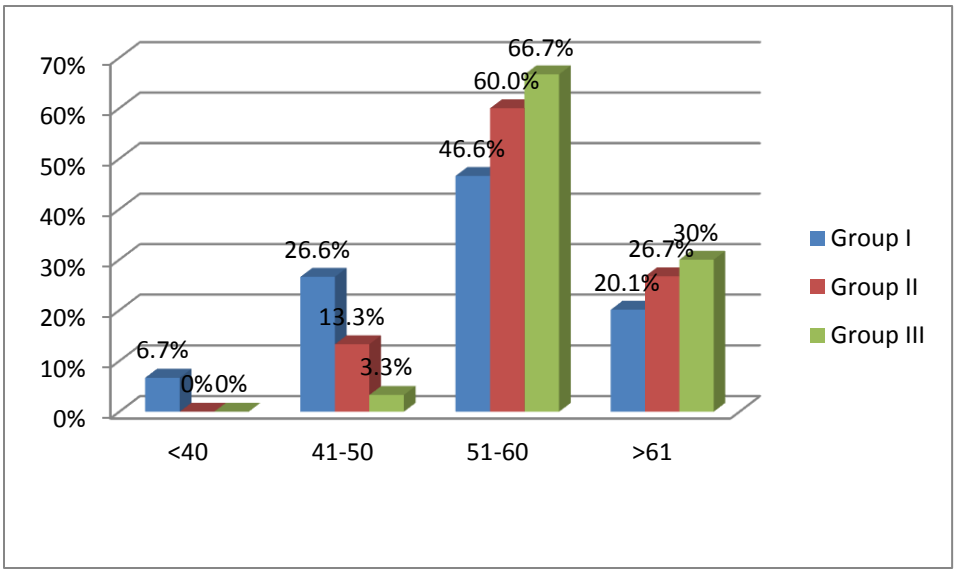
Table 20. Multiple Comparison of atd angle of left hand in all three Groups

		Sig
Group I (Control)	**Group II (Oral Leukoplakia)**	≤0.001
	Group III (OSCC)	≤0.001
Group II (Oral Leukoplakia)	**Group I (Control)**	≤0.001
	Group III (OSCC)	≤0.025
Group III (OSCC)	**Group I (Control)**	≤0.001
	Group II (Oral Leukoplakia)	≤0.025

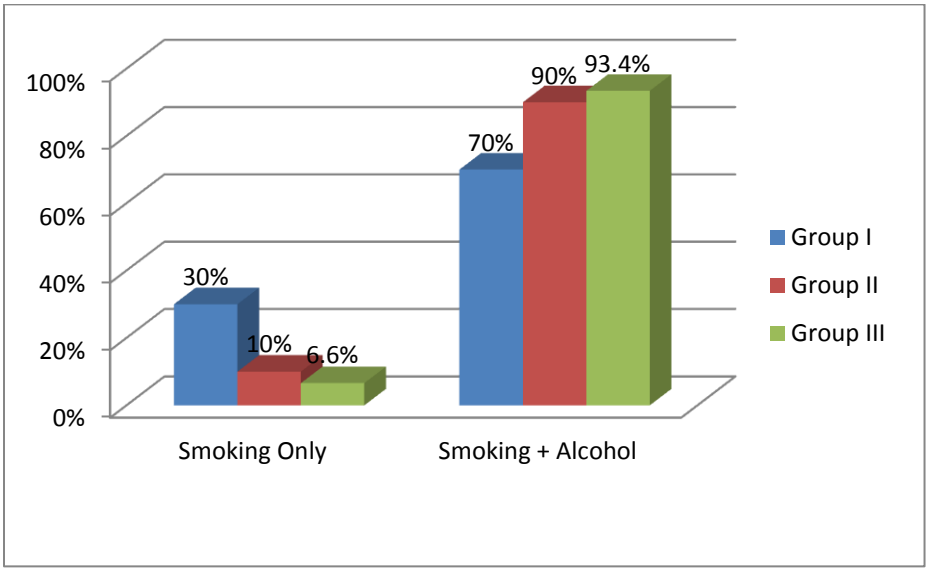
Graph 1. Distribution of subject according to Sex



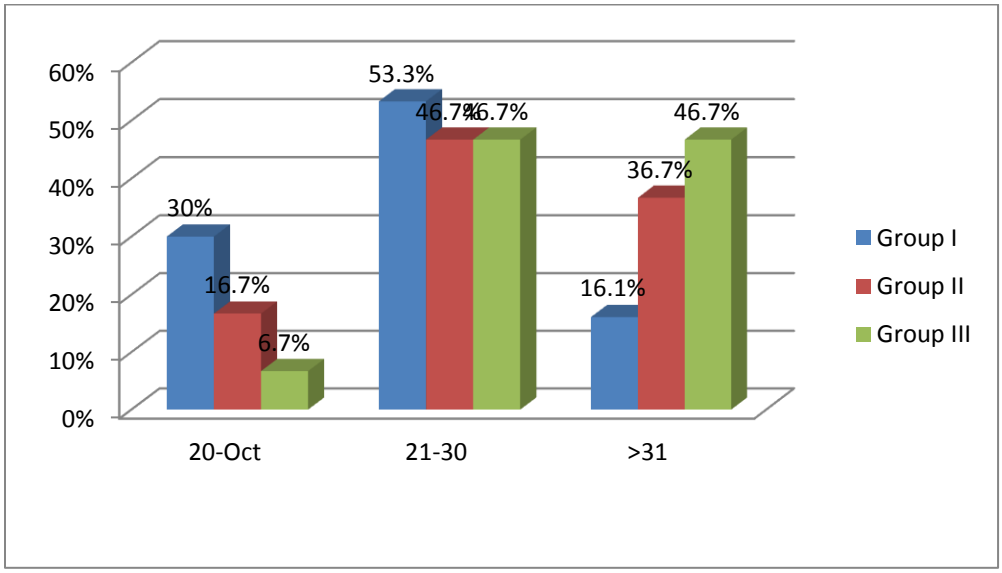
Graph 2. Distribution of Subjects according to Age



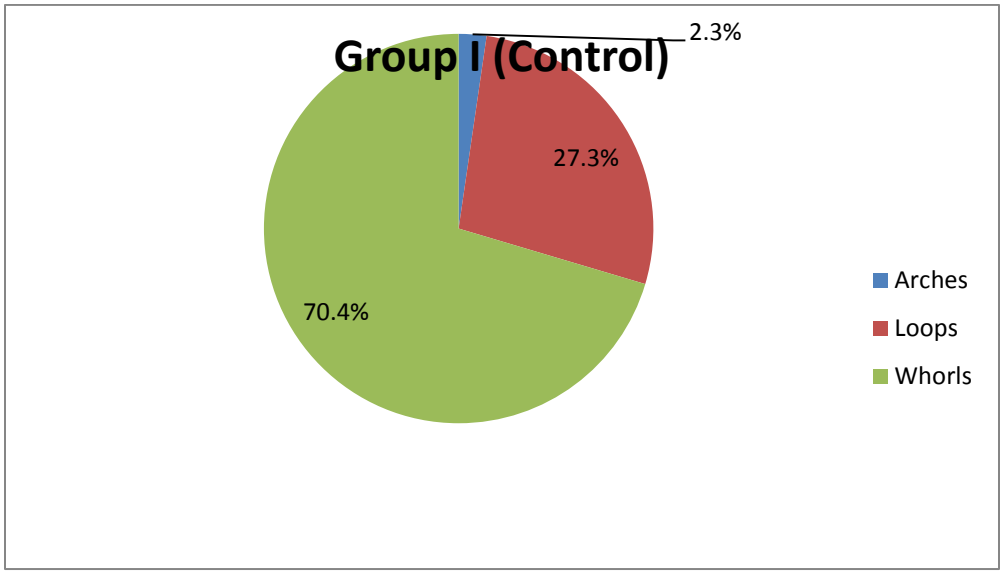
Graph 3. Distribution of Subjects according to Habits



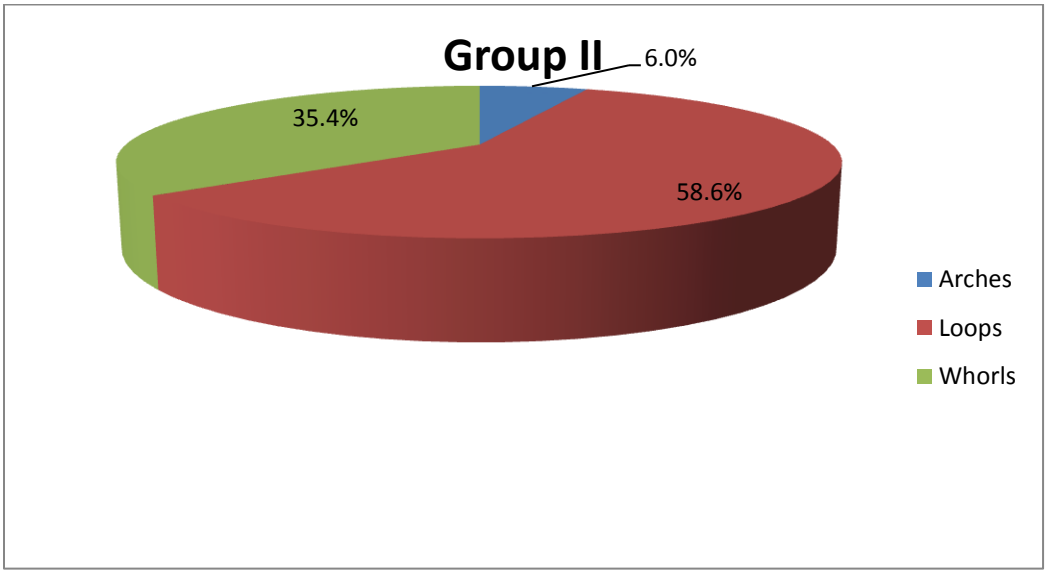
Graph 4. Distribution of Subjects according to duration of Habits



