ROLE OF DERMATOGLYPHICS IN MALIGNANT AND POTENTIALLY MALIGNANT DISORDERS OF THE ORAL CAVITY - A CROSS-SECTIONAL STUDY

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CERTIFICATE BY THE GUIDE

This is to certify that **Dr.N.THILAGAVATHI,** Post graduate student (2017-2020) in the Department of Oral Medicine and Radiology (**Branch IX)**, Tamilnadu Government Dental College and Hospital, Chennai 600003, has done this dissertation titled **"Role of dermatoglyphics in malignant and potentially malignant disorders of the oral cavity – a cross-sectional study"** under my direct guidance and supervision in partial fulfillment of the M.D.S. degree examination in May 2020 as per the regulations laid down by Tamilnadu Dr. M.G.R. Medical University, Chennai- 600 032 for M.D.S., Oral Medicine and Radiology (Branch – IX) degree examination.

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TITLE OF DISSERTATION	**"ROLE OF DERMATOGLYPHICS IN MALIGNANT AND POTENTIALLY MALIGNANT DISORDERS OF THE ORAL CAVITY - A CROSS-SECTIONAL STUDY"**
PLACE OF STUDY	**Tamil Nadu Government Dental College and Hospital, Chennai-600003**
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I, **Dr.N.THILAGAVATHI,** hereby declare that no part of the dissertation will be utilized for gaining financial assistance/any promotion without obtaining prior permission of the Principal, TamilNadu Government Dental College and Hospital, Chennai 600003. In addition, I declare that no part of this work will be published either in print or in electronic media without the guide who has been actively involved in the dissertation. The author reserves the right to publish the work with the prior permission of the Principal and Guide, TamilNadu Government Dental College & Hospital, Chennai - 600003.

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LIST OF CONTENTS

ABBREVIATIONS

PMD	Potentially Malignant Disorder
OL	Oral Leukoplakia
OSF	Oral Submucous Fibrosis
OSCC	Oral Squamous Cell Carcinoma
WHO	World Health Organisation
PVL	Proliferative Verrucous Leukoplakia
TSN	Tobacco Specific Nirosamines
GST	Glutathione S Transferase
LH	Loss Of Hetrozygosity
MI	Microsatellite Instability
EGFR	Epidermal Growth Factor Receptor
APC	Adenomatous Polyposis Coli
AFIS	Automated Fingerprint Identification Systems

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Urkund Analysis Result

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ABSTRACT

TITLE: "Role of dermatoglyphics in malignant and potentially malignant disorders of the oral cavity – a cross-sectional study".

AIM:

The aim of the present study is to identify the genetic predilection of malignant and potentially malignant disorders based on the dermatoglyphics pattern.

OBJECTIVES:

1. To compare the dermatoglyphics of
   A) patients with malignant and potentially malignant disorders of oral cavity
   B) patients with habits but without lesion
   C) patients without habit and
2. To analyse which dermatoglyphic pattern is common among the patients with malignant and potentially malignant disorders.

METHODS:

Fingerprints and palm prints were studied in 300 patients from Tamilnadu government dental college and hospital were studied, who were randomly divided into three groups: (A) 100 subjects with OSF, OL and OSCC, (B) 100 patients with habits and no lesions, and (C) 100 healthy controls, for the purpose of finding patterns that could identify patients with PMDs and OSCC. Finger and palm prints were taken by the ink method. Prints were analysed.

RESULTS:

The results were tested for statistical significance. Weighted kappa statistics were used to evaluate the inter- and intra-observer agreement. It was observed that the arch pattern (9.3%) was pre-dominant in group A compared with control group B(3.4%) and group C(0.6%) with a decrease in whorl pattern (39.5%) in group A when compared with the controls group B(45.7%) and group C(50.7%)and the difference was statistically significant ($P < 0.01$). The study group demonstrated an decrease in the mean total finger ridge count and ATD angle as compared to the controls and the result was found to be highly significant ($P < 0.05$).

CONCLUSION: Dermatoglyphics can be implemented as a screening tool in patients with PMDs and OSCC. Thereby, we can identify patients at increased risk for oral cancer so that risk reduction measures or earlier therapy may be instituted.

KEYWORDS: Dermatoglyphics, Oral leukoplakia, Oral Squamous cell carcinoma, Oral Submucous fibrosis

INTRODUCTION

Dermatoglyphics is defined as the study of the epidermal ridges and their configurations on the fingers, palms and soles. The term dermatoglyphics was coined by Cummins and Midlo in 1926 which was derived from the Greek word derma meaning skin and glyphics meaning carvings.[1]

The formation of the dermatoglyphics pattern occurs between 7th and 21st week of intrauterine life and gets fully completed by 7th month of intrauterine development. Finger ridge counts and frequencies of all palm patterns follow the genetic modes of major genes and it would never be influenced by environment or age factors. It does not change throughout the life of individuals except in events such as bruises and cuts of the fingertips[2].These finger and palmar prints are permanent variables and inherited, differ amongst parents and their children, siblings and even in monozygotic twins.

Because of these characteristics it has been used in forensic department for the identification of the individuals, and in the study of several genetic abnormalities. The first study on genetic abnormalities with dermatoglyphics pattern were done by Harold Cummins in Down's syndrome patient.[3]

Oral leukoplakia is considered as one of the most common premalignant lesion occuring in the oral cavity with increased risk of malignant transformation ranging from 0.6% to 20%. Oral leukoplakia (OL) is defined as a white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.[4]

Oral submucous fibrosis (OSF) on the other hand is a premalignant condition associated mainly with tobacco chewing characterized by abnormal collagen deposition, ulceration, xerostomia, burning sensation and restricted mouth opening. It predominantly affects South East Asian population. The risk of malignant transformation is 1.5% to 15%.[5]

Oral squamous cell carcinoma (OSCC) is the most common malignant tumour of the oral cavity which arises from the stratified squamous cell epithelium of the oral mucosa.

Though various epidemiologic studies suggest that the use of tobacco (chewing or non-chewing) is an important risk factor for the development of malignant and potentially malignant disorder of the oral cavity, not all individuals develop the same. It seems genetic predisposition could be an underlying mechanism. It increases the susceptibility of malignancy with disordered function of the genes controlling the fate of chromosomally damaged cells and the cell cycle tumor suppressor genes and genes involved in cell signaling (proto-oncogenes and oncogenes).

Hence the assessment of genetic abnormalities also plays an essential role with the diagnostic procedure of PMD (Potentially malignant disorder) and OSCC. It can be determined by various genetic studies. As such these procedures are complex and expensive, hence dermatoglyphics can be efficiently employed with other clinical signs as a screening procedure as a noninvasive, simple, and inexpensive procedure for the assessment of genetic susceptibility. [6]

AIM AND OBJECTIVES

AIM:

The aim of the present study is to identify the genetic predilection of malignant and potentially malignant disorders based on the dermatoglyphics pattern.

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  A) Patients with malignant and potentially malignant disorders of oral cavity

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  C) Patients without habit and

- To analyse which dermatoglyphic pattern is common among the patients with malignant and potentially malignant disorders.

REVIEW OF LITERATURE

POTENTIALLY MALIGNANT DISORDER:

In 1978 World Health Organisation(WHO) coined the terms 'pre-cancer', 'precursor lesions', 'pre-malignant', 'intra epithelial neoplasia' to broadly describe the clinical presentations that may have a potential to become cancer. They classified it into two group

- A precancerous lesion is 'a morphologically altered tissue in which oral cancer is more likely to occur than in its apparently normal counterpart';
- A precancerous condition is 'a generalized state associated with a significantly increased risk of cancer'.

At earlier times, it was considered that the origin of a malignancy in the mouth of a patient with precancerous lesion would correspond with that site of precancer. On the other hand, in precancerous conditions, cancer may develop in any anatomical site of the mouth or pharynx. Now it is known that even the clinically 'normal' appearing mucosa in a patient having a precancerous lesion may have dysplastic features on the contralateral anatomic site which suggest a pathway to malignant transformation, and that cancer could arise even in apparently normal tissue. Hence, it did not favour subdividing precancer to lesions and conditions and thus all clinical presentations that carry a risk of cancer gave under the term '*potentially malignant disorders*' - to reflect their widespread anatomical distribution.

In 2005 WHO described potentially malignant disorders as 'lesions and conditions that have the risk of malignant transformation either during the time of

initial diagnosis or at a future date'. Not all lesions described under this term may transform to cancer, rather that there is a family of morphological alterations amongst which some may have an increased potential for malignant transformation. Potentially malignant disorders of the oral mucosa are also risk indicators of likely future malignancies elsewhere in (clinically normal appearing) oral mucosa and not only site specific predictor.[7]

ORAL SUBMUCOUS FIBROSIS:

TERMINOLOGY

In 1952, Schwartz found five Indian women, from East Africa, with "atrophia idiopathica (tropica) mucosae oris." in 1953, Joshi an indian coined the term submucous fibrosis of the palate and pillars. Other names includes diffuse oral submucous fibrosis, idiopathic scleroderma of the mouth, idiopathic palatal fibrosis and sclerosing stomatitis.

DEFINITION: (Pindborg JJ 1966)

Oral Submucous fibrosis may be defined as an insidious, chronic disease affecting any part of the oral cavity and sometimes extending to pharynx. Although occasionally preceded by or associated with vesicle formation, it, is always associated with a juxta-epithelial inflammatory reaction followed by a fibroelastic change of the lamina propria, with epithelial atrophy leading to stiffness of the oral mucosa and causing trismus and inability to eat with burning sensation of the mouth.

EPIDEMIOLOGY:

Pindborg JJ et al., (1968) [8] in their epidemiological survey on OSF concluded that there is an increased incidence in urban population with incidence rate of 0.18%-1.2% when compared with rural population it is 0.04% - 0.4% .

Ranganathan K et al., (2004) [9] in a study conducted at Chennai, South India, reported a mean age of 32.4±10.4 years and median age of 29 years for OSF occurrence. The youngest and oldest ages of occurrence in his study was 16 and 76 years in males and 24 and 57 years in females.

ETIOPATHOGENESIS:

OSF is multifactorial in origin. Most studies on OSF have emphasized only the role of irritant substances acting locally on the oral mucosa. An equally important second aspect which needs to be considered is the pre-conditioning of the oral mucosa by a prolonged, chronic deficiency of iron and/or vitamin B complex. Such conditions are much more commonly seen among Indian females than males, which may explain the higher incidence of OSF among females.[10]

LOCAL FACTORS:

The pathogenesis of OSF was at first linked with the continuous and prolonged action of mild irritants on the oral mucosa.

1. **Areca nut (betel nut):**

The relationship of OSF to chewing of areca nut/quid or pan masala has been directly related to OSF, whereas chewing or smoking tobacco did not increase the risk for OSF.

Betel quid is composed of betel leaf (piper betle), areca nut (*Areca catechu*) and slaked lime (calcium hydroxide).

Tobacco quid contains betel leaf (piper betle), areca nut (*Areca catechu*), slaked lime (calcium hydroxide) with tobacco.

Pan masala is a betel nut product. There are different types of pan masala available called pan parag, gutkha, mawa etc. Gutkha and mawa are currently and widely used. However gutkha has been banned but still available in the grey market.

Gutkha is a chewing tobacco preparation made of crushed areca nut, tobacco, catechu paraffin wax, slaked lime and sweet or savory flavourings.

Mawa is a mixture small pieces of arecanut, processed tobacco and slaked lime,

Role of areca nut in pathogenesis of OSF:

- Arecoline, an active alkaloid found in betel nuts. Stimulates fibroblasts to increase production of collagen by.
- To elevate the mRNA and protein expression of cystatin C, a nonglycosylated basic protein consistently up-regulated the variety of fibrotic diseases, in a dose-dependent manner in persons with OSF.
- Areca nuts have also been shown to have a high copper content, and chewing areca nuts for 5-30 minutes significantly increases soluble copper levels in oral fluids. This increased level of soluble copper supports the hypothesis as an initiating factor in individuals with OSF.[11]

2. Capsacin:

Sirsat & Khanolkar et al., [10] verified this by applying capsaicin, the active irritant in chillies (Capsicum annum, which is used to spice food), to rat palates and found only a limited connective tissue response in the unimpaired animal. In proteindepleted or vitamin B-deficient animals, however, the response was more widespread and extensive.

SYSTEMIC FACTORS:

1) Nutritional deficiencies:

- Iron deficiency anemia, Vitamin B complex deficiency and malnutrition are promoting factors that derange the repair of the inflamed oral mucosa, leading to defective healing and resultant scarring.
- The resultant atrophic oral mucosa is more susceptible to the effects of chillies and betel nuts. Mucosal changes similar to those in Vitamin B and Iron deficiency are seen in oral sub mucous fibrosis.[11]

2) **Genetic and Immunologic Processes:**

More recently, the expression profiles of genes in OSF and normal oral mucosa have been studied more intensively. In one study, 14,500 genes were analyzed using gene chip arrays. The study demonstrated 716 genes were upregulated and 149 genes were downregulated in OSF. The gene expression profiles of normal controls and OSF patients were clearly distinct, in particular the genes involved in immune response, inflammatory response, and TGF-β–induced epithelial–mesenchymal transition.[12]

In a comprehensive analysis of water-soluble and ethanol-soluble areca nut constituents, it was demonstrated that both alkaloid and polyphenol fractions induced TGF-β signaling in human keratinocytes. Involved genes included *TGF-β2*, *SMAD-3*, matrix metalloproteinase (*MMP)1*, *MMP2*, and *MMP9*, and others. In contrast, no TGF signaling was induced in fibroblasts.[13]

It can be assumed that direct effects on epithelial cells with TGF-β activation can suppress antifibrogenetic cytokines, including bone morphogenetic protein-7 and stimulated fibroblast activity. Both OSF and OSCC development are quite complex and it is unlikely that a single factor is responsible.[14]

STAGES OF ORAL SUBMUCUOUS FIBROSIS (OSF):

Pindborg JJ in 1989 divided OSF into three stages

Stage 1:

- Stomatitis includes erythematous mucosa, vesicles, mucosal ulcers, melanotic mucosal pigmentation, and mucosal petechia.

Stage 2:

- Fibrosis occurs in ruptured vesicles and ulcers when they heal, which is the hallmark of this stage. Early lesions demonstrate blanching of the oral mucosa.
- Older lesions include vertical and circular palpable fibrous bands in the buccal mucosa and around the mouth opening or lips, resulting in a mottled, marble like appearance of the mucosa because of the vertical, thick, fibrous bands running in a blanching mucosa. Specific findings include the following:

- Reduction of the mouth opening (trismus).
- Stiff and small tongue.
- Blanched and leathery floor of the mouth.
- Fibrotic and depigmented gingiva.
- Rubbery soft palate with decreased mobility.
- Blanched and atrophic tonsils.
- Shrunken budlike uvula.
- Sinking of the cheeks, not commensurate with age or nutritional status.

Stage 3:

- Squeal of OSF are as follows:
- Leukoplakia is precancerous and is found in more than 25% of individuals with OSF.
- Speech and hearing deficits may occur because of involvement of the tongue and the eustachian tubes.[15]

DIAGNOSIS:

The hallmark of diagnosing OSF is by clinical examination. Clinically, one or more of the following symptoms should be present:

- Blanching of oral mucosa defined as a persistent, white, marble-like appearance of the oral mucosa, which may be localized, diffuse or reticular
- Tough, leathery texture of the mucosa
- Palpable, whitish, fibrous bands.
- On histopathologic examination OSF is characterized by epithelial atrophy with loss of rete ridges and hyalinization of the lamina propria and the underlying muscle.

- The initial pathology of OSF is characterized by mixed inflammation and edema, and large fibroblasts. Later, collagen bundles with early hyalinization are seen, and the inflammatory infiltrate contains lymphocytes and plasma cells, occasionally resembling lichenoid mucositis.[16]

MANAGEMENT:

Conventional therapies in the treatment of OSF are empirical and symptomatic in nature. The major targets of treatment can be summarized as:

-anti-inflammatory

-oxygen radical-scavenging

-antifibrotic drugs.

- Depending on severity of disease, physical therapy and/or surgery is added to drug therapy.
- Patients might benefit from physical therapy in conjunction with drug treatment. The more advanced OSF is, the more limited is the efficacy of pharmacological treatment. Patients may benefit from surgery or laser surgery in such situations.
- During the early inflammatory phase of OSF, corticosteroids are of potential benefit, as suggested by in vitro studies. OSF has also been treated with hyaluronidase, chymotrypsin and collagenase, pentoxifylline, nylidrin hydrochloride, iron, and lycopene among others, but the level of evidence for any of these attempts is low.
- A 6-week course of intralesional injections of 4 mg dexamethasone/mL and 1,500 U hyaluronidase twice weekly improved trismus and other clinical parameters associated with fibrosis. In addition, autofluorescence

of the affected mucosa normalized for collagen and nicotinamide adenine dinucleotide (reduced form) spectra.

- A combination of micronutrients and minerals was evaluated in a single-arm study. Significant improvement in symptoms was observed after 1–3 years of treatment. The interincisor distance was stable in 49% of patients and improved in 41%, and leukoplakia regressed.
- Oxitard is a phytopharmacological complex of antioxidant activity.

In Santosh Patil et al., (2014) done a study in a group of 120 OSF patients, the efficacy of oxitard two capsules per day was compared to topically applied 0.5% aloe vera three times daily for 3 months. Subjective symptoms like burning pain and difficulty in swallowing, and mouth opening and tongue protrusion were significantly more improved with oxitard.

- Lactoferrin is a biologically active compound of bovine milk. Lactoferrin can also be produced by recombinant technology. The compound is not only immune modulating, resulting in increased antiviral and antibacterial activity of intestinal mucosa, but improves cancer surveillance and has anti-inflammatory effects.
- IFN-γ that inhibits the collagen synthesis was given intralesionally in an open uncontrolled study. IFN-γ treatment showed improvement in the patient's mouth opening with a net gain of 8±4 mm (42%) of interincisor distance 6 months later. Histochemical investigations demonstrated effects on inflammation and collagen metabolism in favor of antifibrotic activity.

LEUKOPLAKIA

DEFENTION:

The WHO in 1997 described leukoplakia as "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion".[17]

In 2007, Warnakulasuriya et al defined as "Leukoplakia should be used to recognize white plaques of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer" and stated that leukoplakia is a clinical term and the lesion has no specific histology. It may show atrophy or hyperplasia (acanthosis) and may or may not demonstrate epithelial dysplasia.[18]

INCIDENCE AND PREVALENCE:

The author **Stefano Petti done a study in 2003** stated that the true global leukoplakia prevalence is very likely to fall between 1.7% and 2.7% and the average of the annual malignant transformation rate was 1.36%.[19]

MALIGNANANT TRANSFORMATION:

The carcinomatous transformation of oral leukoplakia is not predictably associated with tobacco smoking, and the frequency of carcinomatous transformation of idiopathic leukoplakia is higher than that of tobacco-associated leukoplakia.