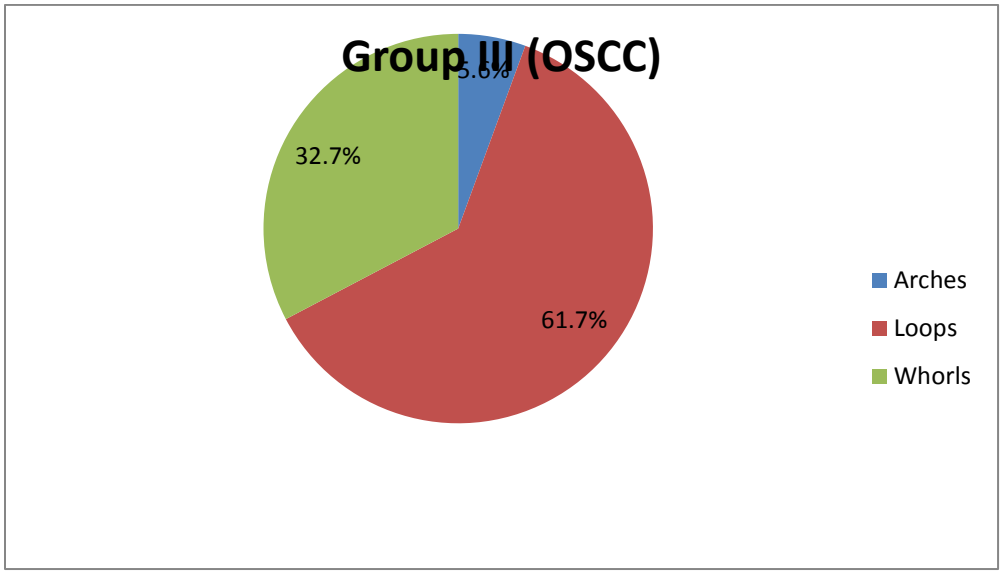
Graph 5. Frequency of finger print pattern in Group I (Controls)



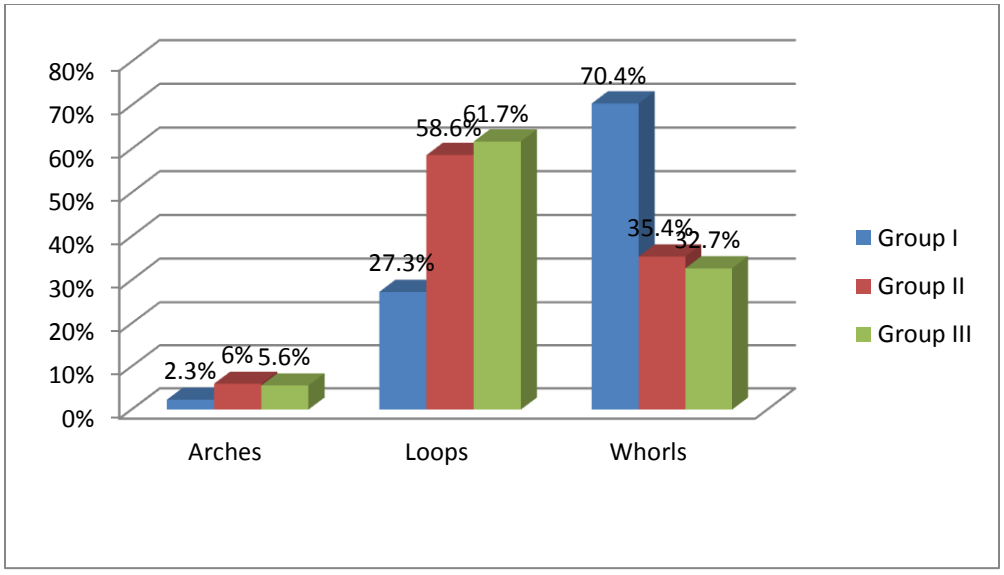
Graph 6. Frequency of finger print pattern in Group II (Oral Leukoplakia)



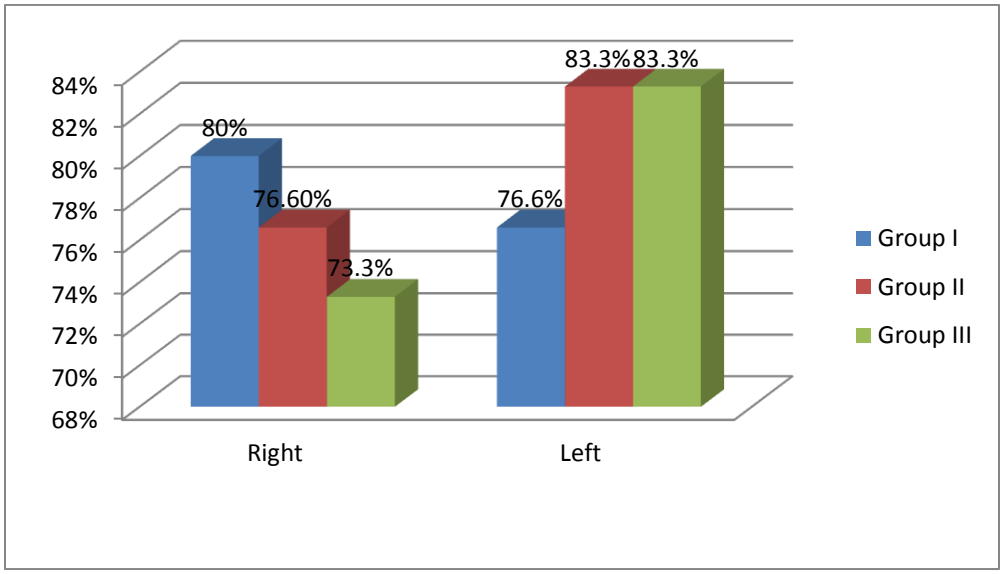
Graph 7. Frequency of finger print pattern in Group III (OSCC)



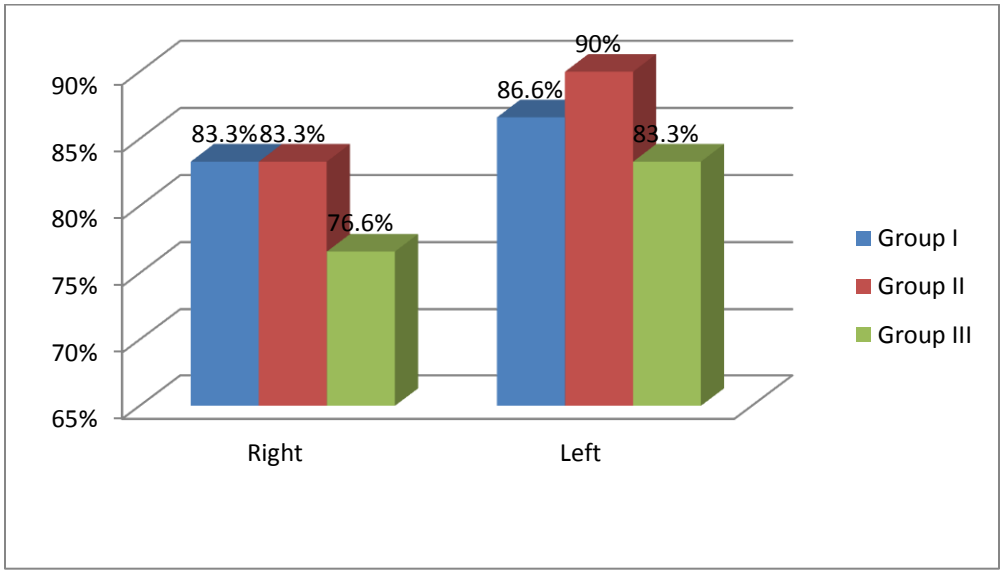
Graph 8. Frequency of finger print pattern in all three study Groups



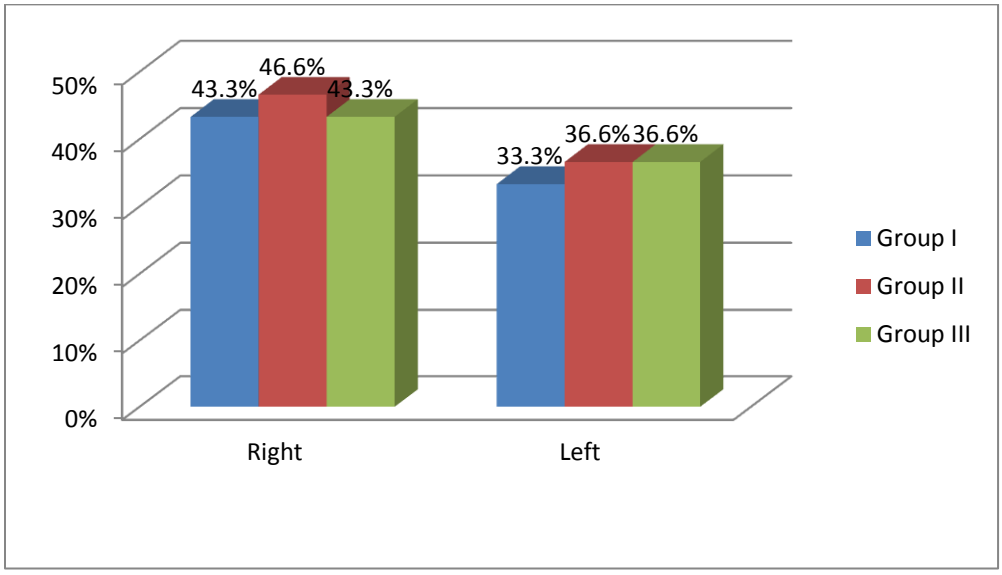
Graph 9. Frequency of Hypothenar Pattern in all three Groups