The rates of progression of large oral leukoplakias (>5 mm) and of leukoplakias at sites in the mouth known to be at most risk of developing

carcinoma (floor of mouth, ventrolateral surface of tongue, and maxillary retromolar/soft palate region) are greater than for smaller leukoplakias or for leukoplakias at other sites in the mouth.[20]

Charles A. Waldron et al., [21] in his study stated that the risk of epithelial dysplasia, carcinoma in situ, or carcinoma varied between the anatomical locations of leukoplakia. The incidence of epithelial alteration, ranging from dysplasia to carcinoma, was 42.9% for lesions of the floor of the mouth, 24.2% for tongue lesions, and 24.0%for lip leukoplakias. The incidence of similar epithelial alterations in other sites varied from 18.8% for palatal lesions to 11.7% for leukoplakias of the retromolar area.

Brouns ER et al[22] in 2014 in his study 16 of 144 patients (11%), malignant transformation occurred between 20 and 94 months (mean 57.0 months) after the first visit, the annual malignant transformation rate being approximately 2.6%. A large size of the lesion (≥ 4 cm) showed to be the only statistically significant predictor of malignant transformation ($P = 0.034$).

Louis S. Hansen et al[23] done a long-term study of 30 patients with proliferative verrucous leukoplakia (PVL). Patients were followed for 1 to 20 years. In his study he reported that Thirteen died of or with their disease, 14 were alive with PVL, and 3 were alive without PVL at last contact. PVL rarely regressed despite therapy. All patients who died had persistent or recurrent disease.

ETIOLOGY:

The etiology of OL is considered multifactorial, but smoking is appreciated to be a frequently involved factor. It is much more common among smokers than among non-smokers. There are conflicting results of studies related to the possible role of human papillomavirus infection particularly with regard to exophytic, verrucous leukoplakia. [24]

G. Ramaswamy et al[25] in 1996 done a study stated that the serum vitamin levels of vitamin A, B12, C, beta carotene and folate acid were significantly decreased in patients with oral leukoplakia compared to controls, whereas serum vitamin E was not.

CLINICAL TYPES

There are two main clinical types. The distinction of these is purely clinical, based on surface colour and morphological (thickness) characteristics, and do have some bearing on the outcome or prognosis

1. **Homogeneous leukoplakia** - uniformly flat, thin and exhibit shallow cracks of the surface keratin. The risk of malignant transformation is relatively low.
2. **Non-homogeneous leukoplakia** - a much higher risk of malignant transformation
    - **speckled:** mixed, white and red, but retaining predominantly white character
    - **nodular:** small polypoid outgrowths, rounded red or white excrescences;
    - **verrucous:** wrinkled or corrugated surface appearance.

The consensus view of the working group was that broadly dividing leukoplakia to homogeneous or nonhomogeneous categories was imprecise and of limited value. However, those with mixed white and red plaques should be recognized as having a higher risk status. These are to be denoted as erythroleukoplakia'.

- **Proliferative verrucous leukoplakia (PVL):** a disease of unknown origin, which exhibits a strong tendency to develop areas of carcinoma. PVL begins as a simple hyperkeratosis but tends to spread and become multifocal. PVL is slow-growing, persistent, and irreversible, and in time areas become exophytic, wartlike, and apparently resistant to all forms of therapy as recurrence is the rule. The disease was most commonly seen in elderly women and had been present for many years.[18]

OLEP classification system[26]

L (size of the leukoplakia)

L1 - Size of single or multiple leukoplakias together ≤2 cm

L2 - Size of single or multiple leukoplakias together 2 to 4 cm

L3 - Size of single or multiple leukoplakias together ≥4 cm

Lx - Size not specified

P (pathology)

P0 - No epithelial dysplasia (includes ``no or perhaps mild epithelial dysplasia")

P1 - Distinct epithelial dysplasia (includes "mild to moderate" and "moderate to possibly severe" epithelial dysplasia)

Px - Absence or presence of epithelial dysplasia not specified in the pathology report

OLEP staging system

Stage I - L1P0

Stage II - L2P0

Stage III - L3P0 or L1L2P1

Stage IV - L3P1

MANAGEMENT:

Management of oral leukoplakia should begin with elimination of risk factors (if any) such as tobacco abuse, betel chewing, alcohol abuse, superimposed candida infection over the lesion etc.

Conservative treatment includes use of chemopreventive agents such as vitamins (vitamins A, C, E), fenretinide (Vitamin A analogue), carotenoids (betacarotene, lycopene), bleomycin, protease inhibitor, anti-inflammatory drugs, green tea, curcuma etc.

Surgical treatment includes conventional surgery, electrocoagulation, cryosurgery, and laser surgery (excision or evaporation).

The main purpose of oral leukoplakia management is to avoid malignant transformation of the lesion or if this happened to detect this in early stages.[27]

ORAL CARCINOMA:

Oral cancer is the sixth most common cancer worldwide, with a high prevalence in South Asia.[28] It Is traditionally defined as a squamous cell carcinoma (OSCC), because in the dental area, 90% of cancers are histologically originated in the squamous cells.[29]

EPIDEMIOLOGY:

The areas characterised by high incidence rates for oral cancer (excluding lip) are found in the South and Southeast Asia (e.g. Sri Lanka, India, Pakistan and Taiwan), parts of Western (e.g. France) and Eastern Europe(e.g. Hungary, Slovakia and Slovenia), parts of Latin America and the Caribbean (e.g. Brazil, Uruguay and Puerto Rico) and in Pacific regions(e.g. Papua New Guinea and Melanesia).

Age and gender:

In most countries around the world, oral cancer is more common in men than in women. The reported sex differences are attributable to heavier indulgence in risk habits by men and exposure to sunlight (for lip cancer) as a part of outdoor occupations. The ratio of males to females diagnosed with oral cancer, however, has declined over the decades and is now about 1.5:1 . The risk of developing oral cancer increases with age and the majority of cases occur in people aged 50 or over.

Anatomic sites:

Tongue is the most common site for intraoral cancer among European and the US populations, amounting to 40–50% of oral cancers. Buccal cancer is more

common among Asian populations due to betel quid/tobacco chewing habits. In Sri Lanka, 40% of oral cavity cancers are found on buccal mucosa. Other intraoral sites for mouth cancer include floor of mouth, gingivae and palate.[30]

ETIOLOGY AND RISK FACTORS:

The two main factors which influence most diseases are genetic and epigenetic factors. Development of oral or head and neck squamous cell carcinoma (HNSCC) is influenced by both these factors.

EPIGENETIC FACTORS:

TOBACCO:

Tobacco in various forms like smoked and smokeless tobacco, have carcinogenic impact in oral cavity. The most important carcinogens in tobacco smoke are the aromatic hydrocarbon benz‑pyrene and the tobacco‑specific nitrosamines (TSNs). Their metabolites covalently bind with deoxyribonucleic acid (DNA) of keratinocyte stem cells forming DNA adducts. These adducts are responsible for critical mutations involved in DNA replication.

The metabolism of these carcinogens involves oxygenation by P450 enzymes in cytochromes and conjugation by glutathione‑S‑transferase (GST). Genetic polymorphisms in the genes coding for these enzymes are suspected to play a key role in the genetic predisposition to tobacco‑induced head and neck cancers[31]

ALCOHOL:

Alcohol consumption has been shown to act synergistically with tobacco in the increased risk of development of oral cancer.[31]

DIET AND NUTRITION:

The relationship between diet and nutrition to the risk of cancer development has been established by several epidemiological and laboratory studies. certain micronutrients decrease the risk of oral cancer development. Certain micronutrients like vitamins A (retinol), C (AA), and E (α‑tocopherol); carotenoids (β‑carotene); potassium; and selenium decrease the risk of oral cancer development. β‑carotene, retinol, retinoids, vitamin C (AA), and vitamin E (α‑tocopherol) are antioxidants that are essential in reducing free radical reactions that can cause DNA mutations, changes in enzymatic activity, and lipid peroxidation of cellular membranes.[31]

VIRUS INFECTION:

HPV are the most common viruses implicated in oral carcinogenesis. HPV are DNA viruses and are epitheliotropic, especially for squamous epithelia. Certain HPV types, referred to as 'high‑risk' types are associated with OSCC and oral premalignant lesions. They are HPVs 16, 18, 31, 33, 35, and 39.

The major evidence of the role of HPV in cancer development is that their genes and gene products are capable of disturbing the cell cycle machinery. HPV encodes two major oncoproteins namely, E6 and E7. The E6 and E7 proteins have been shown to bind and destroy p53 and Rb tumor suppressor genes, respectively,

thereby disrupting the cell cycle with loss of control on DNA replication, DNA repair, and apoptosis.[32]

FUNGAL INFECTION:

Fungal infections caused by Candida species, in particular, *Candida albicans* has been implicated in the pathogenesis of oral premalignant lesions. Superficial fungal hyphae of *Candida albicans* have been found superimposed on leukoplakia, especially nodular leukoplakia, many of which have undergone malignant transformation. The doubt of whether Candida invasion is a secondary event or causal in oral premalignant lesions is still uncertain and debatable. Candida species are commensals in the oral cavity which become opportunistic during host's immunosuppression due to systemic diseases or drug therapy. Besides immunocompromised individuals, Candida infection can coexist or be associated with other risk factors like irondeficiency and in chronic smokers which may prove synergistic in the development of oral cancer. There is evidence that Candida possesses necessary enzymes fromdietary substances to produce nitrosamines and chemicals that have been implicated in carcinogenesis. A recent study showed relationship between oral yeast carriage and epithelial dysplasia yet again, the actual role of yeast in the development of epithelial dysplasia is uncertain.[31]

DENTAL FACTORS:

Poor oral hygiene, poor dental status (sharp/fractured teeth due to caries/trauma), and chronic ulceration from an ill-fitting denture has been suggested to promote neoplasm in the presence of other risk factors. There has

been difficulty in obtaining the evidence whether dental factors influence oral cancer development. This is due to the presence of coexisting risk factors like smoking and alcohol consumption. Nevertheless, an experimental study in hamsters has shown that chronic trauma in addition to carcinogen application could promote tumor development. In this study, mechanical irritation by scratching with a pulp cleaner has been shown to significantly increase the incidence of a chemical carcinogen-induced tongue carcinoma. Therefore, it is prudent to closely monitor patients with known risk factors for signs and symptoms of irritation from teeth and appliances.[31]

GENETIC FACTORS:

Genetic predisposition has been shown to be an important risk factor in the development of OSCC. A study by Copper e*t al.,* who followed up first-degree relatives of 105 head and neck cancer patients, found that 31 of these subjects developed cancers of respiratory tract and upper aerodigestive tract. However, population-based studies to determine the genetic or familial disposition to oral cancers are limited by the coexisting risk factors like smoking and alcohol. It is also believed that certain individuals inherit the susceptibility of inability to metabolize carcinogens or procarcinogens and/or an impaired ability to repair the DNA damage. As discussed earlier about the metabolism of tobacco carcinogens, genetic polymorphisms in the genes coding for the enzymes (P450 enzymes and XMEs) responsible for tobacco carcinogen metabolism are suspected to play key role in the genetic predisposition to tobacco-induced head and neck cancers.[31]

MOLECULAR PATHOGENESS:

Oral carcinogenesis is a multistep process in which genetic events lead to the disruption of the normal regulatory pathways that control basic cellular functions including cell division, differentiation, and cell death. Genetic alterations known to occur during carcinogenesis including point mutations, amplifications, rearrangements, and deletions.[33]

GENOMIC INSTABILITY:

Genomic instability such as loss of hetrozygosity (LOH) and microsatellite instability (MSI) are frequently observed in cancer.

In a recently proposed model of tumor progression in HNSCC, deletions of chromosomes 3p, 9p, and 17p have been associated with the transition from normal mucosa to dysplasia, whereas carcinomas were characterized by additional deletions of 4q, 6p, 8, 11q, 13q, and 14q.

Ulrike Bockmuhl et al[33] in his study showed that all well-differentiated carcinomas revealed a characteristic pattern of alterations consisting of DNA loss of chromosomes 3p, 5q, and 9p associated with a DNA overrepresentation of 3q. The profile of all undifferentiated carcinomas showed additional changes, ie, deletions of chromosomes 4q, 8p, 11q, 13q, 18q, 21q, and overrepresentations of 1p, 3q, 11q13, 19, and 22q.

ONCOGENE:

Oncogenes are altered growth promoting regulatory genes that govern the cells' signal transduction pathways, and mutation of these genes leads to either

overproduction or increased function of the excitatory proteins. Several oncogenes have been implicated in oral carcinogenesis. Aberrant expression of the proto-oncogene epidermal growth factor receptor (EGFR/c-erb 1), members of the ras gene family, c-myc, int-2, hst-1, PRAD-1, and bcl-1 is believed to contribute towards cancer development.[33]

TUMOUR SUPPRESSOR GENE:

Oncogenes alone are not sufficient to cause oral cancer and appear to be initiators of the process. The crucial event in the transformation of a premalignant cell to a malignant cell is inactivation of cellular negative regulators—tumour suppressor genes—and is regarded to be a major event leading to the development of malignancy.

The tumor suppressor genes like p53, doc-1, the retinoblastoma gene, and adenomatous polyposis coli APC The p53 protein blocks cell division at the G1 to S boundary, stimulates DNA repair after DNA damage, and also induces apoptosis.

Mutation of p53 occurs either as a point mutation, which results in a structurally altered protein that sequesters the wild-type protein, thereby inactivating its suppressor activity, or by deletion, which leads to a reduction or loss of p53 expression and protein function.[34]

TNM classification of OSCC- AJCC

Primary tumour (T)

Tx – Primary tumour cannot be assessed

Tis – Carcinoma in situ

T1- Tumour ≤ 2cm with depth of invasion ≤ 5mm

T2 - Tumour ≤ 2cm with depth of invasion > 5mm or

Tumour >2cm and ≤4 cm with depth of invasion ≤ 10mm

T3 - Tumour >2cm and ≤4 cm with depth of invasion > 10mm or

Tumour >4cm with depth of invasion ≤ 10mm

T4 – Moderately advanced or very advanced local disease

T4a - Moderately advanced local disease

Tumour >4cm with depth of invasion > 10mm or

Tumour invades adjacent structures only

T4b – Very advanced local disease

Tumour invades masticator space, ptreygoid plates or skull base

Regional lymph node – N

Nx – Regional lymph nodes cannot be assessed

N0 – No regional lymph node metastasis

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

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

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

N2c - Metastasis in bilateral or contralateral lymph nodes, none larger than 6cm in greatest dimension

N3 – Metastasis in a lymph node larger than 6cm in its greatest dimension

M – Distant metastasis

M0 – No distant metastasis

M1 – Distant metastasis

Mx – Distant metastasis cannot be assessed

AJCC Prognostic Stage Groups

TisN0M0 – 0

T1N0M0 – I

T2M0N0 – II

T3N0M0 – III

T1/T2/T3N1M0 – III

T4aN0/N1M0 – IVA

T1/T2/T3/T4aN2M0 – IVA

Any TN3M0 – IVB

T4banyNM0 – IVB

AnyTanyNM1 – IVC

ORAL CANCER SURVIVAL RATES:

Despite recent diagnostic and therapeutic advances, the 5-year survival rate for oral cancer has persisted at less than 50% over the last 50 years owing to the following reasons: first, the bulk of oral cancer cases, i.e. around 60% of

cases, were noticed in their advanced stages (III and IV). Secondly, the maximum number of oral cancer cases runs the risk of transforming into secondary tumors ("field cancerization phenomenon") compared with other cancers.

The 5-year recurrence-free survival rate in oral cancer patients is 80% for those diagnosed in stage I and only 20% for those diagnosed in stage IV. Moreover, diagnosis at early stages of oral cancer greatly reduces treatment related morbidity and improves the long-term survival rate. Patients with a history of oral cancers are at risk of developing secondary tumors at a rate of 3.7% per year. Because of "field cancerization," one fourth of all oral cancer-related deaths are caused by secondary tumors. Hence, patients who are effectively treated for oral cancer should be closely monitored, preferably using a noninvasive diagnostic test.[35]

DERMATOGLYPHICS

Dermatoglyphics is the study of finger prints from palms, fingers, soles and toes of both humans and animals. It is epidermal in origin. The fingerprints are permanent which are not the same even in monozygotic twins and it is the last one that decomposes after a person dies.[36]

HISTORICAL BACKGROUND OF DERMATOGLYPHICS

The science of dermatoglyphics has its own significance. It can be used for medical studies, for identification of the person, identification in the crime, physical anthropology. It had been obtained from the research work of four people-Henery Faulds, William Herschel, Francis Galton and E.R. Henry.

William Herschel (1858) was the first to experiment with fingerprints in India promoted finger print system and proved that the fingerprint never get altered with age.

Galton (1892) is the one who brought the fingerprint for personal identification and its importance in disease identification. His original classification of whorls, loops, arches, still hold good in the field of dermatoglyphics and he proved the hereditary basis of fingerprints. He had tried to establish the three fingerprint patterns with standard methods and obtained the results from the collected data.

Sir Edward Henry (1893) improved system of classification was accepted and is been used even today. He published the book 'The classification and uses of fingerprints,' commencing a modern era of fingerprint identification

Cummins and Midlo (1926) coined the term dermatoglyphics. Dr Harold Cummins father of dermatoglyphics done a classic work in the year 1943, "Fingerprints, Palms and Soles: An introduction to Dermayoglyphics" which has been accepted and used as a standard reference.[37]

As the state-of-the-art which led to the computerization of fingerprint record files. Automated Fingerprint Identification Systems (A.F.I.S.) is working in many parts of the country.

A. F. I. S. not only stores record cards in computer memory, it will also match latent fingerprints from crime scenes to its data bank.

WORKING PRINCIPLES OF DERMATOGLYPHICS:

1) FIRST PRINCIPLE:

A fingerprint is an individual characteristic. No two digits have similar ridge characteristics

2) SECOND PRINCIPLE:

A fingerprint will never change during an individual's lifetime.

3) THIRD PRINCIPLE:

Fingerprints have general ridge patterns which make it possible to classify systematically.

IMPORTANCE:

In the present society it becomes one of the important tools for human biologist. Personal identification which was pointed out by Henry Fauld (1880) and Francis Galton (1892) which led to the greatest contribution in the law enforcing departments for the identification of the criminals. Used in biometrics based electronic gadgets to prevent impersonation. The value of dermatoglyphics increases by its advancing use in the clinical investigations with the rapid growth in human genetics along with the discovery of chromosomal aberration in human. Used in anthropology department not only in the context of twin diagnosis (monozygotic and dizygotic) but also in disputed paternity diagnosis, primatology and biological variation among different populations etc.

Methods of Recording Dermatoglyphics

Reviewing the literature one can find number of methods for recording dermatoglyphics. Variability lies in their requirements for equipment, time and experience, and in the quality of the prints produced.

The various methods that can be employed are:

i) Ink method,

ii) Inkless method,

iii) Transparent adhesive tape method, and

iv)Photographic method.