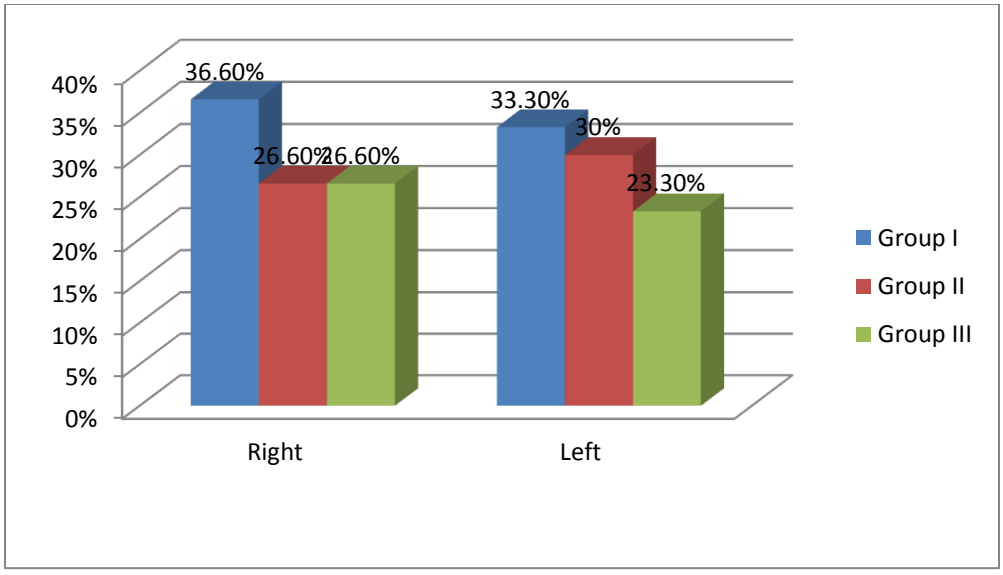
Graph 10. Frequency of Thenar I_1 Pattern in all three Groups



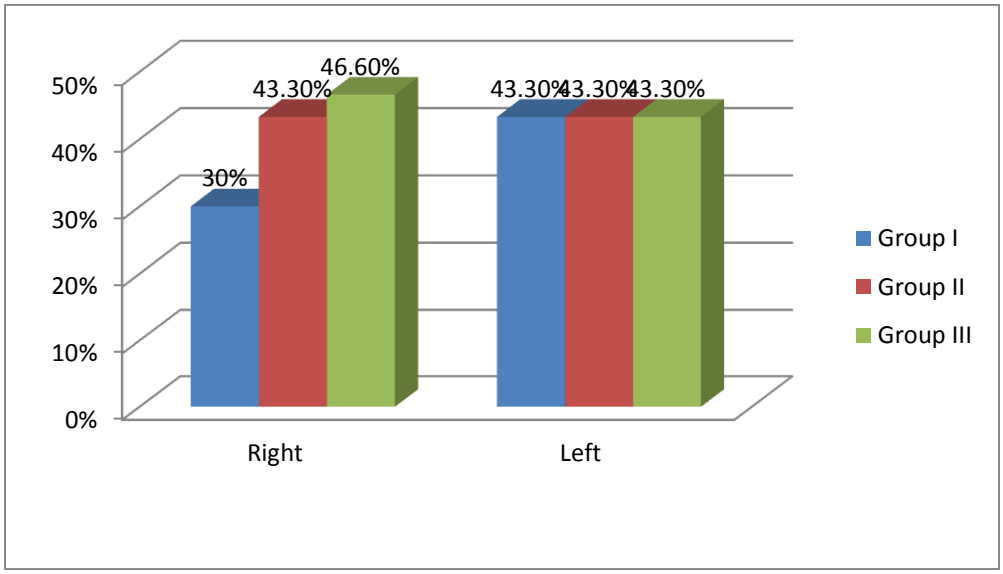
Graph 11. Frequency of Thenar I_2 Pattern in all three Groups



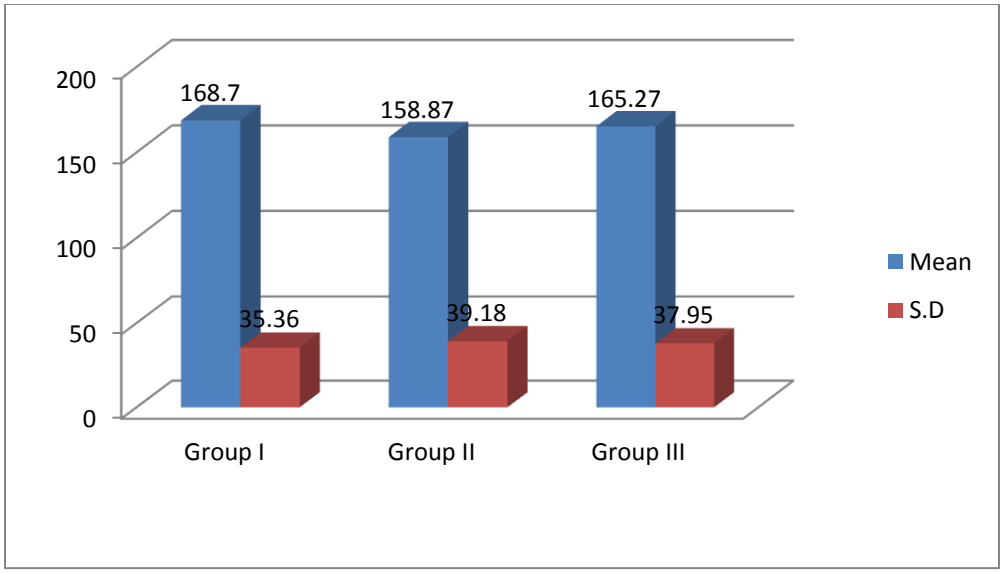
Graph 12. Frequency of Thenar I_3 Pattern in all three Groups



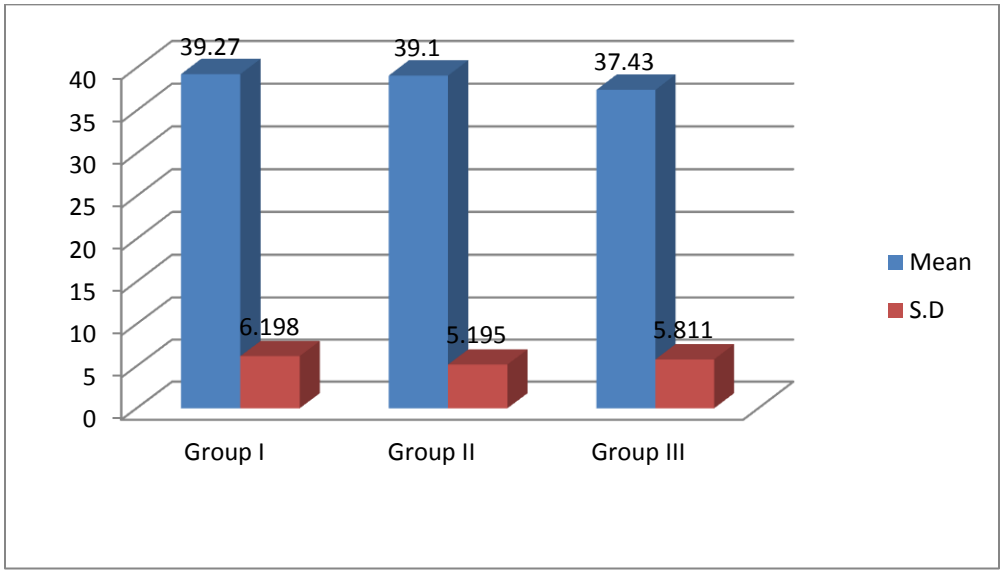
Graph 13. Frequency of Thenar I_4 Pattern in all three Groups



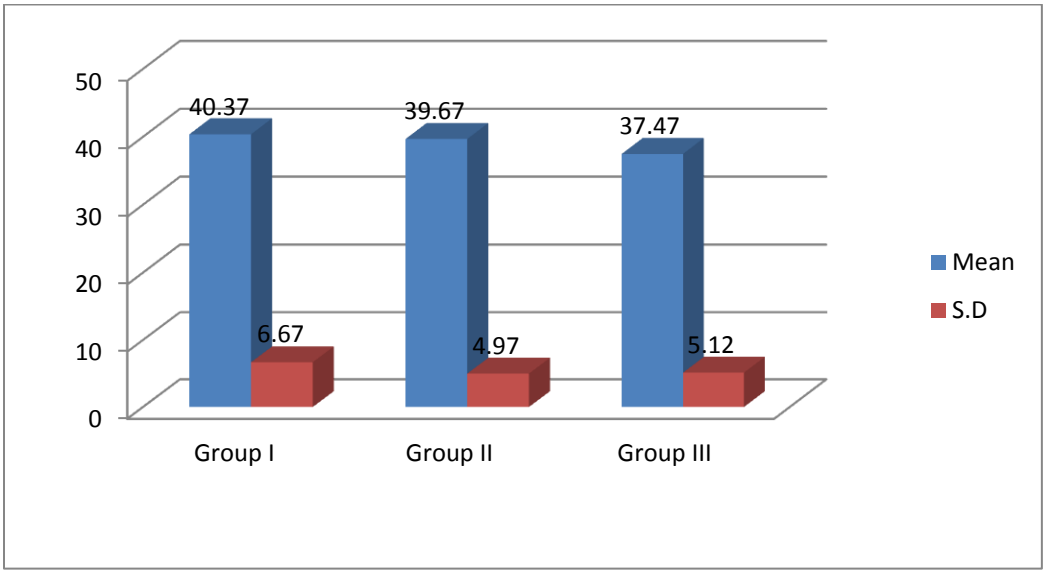
Graph 14. Comparison of total finger ridge court in all three Groups