The most common method used for dermatoglyphic studies is the ink method first described by Cummins and Midlow (1943). Other methods have been tried which include a 'Scotch-tape India-ink' method which is an inkless method using sensitizing fluid, adhesive tape, powder, and carbon paper. Recent "hi-tech" methods are generally computer based and begin by scanning prints with a video camera followed by digitizing the print features which are then subjected to analysis. Okajima (1975) developed a method to study ridges on the dermal surface instead of the epidermal surface using chemical treatment and staining with toluidine blue that can be done even in fetuses from the 14th gestational week. Misumi et al (1984) used scanning electron microscope. Others have used Rubber and Plaster of Paris casts also. These methods are costly or cumbersome.

FIXED POINTS IN THE IMPRESSION:

a)INNER TERMINUS OR CORE

B)OUTER TERMINUS OR DELTA

CLASSIFICATION OF FINGER RIDGE PATTERN:

Galtone stabilised the first classification of finger prints in 1888. He classified the three basic fingerprint patterns based on the number of triradii (deltas) which was later revised by E.R. Henry.

1) LOOP:

Around 60-65% of the population has loops. The ridges pass from one side of the digit curves and exit from the same side. The ridges curve around only one extremity of the pattern which forms the head of the loop. From the opposite extremity of the pattern, ridges flow to the margin of the finger, this extremity of the pattern is described as 'open'. Subdivided into 2 types.

a)Ulnar loop: Here the loop open medially towards the little finger. As ulna is the medial bone.

b)Radial loop: Here the loop open laterally towards the thumb. As the radius is the lateral bone. All loops must have one delta. The core is the center of the loop.

2) WHORL:

Around 30-35% of the population has whorl. It is distinguished by concentric design with the ridges make circuits around the core. It has 4 types

a) Plain: it forms the complete circuit and an imaginary line one delta to the other must touch a whorl ridge.

b) Central pocket: it also makes a complete circuit, here the imaginary line from one delta to the other cannot touch a whorl ridge.

c) Double loop: here two loops combined to make one whorl.

d) Accidental : other types not in these three category is accidental.

True whorls typically possess two deltas.

3)ARCH:

Only 5% of the population has arches. Arch ridges tend to enter from one side of the digit and exit on the other side. 2 types

a)plain arches: it tend to show a wave like pattern.

b)Tented arches: it shows a sharp spike at the center of the arch.

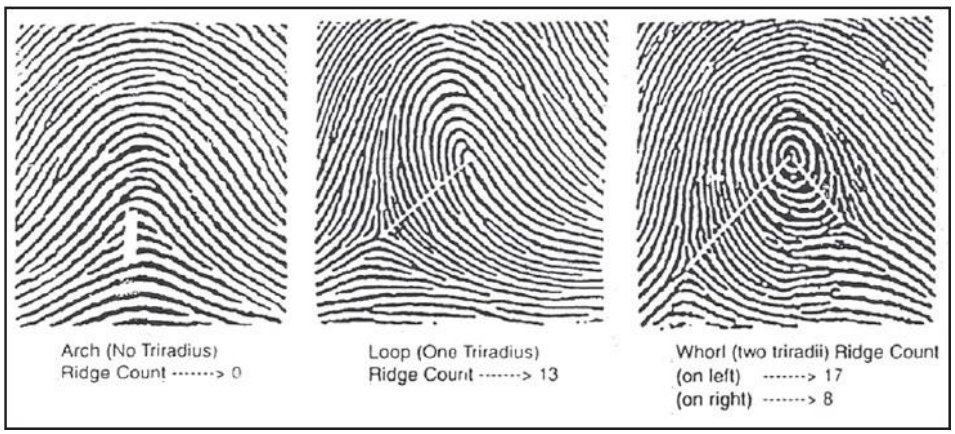
Arches do not have deltas or cores.

TOTAL FINGER RIDGE COUNT (TFRC)

It is the sum of ridge counts on all the fingers of both hands. it is a quantitative trait with inheritance.

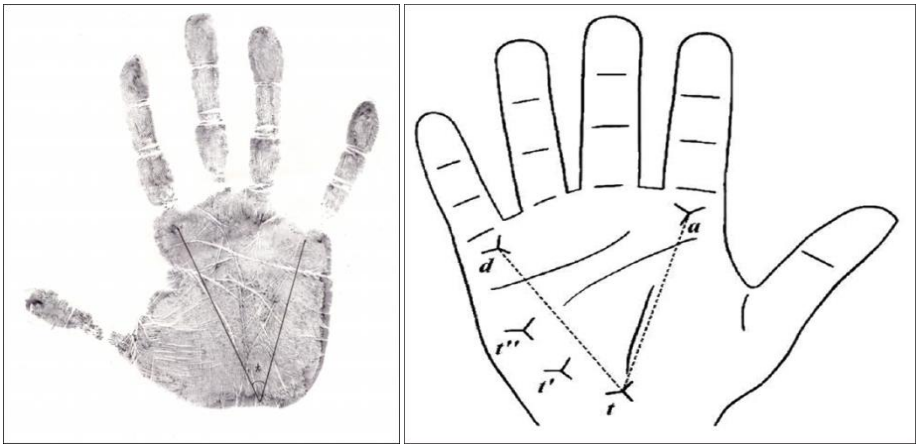
1. One ridge must be a looping ridge.
2. The delta and core are not included in the count.
3. Fragments and dots are counted as ridges only if they appear as prominent as the surrounding ridges.
4. If there is a bifurcation, both of its arms should be counted.
5. If the delta is on the only loop, there will beno ridge cont. "0" count
6. White space must present between the delta and the first ridge count.

TFRC = adding individual ridge counts of 10 fingers. Arches have a ridge count of zero. For loops, the number of ridges between the tri-radius and the center or core of the pattern is counted. For whorls, from each tri- radius to the center of the fingerprint is counted and the higher of the two possible counts is recorded.[38]



ATD Angle:

An angle formed by lines drawn from the digital triradius (a) to the axial triradius (t) and from this to digital triradius (d) is called atd angle. The more distal the position of t, the larger is the atd angle.



DERMATOGLYPHICS AS A BIOMARKER:

DERMATOGLYPHICS AND SYNDROMES:

Trisomy 21 (Down's Syndrome):

The hands and feet of a patient with trisomy 21 are generally short and broad. There is a high frequency of simian creases, incurved fifth digit (clinodactyly) with or without a short or missing middle phalanx, and a wide space between the first and second toe, with a deep plantar crease. The dermatoglyphic features more frequent in trisomy 21 than in normal individuals include ulnar loops; radial loops on the fourth and fifth digit; I3 patterns; high triradii, often with a hypothenar pattern[39]

Turner's Syndrome:

Short fifth finger. And the Atd angle is more compared to the normal individual Atd > 120.Ab ridge count > 105.Bilateral hypothenar pattern will be present [39]

Rubinstein-Taybi syndrome11:

Bilateral I2 and I3 patterns will be present. Four or more arches in the fingertips.[39]

DERMATOGLYPHICS IN DENTISTRY:

DENTAL CARIES:

- **PR Abilash et al, [40]** in his case – control study on 1250 children in the age group of 5 to 12 years from Chennai corporation school showed that, the dental caries susceptibility of an individual increased with incidence of whorl pattern and it decreased with incidence of loop pattern.
- **Rokaya H. Ahmed et al[41]** done a study on 32 patients with dental caries who replaced it with amalgam and composite fillings then compared with 15 normal persons. The result of his study shown that in patient group there was Increased incidence of whorl pattern whereas in control group there was increased incidence of loop pattern, Total finger ridge count was higher in study group compared to the control group.

The patterns in the thenar and I-interdigital area were only arches in control group while in patient group were arches and radial loops. Moreover, in the IV-interdigital area, a higher number of distal loops found in control group while the patient group has a higher frequency of arch patterns. In the hypothenar area, some whorl patterns appeared only in the patient group, Atd angle was between 45 and 56° which was more than 56 degrees in the control group.

CLEFT LIP AND PALATE:

L Mathew et al[42] done a Dermatoglyphic study in 50 oral cleft children and 50 normal children showed that the oral cleft individuals had an increased frequency of ulnar loops as the ridge configuration as compared to the normal children who had a higher frequency of whorl patterns. And the mean atd angle in

the cleft children was 47.440 which is greater than 450 and the mean atd angle of the normal children was 41.820. The fluctuating asymmetry of the atd angle of the oral cleft children were found to be increased in the higher ranges of 6-90(14%) and >100 (12 %) when compared to the normal children.

POTENTIALLY MALIGNANT AND MALIGNANT DISORDERS:

Veena Kulkarni[43] did a study consisted of 150 randomly selected subjects categorized into 3 groups. 50 patients with OSF, 50 gutkha chewing subjects without OSF and 50 normal subjects without gutkha chewing habit showed that. Significant findings in qualitative analyses of OSF patients include Increase in frequency of arches, both hands taken together. Decrease in frequency of simple whorls, both hands taken together. Increase in pattern frequency in thenar/I area in both hands. Significant findings in quantitative analysis of OSF patients include; Decrease in atd angle in both right and left hands.

Elluru Venkatesh et al[1] in their study 30 subjects with OSCC, 30 subjects with oral leukoplakia and 30 healthy controls were evaluated qualitatively and quantitatively. Among oral leukoplakia group 06.30% have arches, 06.30% have loops and 30.70% have whorls Among OSCC 07.00% have arches, 60.70% have loops and 32.30% have whorls Among control group 02.00% have arches, 30.00% have loops and 68% have whorls, Showed than Arches and loops were more frequent in cases than in controls whereas whorls were more frequent in control group.