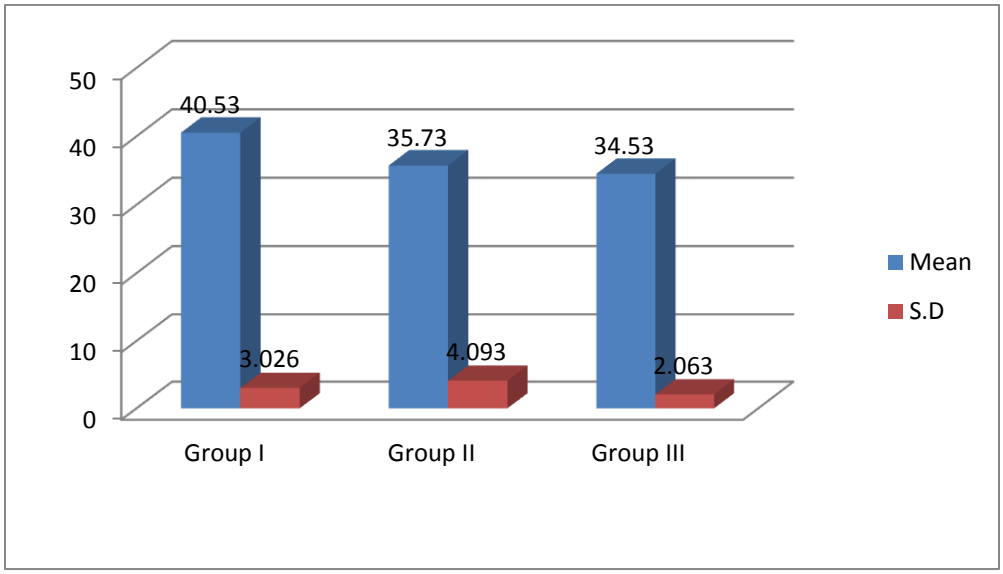
Graph 15. Comparison of ab court of right hand in all three Groups



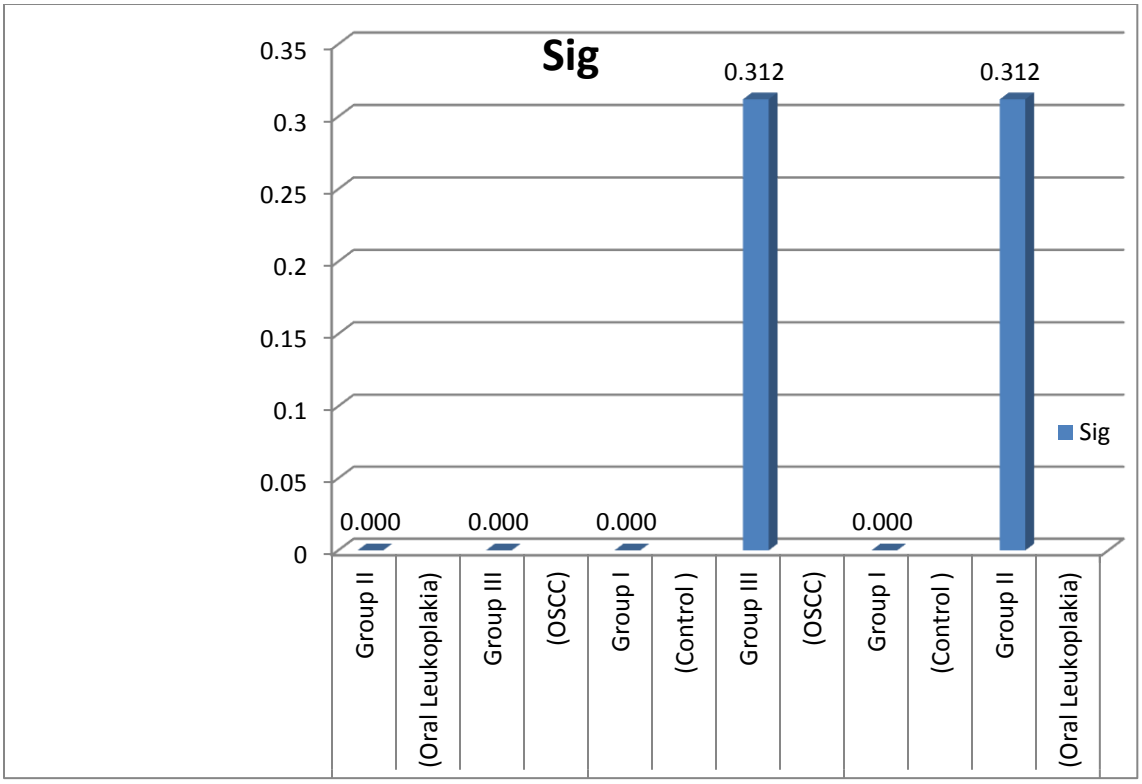
Graph 16. Comparison of ab court of left hand in all three Groups



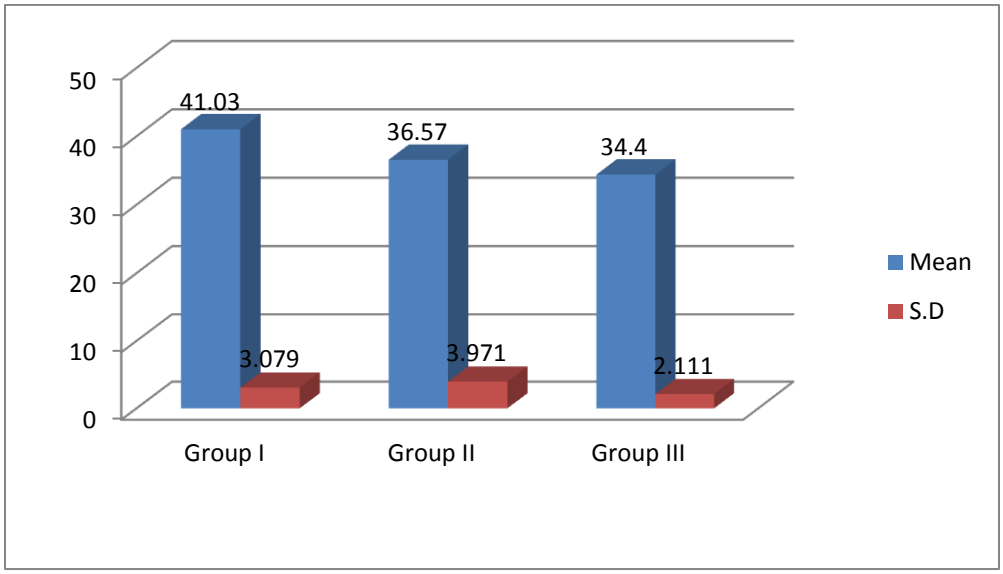
Graph 17. Comparison of atd angle of right hand in all three Groups



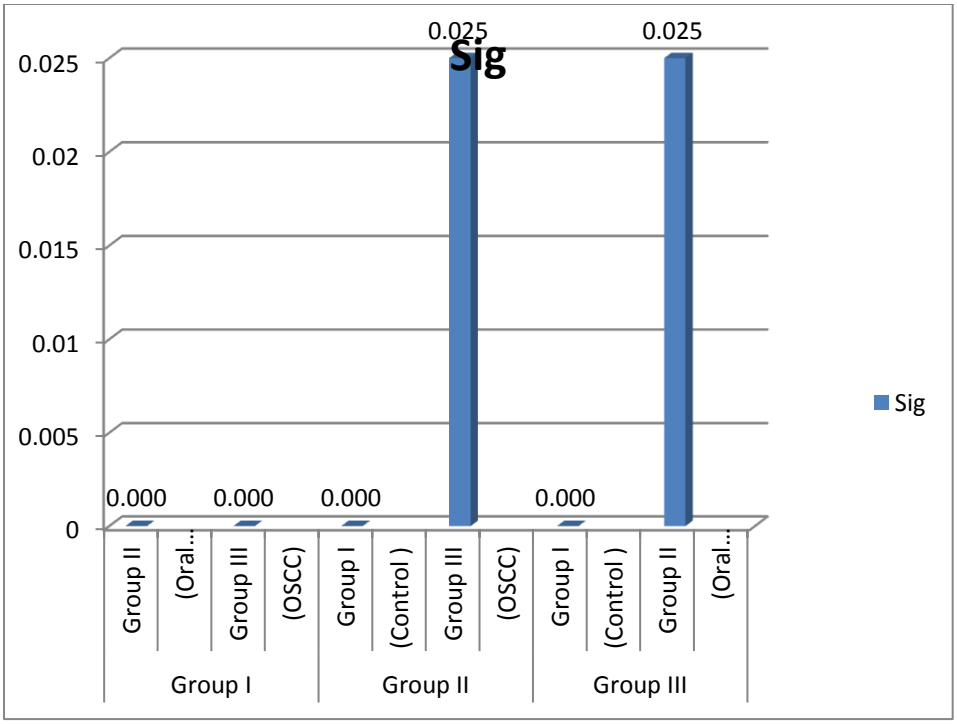
Graph 18. Multiple Comparison of atd angel of right hand in all three Groups



Graph 19. Comparison of atd angle of left hand in all three Groups



Graph 20. Multiple Comparison of atd angle of left hand in all three Groups



Dermatoglyphics is the study of pattern traceries of fine ridges on fingers, palm and sole have been a useful tool for personal identification and determination of paternity for quite some time. It proved important due to the fact that (1) unlike most human traits; dermal ridges and the configurations formed by them are not affected by age. (2) Detailed structure of individual ridges is extremely variable and (3) throughout postnatal life they are not affected by environment.[12]

In the recent decades, a considerable improvement has been achieved in the concept of relation between the types of pattern of lines in the fingers and some individual disorders. The pattern of lines in the finger of hand as a method of diagnosis has been documented in medicine. Cummins and Tompson presented dermatoglyphic pattern in patients with Downs' syndrome when its cause was unknown. Based on the dermatoglyphic pattern of cases, they found that the genetic factors are the main cause of this disease. [17]

Abnormal Dermatoglyphic patterns have been observed in several non chromosomal genetic disorders and other diseases whose etiology may be influenced directly or indirectly, by genetic inheritance. A significant link has been established by pioneer workers between ridge pattern in congenital heart diseases, Diabetes, Lung Tuberculosis, Leprosy, Epilepsy and Bronchial Asthma.[12]

Atasu M and Telatar H[4] on 1968 studied the dermatoglyphic pattern on different cancers in 201 turkish patients. Results showed increase in

whorls and decrease in loops. During 1973 Fuller IC conducted study with different cancers and found decrease in ridge counts compared to that of controls. It is suggested that many genes which take part in the control of finger print and palm dermatoglyphic development distinguished cancer patients from the general population. It is possible that these genes also predispose to the development of malignancy.