Lakshmana N et al[3] based on their study conducted that loops were found to be the predominant fingerprint patterns with a frequency of 59.73% followed by whorls, 34.14% and arches, 6.13% in OSCC (Group 1) whereas loops were found to be the predominant fingerprint patterns with a frequency of 61.2% followed by whorls, 32% and arches, 6.8% in oral leucoplakia and OSF (Group 2). As far as the control group (Group 3) was concerned, whorls were the predominant fingerprint patterns in this group with a frequency of 50.27% followed by loops, 44% and arches, 5.73%. Thus In oral leukoplakia, OSF and OSCC patients, loops were found to be the predominant finger ridge patterns whereas whorls were predominant in the control group.

A study conducted by **Athreya Vijayaraghavan et al[44]** showed that there is predominance of arches and loops, presence of hypothenar pattern, decrease in mean ATD angle and total AB ridge count in OSF and Oral Cancer patients. on comparison with the controls, the ATD angle in the OSF and oral cancer patients was less than 45°. In OSF patients, it was 41.88° with a standard deviation of ±2.21 and in oral cancer patients it was 40.95° with a standard deviation of ±2.46.

A study conducted by **Gupta et al. (2013) [45]** also showed promising results by observing an increased frequency of arches and ulnar loop patterns on fingertips with a decreased frequency of simple whorl patterns on fingertips and a decreased frequency of palmar accessory triradii on the right and left hands in OSCC patients. Significant findings in OSF patients included an increase in the frequency of arches and ulnar loop pattern, decrease in the frequency of simple

whorl patterns on fingertips, decrease in ATD angle on the right hand, and a decrease in the frequency of palmar accessory triradii on the right hand

Tamgire Dw et al[46] did a digit wise comparison and found highly significant decrease in simple whorl pattern on left little finger as compared with OSF group. Increase in composite whorl pattern on left little finger OSF subjects when compared with control. 3) Decrease in composite whorl pattern of right index finger in OSF when compared with control. 4) Increase in simple whorl pattern on right thumb in OSF when compared with control. 5) Increase in composite whorl pattern on left thumb in OSF as compared with control. 6) Decrease in radial loop on left index finger in OSF when compared with control.

MATERIALS AND METHODS

Patients attending the outpatient Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital with oral cancer, oral leukoplakia and oral submucous fibrosis were selected for the study.

STUDY POPULATION:

Total population of 300 patients were included in the study. They were categorized into three groups, as mentioned below:

Group A: It includes100 patients with oral leukoplakia (OL), oral submucous fibrosis (OSF), and oral squamous cell carcinoma (OSCC).

Group B: It includes 100 patients with habits (such as tobacco smoking or chewing and consumption of alcohol) and without lesions.

Group C: it includes 100 healthy patients without any habits.

INCLUSION CRITERIA:

- Clinically diagnosed OSF, histopathologically confirmed Leukoplakia and oral squamous cell carcinoma patients.
- Individuals with habit and without disease.
- Healthy individual without any disease and habit

EXCLUSION CRITERIA:

- Patient who are not able to co-operate
- Patients without histopathological confirmation of premalignant and malignant lesion.

- Patients who are not willing to participate in the study.
- Patients with oral lesions due to other etiologic factors like cavities, sharp tooth irritation, dentures, aphthous ulcers, etc.

The approval from the ethical committee was obtained prior to the study. The procedure was explained to the patients and Informed consent was obtained from them before getting the finger prints. It was explained to the patient, that the purpose of procuring the finger prints was only for academic purpose, would never be misused and that the confidentiality would be maintained.

METHODOLOGY

A detailed history of clinically diagnosed cases of OSF, OL, and OSCC was taken and recorded in a case history proforma. Incisional biopsy was done. After histopathologic confirmation they were included in the study. The finger and palm prints of patients with history of tobacco-related habits (smoking/smokeless), pan masala, betel nut chewing (Group A and B), and normal patients of similar age group and sex without any tobacco-related habits (Group C) were taken for the study.

The finger and palm prints were obtained by the Ink method one of the most common method, which was described by Cummins and Midlo in 1943. The armamentarium used in study includes duplicating ink, stamp pad, white paper, magnifying lens, protractor, scale and pencil. Patients were asked to wash the hands thoroughly washed with soap before taking prints to remove oil or any dirt. Then the ink was applied on the palms and it was spread evenly on the palms and fingers.

Then the patient was asked to keep the finger on white paper and firm pressure was applied on the center of the dorsum of hand, and inter digital areas. The finger prints were recorded by placing it on a white paper with one lateral aspect of the finger and then it is rolled it towards the opposite side. It is then analysed with the use of magnifying lens. The dermatoglyphics patterns were analysed both qualitatively and quantitatively.

QUALITATIVE ANALYSIS:

Fingerprint patterns were classified as loops, whorls and arches based on Galtone's classification.

- Patterns of all the 10 fingers in both hands were analysed.

  The frequency of each pattern was recorded and the percentage of pattern frequency was calculated for the entire group.
- Palmar patterns were observed in the hypothenar (Hy), thenar/interdigital 1 (Th/I1) area, and interdigital 2, 3 and 4 (I2,3,4) areas of the palms.

Various patterns encountered in both hands were noted. The frequency of palmar patterns was calculated in both hands and it is compared with the study groups.

QUANTITATIVE ANALYSIS:

The following parameters were analysed on the palmar patterns:

- Total finger ridge count: Total finger ridge count was calculated for all 10 fingers of both right and left hand.

- ATD angle: Generally there are four digital triradii termed as a, b, c and d present at the distal portion proceeding in the radio-ulnar direction in the palm. The axial triradius ‘t’ is found usually near the proximal palmar margin. The ATD angle is a dermatoglyphic trait formed by drawing lines from the triradii ‘a’ below the first digit to the most proximal triradius ‘t’ in the hypothenar region of the palm and from this to the triradii below the last digit ‘d’. ATD angle were measured in both hands.

FIGURES

Figure 1: Armamentarium for biopsy

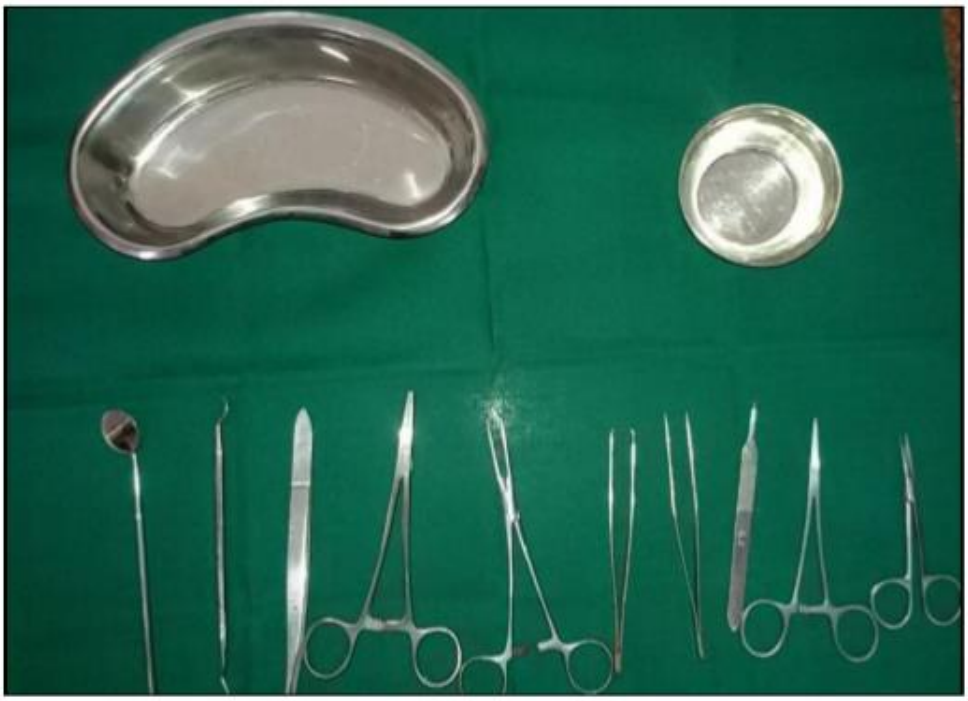


Figure 2: Armamentarium used for dermatoglyphics study

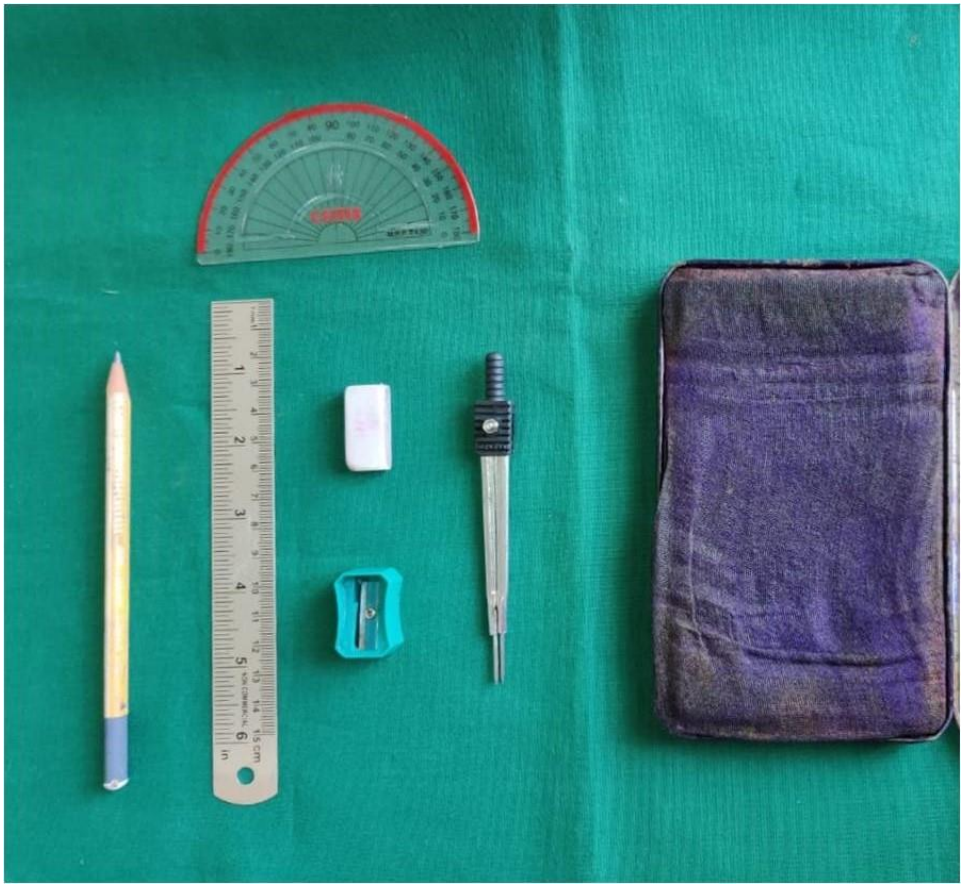


Figure 3: Inked hand for making finger and palmar prints

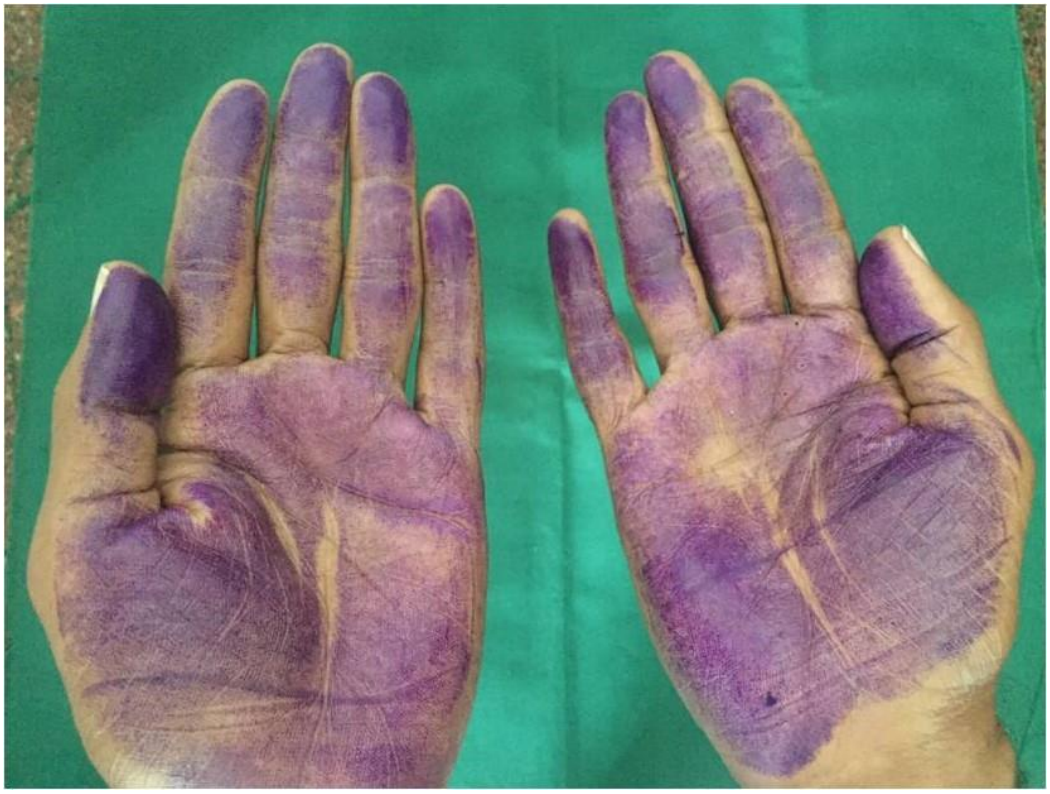


Figure 4: Palmar and finger prints

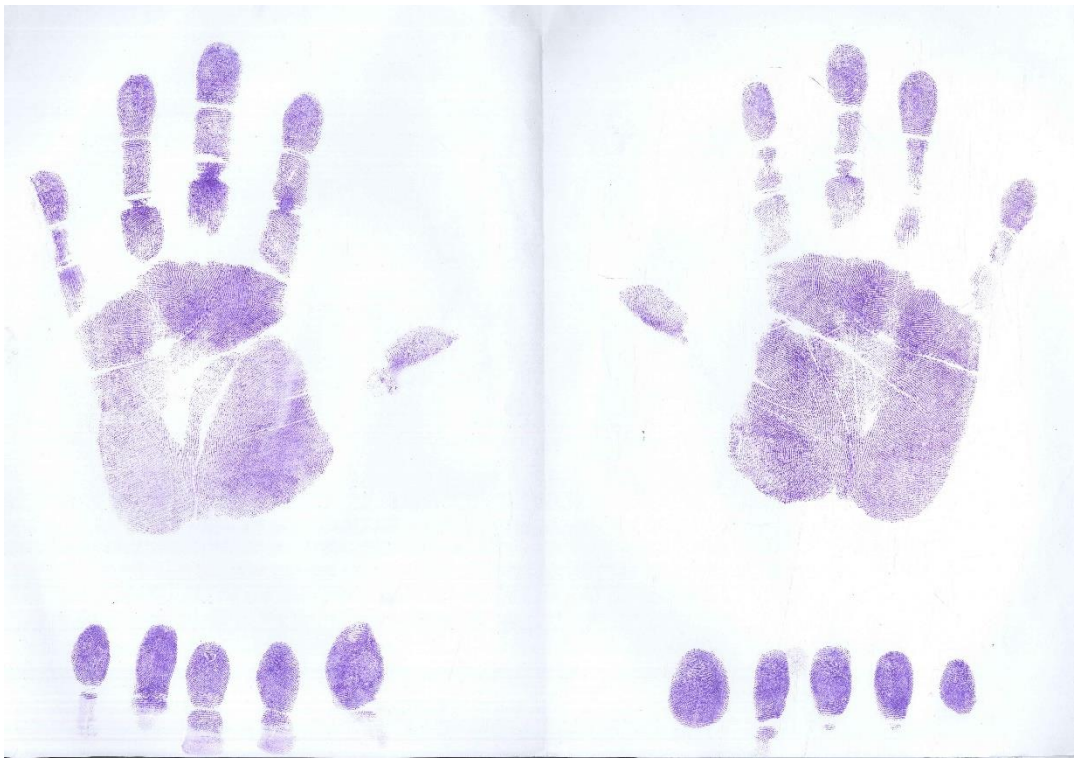


Figure 5: Oral squamous cell carcinoma

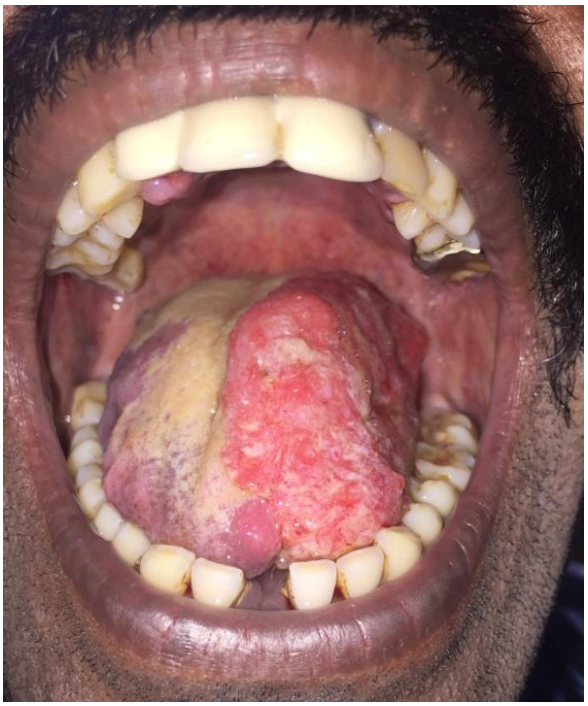


Figure 6: Oral leukoplakia

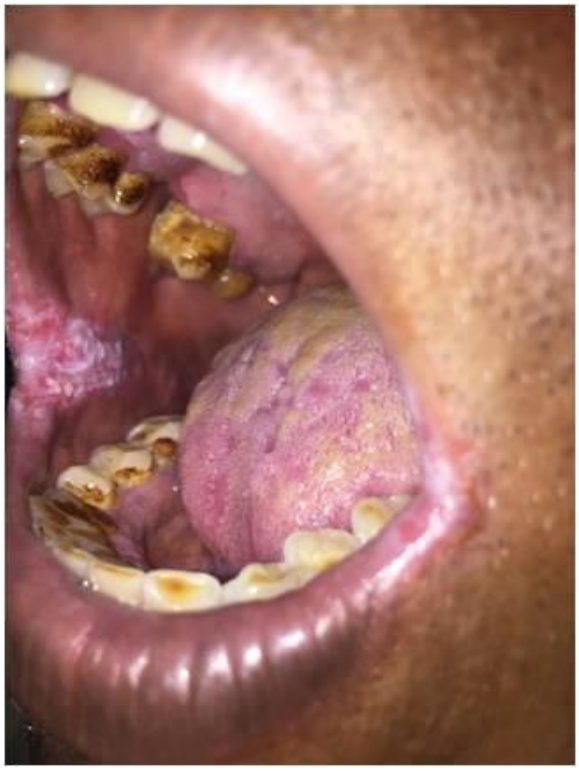
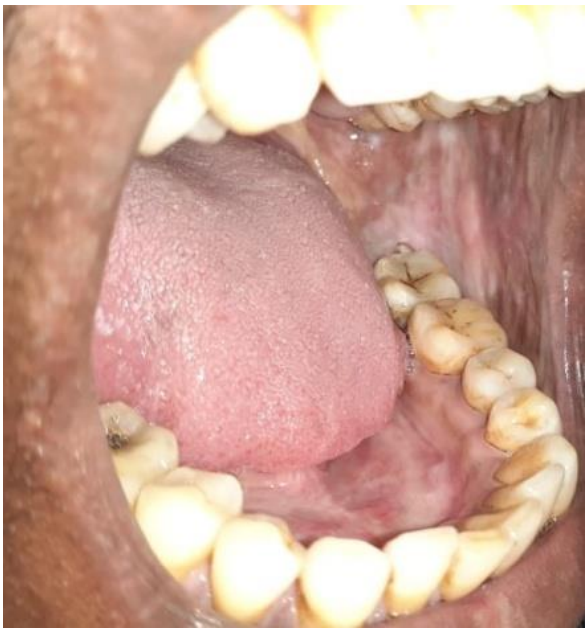
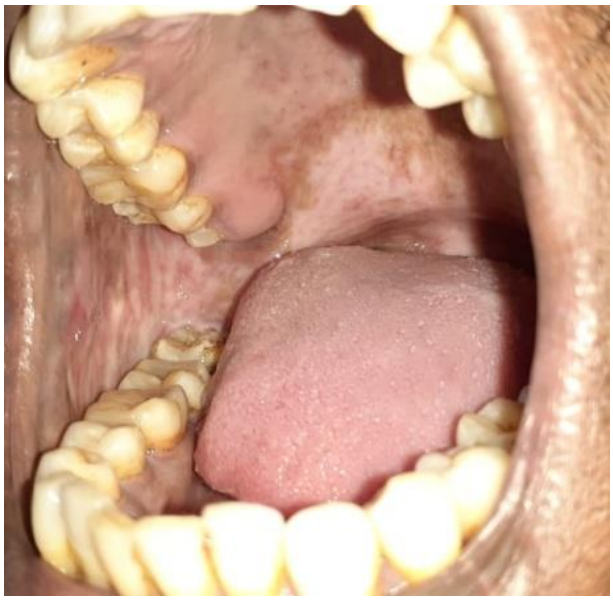


Figure 7: Oral submucous fibrosis





STATISTICAL ANALYSIS

Statistical analysis was done using SPSS version 20 software. Chi-square test was used for qualitative analysis to find the P values. Mean and standard deviations were estimated in the sample for each study group for quantitative analysis. Mean values were compared using one-way ANOVA. When the P valus is < 0.05, it was considered as statistically significant.

RESULTS

The current cross-sectional study was undertaken to compare the dermatoglyphic patterns in subjects of potentially malignant disorders and OSCC with the dermatoglyphic patterns of subjects without lesion.

A total number of 300 patients were included in the study and divided into three groups group A – lesions OSCC, OL and OSF(100) Group B- Partcipants with habits without lesions and group C- Partcipants without habits and lesions (100).The mean age of group A was 47.56, group B was49.26 and group C was 54.32 (Table 1 and chart 1)

In group A 83 were males and 17 were females , in group B 97 were males and 3 were female patient and in group C 92 were males and 8 were female patients (Table 2 and chart 2).

Among group A 60 patients were with Oral Squamous Cell Carcinoma and 28 patients were with Oral Leukoplakia and 32 were with Oral Submucous Fibrosis.

Table 3. shows distribution of different types of finger ridge patterns in Group A, Group B and Group C. Among group A 9.3% have arches, 51.2% have loops, 39.5% have whorls. Among group B 3.4% have arches, 50.9% have loops and 45.7% have whorls. Among group C 0.6% shows arches 48.7% shows loops and 50.7% shows whorls. There was significant increase in frequency of arch pattern in group A compared with group B and group C with the p value of 0.02 shown in chart 3.

Table 4. shows the frequency of distribution of different types of palmar patterns in the thenar/I1, I2,I3,I4 and hypothenar region among different groups. Among group A 26.8% have arches, 43.6% have loops and 29.6% have whorls. Among group 29.31% have arches, 40.94% have loops and 29.74% have whorls. Among group C 27.39% have arches, 44.34% have loops and 28.26% have whorls (chart 4). There was no significant differences in the frequency of distribution of palmar patterns among the three group.($p > 0.05$)