Tobacco exposure and alcohol exposure are the major determinants of head and neck squamous cell carcinoma (HNSCC). Since only a fraction of exposed individuals develops cancer, however, an intrinsic susceptibility to environmental genotoxic exposures has also been suggested as playing a role in carcinogenesis. Within the general population, there may exist varying degrees of DNA maintenance capability.[76]

This study deals with the evaluation of difference in Palmar dermatoglyphics of Oral Leukoplakia, Oral Squamous cell carcinoma and control group. This study was conducted between April 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Dr. Rai Memorial Medical and Cancer Centre, Chennai.

A case control study was conducted in which 90 subjects were selected. The study subjects were categorized into three groups: Group I consist of 30 healthy individual with the habit of smoking but no evident lesions; Group II, 30 patients suffering from Oral leukoplakia; Group III, 30 patients suffering from oral cancer.

Patients only with the habit of smoking were included in the study, in control group smoking tobacco of any form ; more than 10 numbers for more than 10 years were included in the study. Individuals with the habit of chewing and with the habit of both smoking and chewing and individuals with dermatological diseases or disorders or syndromes which affects the palmar region were excluded from the study.

In the present study all the 90 individuals are males (100%). As per the inclusion criteria only persons with smoking habits were included in the study. Hence the study result shows that the females with the habit of smoking are nil. The survey report submitted by international institute of population sciences, 2000 shows that prevalence of smoking above the age group of 30 in India was reported to be 41.2% in males and 3.9% in females.[48] In the present study the percentage of females is nil which may be because of the low sample size.

The age of the subjects included in the study ranges between 35-70 years. So the subjects were divided into four age groups which are as follows: less than 40 years, 41-50 years, 51-60 years and above 61years. Among the 30 in control group, 2(6.7%) were less than 40 years, 8(26.6%%) were between 41-50 yrs , 14(46.6%) were between 51-60 years and 6(20.1%) were above 61 years. Among the 30 in oral leukoplakia group, 4(13.3%) were between 41-50 years, 18(60%) were between 51-60, and 8(26.7%) were above60 years. Among the 30 in oral cancer group, 1(3.3%) was between 41-50 years, 20(66.7%) were between 51-60 years, 9(30%)

were above 60 years. The p value is ≤0.018 which is statistically significant. This shows the positive correlation between the age and the occurrence of disease. This is in accordance with the following study.

Bánóczy J [45] made a follow-up study with 670 patients with oral leukoplakia during a 30-year-period showed cancer development in 40 cases. The age distribution revealed the prevalence of leukoplakia in the age-group 51-60 years; that of carcinoma in the age-group of 61-70 years. The sex distribution showed a male-female ratio of 3.2: 1 in the leukoplakia-group, and a 1.9 : 1 ratio in the carcinoma-group.

The distribution of habits was grouped as smoking only and smoking plus alcohol consumption. In Group I(controls), 30% had the habit of only smoking and 70% had the habit of smoking plus alcohol consumption. In Group II(oral leukoplakia), 10% had the habit of only smoking and 90% had the habit of smoking plus alcohol consumption. In Group III(oral cancer), 6.6% had the habit of only smoking and 93.4% had the habit of smoking plus alcohol consumption. The p value is ≤0.026 which is significant. This shows a positive correlation between alcohol usage and the occurrence of disease.

This result is in accordance with the following studies. **Dietrich T,**[47] made an analytical study on Clinical risk factors of oral leukoplakia and found the results as, Tobacco smoking as the strongest independent risk factor. The Odds Ratio were 3.00 (0.77-11.8) for < for =10 cigarettes/day and up to 6.01 (2.4-15.0) for >20 cigarettes/day. Diabetes, age and socio-

economic status were found as independent predictors of Oral leukoplakia. Alcohol consumption, race/ethnicity, years of education and Body Mass Index showed no independent association with Oral leukoplakia. Females with a history of estrogen use were less likely to have Oral leukoplakia with an Odds ratio of 0.34 (0.11-1.07).

Prakash C.Guptha[48] made an Epidemiologic study of the association between alcohol habits and oral leukoplakia. The study included 10914 individuals for their tobacco and alcohol habits and examined for the presence of oral leukoplakia. Very few females (1.6%) were found to be alcohol users and they were excluded from further analysis. Among 7604 males, 30.4% used alcohol regularly, 25.4% occasionally and 44.2% were non-users. The prevalence of leukoplakia was significantly higher among regular (5.7%) and occasional (3.9%) users than among non-users (2.9%) of alcohol.

In our study when arches were compared between the three groups, controls had less frequency of arches (2.3%) when compared to oral leukoplakia patients (6.0%) and OSCC patients (5.6%). When loops were compared controls had less frequency of loops (27.3%) when compared to oral leukoplakia patients (58.6%) and OSCC patients (61.7%). When whorls were compared controls had increased frequency of whorls (27.3%) when compared to oral leukoplakia patients (58.6%) and OSCC patients (61.7%). The p value was ≤ 0.001 which is highly significant. This is in accordance with studies conducted by **Hakan Polat et al**[75] and **Venkatesh et al**[5]**.**

In the study conducted by **Hakan Polat et al**[75] the finger print pattern distribution showed 7.2% arches, 57.2% ulnar loops, 2.4% radial loops and 33.1% whorls pattern in oral cancer patients and 3.9% arches, 56.0% ulnar loops, 4.9% radial loops and 35.3% whorls pattern in control group. The frequency of various finger print pattern is compared among the two groups and the result showed increased frequency of arches oral cancer patients. The P value was ≤0.05.

In the study conducted by **Venkatesh et al** [5] the finger print pattern distribution showed 6.3% arches, 63% loops and 30% whorls pattern in oral leukoplakia patients, 7% arches, 60.7% loops and 32.3% whorls pattern in OSCC patients and 2% arches, 30% loops and 68% whorls pattern in control group. The frequency of various finger print pattern is compared among the three groups and the result showed increased frequency of arches and loops in oral leukoplakia and OSCC patients whereas in control group there is an increased frequency of whorls. The x^2 was 109.493 and the P value was ≤0.001

In our study when the hypothenar pattern in the right hand was compared 24(80%) in control, 23(76.6%) in oral leukoplakia patients and 22(73.3%) in OSCC patients had pattern. When pattern in the left hand was compared 23 (76.6%) in control, 25(83.3%) in oral leukoplakia patients and 25(83.3%) in OSCC patients and had pattern. The p value is ≤0.912 which is insignificant. This is in accordance with studies conducted by **Hakan Polat et al**[75] and **Venkatesh et al**[5].

According to **Hakan Polat et al [75]** there is no significant difference observed in the hypothenar pattern between control groups and oral cancer patients. **Venkatesh et al [5]** found the distribution of pattern in hypothenar area among the three groups was statistically insignificant. In oral leukoplakia, 80% in right hand and 90% in left hand had pattern. In OSCC, 76.67% in right hand and 73.3% in left hand had pattern. In control group 80% in right hand and 83.3% in left hand had pattern. The X^2 value was 1.986 and the P value was 0.370.

In our study when the thenar I_1 pattern in the right hand was compared 25(83.3%) in control, 23(76.6%) in oral leukoplakia patients and 25(83.3%) in OSCC patients had pattern. When thenar I_1 pattern in the left hand was compared 26(86.6%) in control, 27(90%) in oral leukoplakia patients and 26 (86.6%) in OSCC patients had pattern. The p value is $\leq$0.993 which is insignificant. This is in accordance with studies conducted by **Hakan Polat et al[75]** and **Venkatesh et al[5]**.

According to **Hakan Polat et al [75]** there is no significant difference observed in the thenar pattern I_1 between control groups and oral cancer patients. **Venkatesh et al [5]** found the distribution of pattern in thenar area I_1 among the three groups was statistically insignificant. In oral leukoplakia, 86.67% in right hand and 76.67% in left hand had pattern. In OSCC, 90% in right hand and 80% in left hand had pattern. In control group 83.3% in right hand and 73.3% in left hand had pattern. The X^2 value was 0.891 and the P value was 0.64.

In our study when the thenar I_2 pattern in the right hand was compared 13(43.3%) in control, 14(46.6%) in oral leukoplakia patients and 13(43.3%) in OSCC patients had pattern. When thenar I_2 pattern in the left hand was compared 10(33.3%) in control, 11(36.6%) in oral leukoplakia patients and 10(36.6%) in OSCC patients had pattern. The p value is ≤0.985 which is insignificant. When thethenar I_3 pattern in the right hand was compared 11(36.6%) in control, 8(26.6%) in oral leukoplakia patients and 8(26.6%) in OSCC patients had pattern. When thenar I_3 pattern in the left hand was compared 10(33.3%) in control, 9(30%) in oral leukoplakia patients and 7(23.3%) in OSCC patients had pattern. The p value is ≤0.926 which is insignificant. When the thenar I_4 pattern in the right hand was compared 9(30%) in control, 13(43.3%) in oral leukoplakia patients and 14(46.6%) in OSCC patients had pattern. When thenar I_4 pattern in the left hand was compared 13(43.3%) in control, 13(43.3%) in oral leukoplakia patients and 13 (43.3%) in OSCC patients had pattern. The p value is ≤0.724 which is insignificant. This is not in consistence with the study conducted by **Hakan Polat et al[75]** and **Venkatesh et al[5]**. This may be because of the low sample size or regional variation.

According to **Hakan Polat et al [75]** there is no significant difference observed in the thenar I_2 and I_3 pattern between control groups and oral cancer patients. The percentage frequency of palmar dermatoglyphic pattern of patients with oral cancer showed less loops on thenar I_4 when compared with controls. The percentage was 24.1% in oral cancer patients

and 45.6% in controls and the p value was ≤0.05 which is statistically significant. **Venkatesh et al [5]** found the distribution of pattern in I_2, I_3 and I_4 area showed increased freaquency of loops in control group as compared to oral leukoplakia and OSCC patients. In oral leukoplakia, 33.33 in right hand and 21.11% in left hand had pattern. In OSCC, 18.88% in right hand and 21.11% in left hand had pattern. In control group 34.44% in right hand and 35.5% in left hand had pattern. The X^2 value was 13.109 and the P value was 0.011.

In our study no significant difference observed when comparing the total finger ridge count between the groups. In controls the mean value was 168.7 and standard deviation was 35.36. In oral leukoplakia patients the mean value was 158.87 and standard deviation was 39.18. In OSCC patients the mean value was 165.7 and standard deviation was 37.95. The p value is ≤0.457 which is insignificant. This is in accordance with studies conducted by **Hakan Polat et al[75]** and **Venkatesh et al[5]**.

Hakan Polat et al [75] found there is no significant difference observed in TFRC. In oral cancer the mean ± S.D. was 117±46.45 in males and 126.95±34.68 in females. In control group the mean±S.D. was 131.47±34.15 in males and 108.53±42.79 in females. The results were statistically insignificant. **Venkatesh et al [5]** found there is no significant difference observed in TFRC. In oral leukoplakia the mean value was 148.1 and the standard deviation was 42.58. In OSCC the mean value was 168.13% and the standard deviation was 43.56. In control group the mean

value was 168.43 and the standard deviation was 40.67. Frequency was 1.866 and the P value was 0.061.

In our study there is no significant difference observed when comparing the ab count on right hand. In controls the mean value was 39.27 and standard deviation was 6.198. In oral leukoplakia patients the mean value was 39.10 and standard deviation was 5.195. In OSCC patients the mean value was 37.43 and standard deviation was 5.811. The p value is ≤0.397 which is insignificant. There was no significant difference observed when comparing the ab count of left hand in all the three groups. In controls the mean value was 40.37 and standard deviation was 6.76. In oral leukoplakia patients the mean value was 39.67 and standard deviation was 4.97. In OSCC patients the mean value was 37.47 and standard deviation was 5.12. The p value is ≤0.121 which is insignificant. This is in accordance with studies conducted by **Hakan Polat et al**[75] and **Venkatesh et al**[5].

Hakan Polat et al [75] found there is no significant difference observed in ab count. In oral cancer the mean ± S.D. was 69.51±14.03 in males and 66.91±.9.64 in females. In control group the mean±S.D. was 76.07±11.89 in males and 74.07±7.28 in females. The results were statistically insignificant.

Venkatesh et al [5] found there is no significant difference observed in ab count. In oral leukoplakia the mean value was 38.77 in right hand and 38.87 in left hand. In OSCC the mean value is 38.57 in right hand and 40.17 in left hand. In control group the mean value in right hand was 40.67 and

41.47 in left hand. The P value was 0.339 for right hand and 0.309 for left hand.

In our study the atd angle of right hand is decreased in oral leukoplakia and OSCC patients when compared to controls. In controls the mean value was 40.53 and standard deviation was 3.026. In oral leukoplakia patients the mean value was 35.73 and standard deviation was 4.093. In OSCC patients the mean value was 34.53 and standard deviation was 2.063. The p value is ≤0.001 which is highly significant. The p value between controls and oral leukoplakia patients is ≤0.001 which is highly significant. The p value between controls and OSCC patients is ≤0.001 which is highly significant. The p value between oral leukoplakia patients and OSCC patients is ≤0.312 which is insignificant. In our study the atd angle of left hand is decreased in oral leukoplakia and OSCC patients when compared to controls. In controls the mean value was 41.03 and standard deviation was 3.079. In oral leukoplakia patients the mean value was 36.57 and standard deviation was 3.971. In OSCC patients the mean value was 34.40 and standard deviation was 2.111. The p value is ≤0.001 which is highly significant. The p value between controls and oral leukoplakia patients is ≤0.001 which is highly significant. The p value between controls and OSCC patients is ≤0.001 which is highly significant. The p value between oral leukoplakia patients and group III is ≤0.025 which is significant. This is in accordance with the study by **Hakan Polat et al**[75] and not in consistence with the study conducted by **Venkatesh et al**[5].

Hakan Polat et al [75] found there is significant difference observed in atd angle. In oral cancer the mean ± S.D. was 87.5±25.42 in males and 85.18±10.02 in females. In control group the mean±S.D. was 97.79±22.82 in males and 104.5±20.87 in females. The p value was ≤0.05 in males which is ststistically significant and the value was ≤0.01 in females which is statistically highly significant.

Venkatesh et al [5] there is no significant difference observed in atd angle. In oral leukoplakia the mean value was 40.33 in right hand and 39.93 in left hand. In OSCC the mean value is 39.93 in right hand and 38.50 in left hand. In control group the mean value in right hand was 40.93 and 39.96 in left hand. The P value was 0.609 for right hand and 0.206 for left hand.

This study deals with the evaluation of difference in Palmar dermatoglyphics of Oral Leukoplakia, Oral Squamous cell carcinoma and control group. This study was conducted between April 2010 to May 2011 in the department of Oral Medicine and Radiology of Ragas Dental College and Hospital, Dr. Rai Memorial Medical and Cancer Centre, Chennai.

A case control study was conducted in which 90 subjects were selected. The study subjects were categorized into three groups: Group I consist of 30 healthy individual with the habit of smoking but no evident lesions; Group II, 30 patients suffering from Oral leukoplakia; Group III, 30 patients suffering from oral cancer.

Patients only with the habit of smoking were included in the study, in control group smoking tobacco of any form; more than 10 numbers for more than 10 years were included in the study. Individuals with the habit of chewing and with the habit of both smoking and chewing and individuals with dermatological diseases or disorders or syndromes which affects the palmar region were excluded from the study.

The patients were made to sit comfortably on a dental chair. Sterile hand gloves were used during examination of the patient. Clinical diagnosis was made and patients who showed characteristic features of Leukoplakia, Oral Cancer and control group were prepared for sample collection. Subjects were asked to wash their hands with soap water, so as to remove any oil or dirt. The glass platen of the scanner is cleaned thoroughly to remove the dust. Then the patient was asked to place the right hand on the

top of the glass platen and instruction given to the patient not to move the hand or not to press the hand hardly against the glass platen. The image is previewed in the laptop screen using the scan Gear starter software then the image of the hand was scanned at 300dpi. The same procedure was repeated for the left hand and the thumb fingers then the images were stored in the laptop.

The finger and palm prints were analysed qualitatively and quantitatively using Photoshop 8.0 software. The qualitative analysis done include, finger print patterns and palmar patterns. The quantitative analysis done include, total finger ridge count, ab count and atd angles.

- In the present study all the 90 individuals are males (100%). As per the inclusion criteria only persons with smoking habits were included in the study. Hence the study result shows that the females with the habit of smoking are nil.
- The age of the subjects included in the study ranges between 35-70 years. Among the 30 in control group, 2(6.7%) were less than 40 years, 8(26.6%%) were between 41-50 yrs, 14(46.6%) were between 51-60 years and 6(20.1%) were above 61 years. Among the 30 in oral leukoplakia group, 4(13.3%) were between 41-50 years, 18(60%) were between 51-60, and 8(26.7%) were above60 years. Among the 30 in oral cancer group, 1(3.3%) was between 41-50 years, 20(66.7%) were between 51-60 years, 9(30%) were above 60 years. The p value is $\leq$0.018 which is statistically significant. This

shows the positive correlation between the age and the occurrence of disease.

- The distribution of habits was grouped as smoking only and smoking plus alcohol consumption. In controls, 30% had the habit of only smoking and 70% had the habit of smoking plus alcohol consumption. In oral leukoplakia patients, 10% had the habit of only smoking and 90% had the habit of smoking plus alcohol consumption. In OSCC patients, 6.6% had the habit of only smoking and 93.4% had the habit of smoking plus alcohol consumption. The p value is ≤0.026 which is significant. This shows a positive correlation between alcohol usage and the occurrence of disease.
- In our study when arches were compared between the three groups, controls had less frequency of arches (2.3%) when compared to oral leukoplakia patients (6.0%) and OSCC patients (5.6%). When loops were compared controls had less frequency of loops (27.3%) when compared to oral leukoplakia patients (58.6%) and OSCC patients (61.7%). When whorls were compared controls had increased frequency of whorls (27.3%) when compared to oral leukoplakia patients (58.6%) and OSCC patients (61.7%). The p value was ≤0.001 which is highly significant.
- When the hypothenar pattern in the right hand was compared 24(80%) in control, 23(76.6%) in oral leukoplakia patients and 22(73.3%) in OSCC patients had pattern. When hypothenar pattern

in the left hand was compared 23 (76.6%) in control, 25(83.3%) in oral leukoplakia patients and 25(83.3%) in OSCC patients and had pattern. The p value is ≤0.912 which is insignificant.

- When the thenar I_1 pattern in the right hand was compared 25(83.3%) in control, 23(76.6%) in oral leukoplakia patients and 25(83.3%) in OSCC patients had pattern. When thenar I_1 pattern in the left hand was compared 26(86.6%) in control, 27(90%) in oral leukoplakia patients and 26 (86.6%) in OSCC patients had pattern. The p value is ≤0.993 which is insignificant.
- When the thenar I_2 pattern in the right hand was compared 13(43.3%) in control, 14(46.6%) in oral leukoplakia patients and 13(43.3%) in OSCC patients had pattern. When thenar I_2 pattern in the left hand was compared 10(33.3%) in control, 11(36.6%) in oral leukoplakia patients and 10(36.6%) in OSCC patients had pattern. The p value is ≤0.985 which is insignificant.
- When thenar I_3 pattern in the right hand was compared 11(36.6%) in control, 8(26.6%) in oral leukoplakia patients and 8(26.6%) in OSCC patients had pattern. When thenar I_3 pattern in the left hand was compared 10(33.3%) in control, 9(30%) in oral leukoplakia patients and 7(23.3%) in OSCC patients had pattern. The p value is ≤0.926 which is insignificant.
- When the thenar I_4 pattern in the right hand was compared 9(30%) in control, 13(43.3%) in oral leukoplakia patients and 14(46.6%) in

OSCC patients had pattern. When thenar I_4 pattern in the left hand was compared 13(43.3%) in control, 13(43.3%) in oral leukoplakia patients and 13 (43.3%) in OSCC patients had pattern. The p value is ≤0.724 which is insignificant.

- In our study no significant difference observed when comparing the total finger ridge count between the groups. In controls the mean value was 168.7 and standard deviation was 35.36. In oral leukoplakia patients the mean value was 158.87 and standard deviation was 39.18. In OSCC patients the mean value was 165.7 and standard deviation was 37.95. The p value is ≤0.457 which is insignificant.
- In our study there is no significant difference observed when comparing the ab count on right hand. In controls the mean value was 39.27 and standard deviation was 6.198. In oral leukoplakia patients the mean value was 39.10 and standard deviation was 5.195. In OSCC patients the mean value was 37.43 and standard deviation was 5.811. The p value is ≤0.397 which is insignificant.
- There was no significant difference observed when comparing the ab count of left hand in all the three groups. In controls the mean value was 40.37 and standard deviation was 6.76. In oral leukoplakia patients the mean value was 39.67 and standard deviation was 4.97. In OSCC patients the mean value was 37.47 and standard deviation was 5.12. The p value is ≤0.121 which is insignificant.

- In our study the atd angle of right hand is decreased in oral leukoplakia and OSCC patients when compared to controls. In controls the mean value was 40.53 and standard deviation was 3.026. In oral leukoplakia patients the mean value was 35.73 and standard deviation was 4.093. In OSCC patients the mean value was 34.53 and standard deviation was 2.063. The p value is 0.000 which is highly significant. The p value between controls and oral leukoplakia patients is 0.000 which is highly significant. The p value between controls and OSCC patients is 0.000 which is highly significant. The p value between oral leukoplakia patients and OSCC patients is ≤0.312 which is insignificant.
- In our study the atd angle of left hand is decreased in oral leukoplakia and OSCC patients when compared to controls. In controls the mean value was 41.03 and standard deviation was 3.079. In oral leukoplakia patients the mean value was 36.57 and standard deviation was 3.971. In OSCC patients the mean value was 34.40 and standard deviation was 2.111. The p value is ≤0.001 which is highly significant. The p value between controls and oral leukoplakia patients is ≤0.001 which is highly significant. The p value between controls and OSCC patients is ≤0.001 which is highly significant. The p value between oral leukoplakia patients and group III is ≤0.025 which is significant.

Thus there is an increased frequency of arches and loops in oral leukoplakia and OSCC patients when compared with controls. In case of controls whorl pattern is predominant. Decreased atd angle in case of oral leukoplakia and OSCC patients when compared with controls.

The palmar pattern will not change after birth. This shows the genetic susceptibility in persons who develops oral leukoplakia and OSCC. Using these parameters, the persons has the habit of smoking and similar pattern can be identified at the earliest and preventive measures can be instituted in the susceptible individuals.

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Group I: Controls

				FINGER PRINT PATTERN			HYPO THENAR PATTERN		THENAR PATTERN-I1		THENAR PATTERN I2		THENAR PATTERN-I3		THENAR PATTERN-I4		TOTAL FINGER RIDGE COUNT	ab COUNT		atd ANGLE	
S. NO	PATIENT NAME	AGE	SEX	ARCH	LOOP	WHORLS	R	L	R	L	R	L	R	L	R	L		R	L	R	L
1	MR.BASKAR	52	M	0	2	8	0	1	1	0	1	0	0	1	0	0	184	32	31	37	38
2	MR. RAMAKRISHNAN	45	M	0	3	7	1	1	1	1	0	1	0	0	0	0	201	47	49	43	40
3	MR.DANRAJ	50	M	1	2	7	1	1	0	1	1	0	1	1	0	0	121	43	45	39	39
4	MR.KAVIKUMAR	47	M	0	4	6	0	1	1	1	0	1	0	0	1	0	210	28	26	51	49
5	MR.PARANTHAMAN	45	M	0	3	7	0	1	1	1	0	1	0	0	0	1	192	49	47	44	42
6	MR.RAMASWAMY	54	M	0	5	5	1	1	1	0	0	0	0	0	0	1	128	33	30	39	42
7	MR.MAILVAGANAN	59	M	0	3	7	1	1	1	1	1	0	0	1	1	1	177	35	38	40	39
8	MR.MANIKANDAN	48	M	1	4	5	1	1	1	1	1	0	0	0	1	0	110	44	49	41	44
9	MR.DHARMARAJ	51	M	0	2	8	1	1	0	1	0	1	0	1	0	0	182	36	38	38	36
10	MR.PARGUNAM	56	M	0	2	8	0	1	1	1	0	0	1	0	0	1	194	35	39	39	44
11	MR.GOVINDASAMY	57	M	0	4	6	1	1	1	1	0	1	0	0	1	1	179	39	41	40	42
12	MR.SADASIVAM	47	M	0	5	5	1	0	1	1	0	0	0	1	1	0	119	41	44	47	49
13	MR.SUBRAMANIAN	52	M	1	2	7	1	1	1	1	0	1	1	0	0	0	196	43	41	43	40
14	MR.JOHN VICTOR	61	M	0	3	7	1	1	0	1	1	0	0	1	0	0	199	37	41	42	44
15	MR.PUNIANATHAN	63	M	0	2	8	1	1	1	1	1	0	0	1	1	0	114	38	39	40	42
16	MR.SANGEEVAN	49	M	1	4	5	1	0	1	1	0	1	0	0	0	0	121	45	44	41	40
17	MR.GEORGE MATHEW	61	M	1	5	4	1	1	1	1	0	0	1	0	0	1	204	28	29	39	39
18	MR.SIVARAMAN	59	M	0	1	9	1	1	1	1	1	0	0	1	0	0	124	41	45	41	43

Group I: Controls

19	MR.KARUPASAMY	58	M	0	2	8	1	1	1	1	1	0	1	0	1	0	116	38	42	42	40
20	MR.KARTHIKEYAN	54	M	0	2	8	1	1	1	1	1	0	0	0	0	0	176	49	47	39	38
21	MR.MOHAMED IBRAHIM	53	M	0	2	8	1	0	1	1	0	1	0	1	0	0	182	29	31	38	41
22	MR.SUBRAMANIAN	65	M	0	3	7	1	1	0	1	0	0	1	0	1	0	171	37	35	41	39
23	MR.PERAMANATHAN	49	M	0	3	7	1	1	1	1	1	0	0	0	0	1	169	30	29	37	40
24	MR.SATHYAN	60	M	1	2	7	0	1	1	1	1	0	0	1	1	0	210	46	46	38	37
25	MR.KULANTHAI	59	M	0	1	9	0	1	1	1	0	1	1	0	1	1	188	37	39	40	38
26	MR.SEETHARAMAN	57	M	0	1	9	1	1	1	1	1	0	0	0	0	0	189	46	47	38	43
27	MR.SAMUEL NADAR	61	M	1	2	7	1	1	1	1	0	0	0	1	0	1	112	44	44	37	40
28	MR.SATHYASEELAN	53	M	0	3	7	1	1	1	1	1	0	1	1	1	0	182	39	41	43	44
29	MR.JAGANATHAN	60	M	0	3	7	0	0	1	0	0	1	1	1	0	1	198	46	49	40	41
30	MR.SIVACHANDRAN	54	M	0	2	8	1	0	0	0	0	0	0	0	0	0	213	43	45	39	38

Group II: Oral Leukoplakia

				FINGER PRINT PATTERN			HYPOTHENAR PATTERN		THENAR PATTERN-I1		THENAR PATTERN-I2		THENAR PATTERN I3		THENAR PATTERN I4		TOTAL FINGER RIDGE COUNT	ab COUNT		atd ANGLE	
S. NO	PATIENT NAME	AGE	SEX	ARCH	LOOP	WHORLS	R	L	R	L	R	L	R	L	R	L		R	L	R	L
1	MR.SATHYASEELAM	59	M	0	7	3	1	1	1	0	1	0	1	1	0	1	112	38	41	32	30
2	MR.KUMARESAN	57	M	1	7	2	1	0	0	1	0	1	0	0	0	0	203	41	43	35	34
3	MR.GANGATHARAN	64	M	1	6	3	1	1	1	1	1	0	1	1	1	0	123	42	45	33	30
4	MR.PARAMESHWARAN	58	M	1	7	2	0	1	1	1	0	1	0	0	0	0	114	24	23	37	36
5	MR.KANADASAN	49	M	0	5	5	1	1	1	1	0	1	0	0	0	1	178	41	38	34	34
6	MR.SIVARAMAN	59	M	2	6	2	1	1	0	1	0	0	0	0	0	1	112	35	37	36	33
7	MR.HANUMANTHA RAO	57	M	1	8	1	1	1	1	1	1	0	1	1	0	1	213	26	24	31	35
8	MR.AGUSTIN	60	M	1	5	4	0	0	1	0	1	0	0	1	0	0	196	37	42	35	36
9	MR.SENTHILNATHAN	63	M	2	6	2	1	1	0	1	0	1	1	0	0	0	194	52	44	36	34
10	MR.MUTHUKRISHNAN	64	M	0	7	3	1	1	0	1	0	0	0	0	0	0	115	39	40	34	32
11	MR.RAJASEKARAN	58	M	1	6	3	1	1	1	1	0	1	0	0	0	1	189	36	38	33	32
12	MR.MUNIRATHNAM	66	M	0	5	5	1	0	1	1	0	0	1	0	0	0	193	49	39	36	35
13	MR.SARGUNAM	61	M	1	6	3	1	1	1	0	0	1	0	0	1	0	113	41	37	35	34
14	MR.PARANTHAMAN	59	M	1	7	2	0	1	1	1	1	0	1	1	0	0	198	43	36	34	36
15	MR.SENGAI	57	M	0	5	5	1	1	1	1	1	0	1	1	1	0	204	37	38	36	35
16	MR.RAMACHANDRAN	63	M	1	7	2	1	0	0	1	0	1	0	0	0	0	191	38	41	35	36
17	MR.SIVARAMAN	58	M	0	6	4	1	1	1	1	0	0	0	0	1	1	197	28	33	37	33
18	MR.SUBRAMANIAN	64	M	1	4	5	1	1	1	1	1	0	1	1	0	0	161	35	42	33	36
19	MR.DHANASEKAR	53	M	1	7	2	0	1	1	1	1	0	0	1	1	0	189	37	38	34	34

Group II: Oral Leukoplakia

20	MR.VIJAYAN	58	M	0	6	4	1	0	1	0	1	0	0	1	0	0	147	39	36	32	30
21	MR.NIZZAR AHAMED	57	M	0	5	5	1	1	1	1	0	1	1	0	0	0	152	39	38	34	35
22	MR.RATHNASAMY	59	M	2	5	3	1	1	0	1	0	0	0	0	1	0	122	44	45	33	35
23	MR.PARISUTHAM	53	M	0	6	4	1	1	1	1	1	0	0	1	0	0	198	36	39	36	37
24	MR.SATHYARAJ	55	M	1	7	2	1	1	1	1	1	0	1	1	0	0	188	37	37	39	38
25	MR.ASHOKAN	57	M	1	5	4	0	1	1	0	0	1	0	0	1	0	189	31	36	32	36
26	MR.PAUL GEORGE	51	M	1	7	2	1	0	0	1	1	0	0	1	0	0	202	33	35	38	
27	MR.RAJARAJAN	58	M	0	8	2	1	1	1	1	0	0	1	0	0	1	96	34	30	36	
28	MR.RAVICHANDRAN	53	M	1	6	3	1	1	1	1	0	0	1	0	0	0	112	37	35	34	
29	MR.ELANCHEZLIYAN	56	M	1	7	2	1	0	1	1	1	1	1	1	1	0	159	36	39	30	
30	MR.VISWANATHAN	62	M	1	6	3	0	1	1	1	0	1	1	0	0	0	198	38	35	36	

Group III: Oral Squmaous Cell Cercinoma

				FINGER PRINT PATTERN			HYPOTHENAR PATTERN		THENAR PATTERN-I1		THENAR PATTERN-I2		THENAR PATTERN I3		THENAR PATTERN I4		TOTAL FINGER RIDGE COUNT	ab COUNT		atd ANGLE	
S.NO	PATIENT NAME	AGE	SEX	ARCH	LOOP	WHORLS	R	L	R	L	R	L	R	L	R	L		R	L	R	L
1	MR.RAVICHANDRAN	49	M	1	6	4	0	1	1	1	1	0	1	0	1	0	124	41	42	34	36
2	MR.PARANTHAMAN	58	M	1	7	3	1	1	1	1	0	1	0	0	0	1	104	44	38	43	47
3	MR.YAZAR ALI	61	M	0	7	3	1	1	1	1	1	0	1	0	0	0	108	26	28	33	35
4	MR. RAMANATHAN	68	M	1	5	4	0	0	1	0	0	1	0	1	1	0	203	39	42	45	44
5	MR.NARASIMAN	56	M	1	7	2	1	1	1	1	0	1	0	0	0	1	123	42	43	40	38
6	MR.CHANDRASEKAR	46	M	0	6	4	1	0	1	1	0	0	0	0	0	1	196	45	41	30	29
7	MR.SHANMUGAM	58	M	0	5	5	1	1	1	1	1	0	1	1	0	1	118	37	40	35	37
8	MR.RAJARATHINAM	59	M	1	6	3	1	1	1	1	0	0	1	1	0	0	184	36	37	34	36
9	MR.KAMALAKANNAN	63	M	1	7	2	0	0	0	1	1	1	0	0	0	0	201	24	26	34	33
10	MR.PAULRAJ	54	M	1	4	5	1	1	1	1	0	0	0	0	0	1	161	41	39	36	37
11	MR.PRABAKARAN	59	M	0	5	5	1	1	1	1	0	1	0	1	0	1	110	42	42	45	43
12	MR.KRISHNAMOORTHY	57	M	2	6	2	1	1	1	1	1	0	0	1	0	0	114	40	41	36	38
13	MR.SUBRAMANIAN	50	M	1	4	5	0	1	1	1	0	1	0	0	1	0	198	39	43	32	35
14	MR.ATHIRAJ	62	M	1	7	2	1	1	1	1	1	0	1	0	0	0	204	43	45	33	34
15	MR.KUMARAPAN	53	M	1	5	4	0	0	1	0	1	0	1	1	0	0	189	44	39	38	34
16	MR.SASIVARNAM	60	M	0	6	4	1	1	0	1	0	1	0	0	0	0	208	40	41	35	35

17	MR.RENGANATHAN	61	M	0	7	3	0	1	1	1	0	0	0	0	1	1	123	39	40	36	37
18	MR.JAMAL AHAMED	54	M	1	6	3	1	1	1	1	1	0	1	0	0	0	163	37	43	35	36
19	MR.PRINCE	57	M	0	6	4	1	1	1	1	0	0	1	1	1	0	196	43	44	34	37
20	MR.KESAVAN	53	M	0	7	3	1	1	1	1	0	0	1	0	0	0	123	41	39	36	38
21	MR.RAJALINGAM	52	M	1	5	4	0	1	1	1	1	1	0	0	0	0	122	42	41	33	34
22	MR.RAJARAMAN	50	M	2	4	4	1	1	1	1	0	0	0	1	1	0	187	39	44	32	36
23	MR.PURUSOTHAMAN	61	M	1	7	2	1	1	1	1	0	0	1	0	0	0	184	45	47	34	33
24	MR.CHANDRAN	64	M	0	6	4	1	1	0	0	1	0	1	1	0	0	192	41	40	32	36
25	MR.SEKAR	58	M	0	7	3	1	1	0	1	0	1	0	1	0	0	114	43	37	34	35
26	MR.BABU RAJ	53	M	1	5	4	1	1	1	1	0	0	1	0	1	0	167	27	26	33	32
27	MR.ANZAR	56	M	1	5	4	1	1	1	1	1	0	0	0	0	1	114	37	39	39	42
28	MR.SELVARAJ	54	M	0	6	4	1	0	1	1	1	0	0	1	0	0	96	38	43	34	33
29	MR.ALAGARASAN	58	M	0	6	4	0	1	0	1	1	1	1	1	1	0	192	41	40	32	33
30	MR.PUSHPARAJ	61	M	1	6	3	1	1	1	1	1	1	0	1	0	1	188	37	40	45	44

RAGAS DENTAL COLLEGE & HOSPITAL
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DEPARTMENT OF ORAL MEDICINE & RADIOLOGY

CASE SHEET PROFORMA

Date:

Serial No. Op. No.

Name: Age/ Sex:

Religion:

Occupation: Income:

Address: Phone no**:**

Study group : Group I / Group II / Group III

Smoking:

- Duration of smoking (<10 yrs ,10-20 yrs / 20-30 yrs / >30 yrs)
- Frequency of smoking per day (<5 times / 6-10times / 11-20times / >20 times)

Alcohol Consumption:

- Duration of alcohol consumption (<10 yrs ,10-20 yrs / 20-30 yrs / >30 yrs)
- Frequency of alcohol consumption per month (<5 times / 6-10times / >11times)

Leukoplakia :

Site :

Size :

Type :

Oral Squamous Cell Carcinoma :

Site :

Staging :

RIGHT HAND

FINGERS	THUMB	INDEX	MIDDLE	RING	LITTLE
DISTAL PHALANX RIDGE PATTERN					
RIDGE COUNT					
INTER DIGITAL SPACE	I1	I2	I3	I4	HYPOTHENAR
RIDGE PATTERN					

AFRC -
TFRC -
a-b count -
atd angle -

LEFT HAND

FINGERS	THUMB	INDEX	MIDDLE	RING	LITTLE
DISTAL PHALANX RIDGE PATTERN					
RIDGE COUNT					
INTER DIGITAL SPACE	I1	I2	I3	I4	HYPOTHENAR
RIDGE PATTERN					

AFRC -
TFRC -
a-b count -
atd angle -

CONSENT LETTER

I _____ the undersigned hereby give my consent for the performance of recording my palm and finger print to study the **" Palmar Dermatoglyphics in the patients of Oral Leukoplakia and Oral Squamous Cell Carcinoma"** conducted by Dr.S.Ramasubramanian under the able guidance of Dr.Capt.S.Elangovan M.D.S., Professor , Department of Oral Medicine and Radiology, Ragas Dental College and Hospital, Chennai. I have been informed and explained the status of my disorder, evaluation procedure, risk involved and likelihood of success. I also understand and accept this as a part of study protocol, thereby voluntarily, unconditionally, freely give my consent without any fear or pressure in mentally sound and conscious state to participate in the study.

Witness/ Representative Patient signature

(If any) Date:

ஒப்புதல் படிவம்

சென்னை, ராகாஸ் பல்மருத்துவக்கல்லூரி மற்றும் மருத்துவமனையின் வாய் மருத்துவம் மற்றும் ஊடுகதிர் துறையின் தலைவர் மற்றும் பேராசிரியர் **மரு.கேப்டன்**, **எஸ்.இளங்கோவன்** அவர்களின் மேற்பார்வையில், முதுநிலை (MDS) பட்டபடிப்பு பயிலும் மரு. S.ராமசுப்பிரமணியன் அவர்கள் மேற்கொள்ளும் வாய் வெண்படலம் மற்றும் வாய் புற்று நோய் உள்ள நோயாளிகளின் உள்ளங்கை தோல் வரையியல் பற்றி ஆராய்ந்து பார்க்க நடத்தும் ஆராய்ச்சிற்கான பரிசோதனைகளுக்கு என்னை உட்படுத்துவதற்கு _____ என்கின்ற நான் எனது மனமுவுந்த பரிபூரண சம்மதத்தினை அளிக்கிறேன்.

மேலும் எனக்கு என்னுடைய நோயின் தன்மையைப் பற்றியும், இப்பரிசோதனை பற்றியும், அதனால் ஏற்படக்கூடிய விளைவுகளைப் பற்றியும் எடுத்து கூறப்பட்டுள்ளது எனவும், இந்த பரிசோதனைக்கு, நான் எந்தவித அச்சமுமின்றி தன்னிச்சையாகவும், தெளிவான முழுமனதுடன் என்னுடைய பரிபூரண சம்மதித்தனை அளிக்கிறேன் என இதன் மூலம் தெரியப்படுத்துகிறேன்.

இப்படிக்கு

சாட்சியாளர்கள்

சென்னை

தேதி