Table 5. shows the mean total finger ridge count(TRFC) of both the right and left hand of the three group. The mean ± standard deviation of group A is 123.88 ± 12.76, for group B it is 121.65 ± 13.55, for group C it is 133.99 ± 8.017. Group A have less total finger ridge compared to Control group C but little compared to group C which was statistically significant with the p value 0.001 shown in chart 5.

Table 6 shows the mean ATD angles of right hand among the three groups. Group A have mean ATD angle of 41.50 ± 4.94, group B have 40.41± 4.19, and group C have 42.15 ± 4.48. Group A have increased ATD angle on the right hand compared to group B but less when compared to group C which was statistically significant with the p value of 0.039 shown in chart 6.

Table 7 shows the mean ATD angle of left hand of the three groups. Group A have mean Atd of 42.62 ± 4.26, group B have 40.180 ± 3.47, and group C have 43.18 ± 4.45. group A have increased ATD angle on the left hand compared to group B but less when compared with group B which was statistically significant with the p value of 0.036 shown in chart 7.

TABLES

Table 1: Age distribution

Groups	Mean	SD
A	47.56	11.46
B	49.28	11.11
C	54.32	9.545

Table 2: Gender distribution of the groups

Group	Gender	N	%
A	Male	83	83.0
	Females	17	17.0
B	Male	97	97.0
	Females	3	3.0
C	Male	92	92.0
	Females	8	8.0

Table 3: Frequency distribution of Finger ridge pattern among three groups

	Group A(%)	Group B (%)	Group C (%)	Chi-square	P value
ARCH	9.3	3.4	0.6*	5.21	0.020
LOOP	51.2	50.9	48.7		
WHORL	39.5	45.7	50.7		

Table 4: Frequency distribution of I1/thenar,I2,I3,I4 and hypothenar pattern among three groups

	Group A(%)	Group B (%)	Group C (%)	Chi-square	P value
ARCHES	26.8	29.31	27.39	2.02	0.42
LOOPS	43.6	40.94	44.34		
WHORLS	29.6	29.74	28.26		

Table 5 : Comparison of Total Finger ridge count between the groups

Variable	Groups	Mean	SD	95% CI for Mean		F	P value
				Lower	Upper		
Total Finger Ridge Count	Group A	123.88	12.766	121.3469	126.4131	31.2	0.001
	Group B	121.65	13.553	118.9608	124.3392		
	Group C	133.99	8.0170	132.3992	135.5808		

Table 6 : Comparison of ATD angle of right hand between the groups (using one-way ANOVA)

Variable	Groups	Mean	SD	95% CI for Mean		F	P value
				Lower	Upper		
ATD Angle Right	Group A	41.500	4.945	40.5188	42.4812	8.27	0.039
	Group B	40.410	4.193	39.6579	41.3221		
	Group C	42.150	4.482	41.2607	43.0393		

Table 7: Comparison of ATD angle of left hand between the groups (using one-way ANOVA)

Variable	Groups	Mean	SD	95% CI for Mean		F	P value
				Lower	Upper		
ATD Angle Left	Group A	42.620	4.261	40.1744	43.8656	6.02	0.036
	Group B	40.180	3.472	39.9910	41.3690		
	Group C	43.180	4.453	42.8964	44.6636		

CHARTS

Chart 1: Age distribution

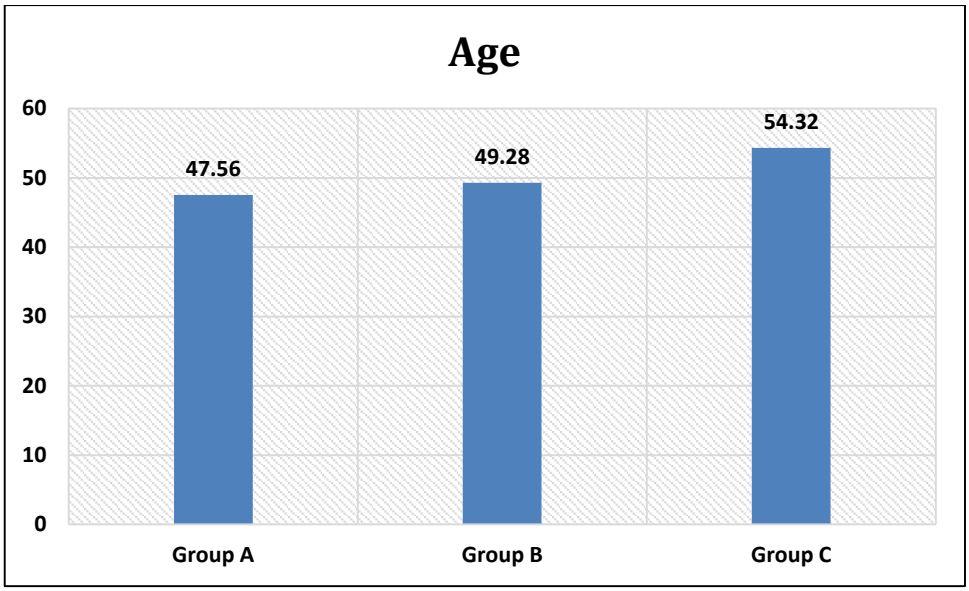


Chart 2: Gender distribution of the groups

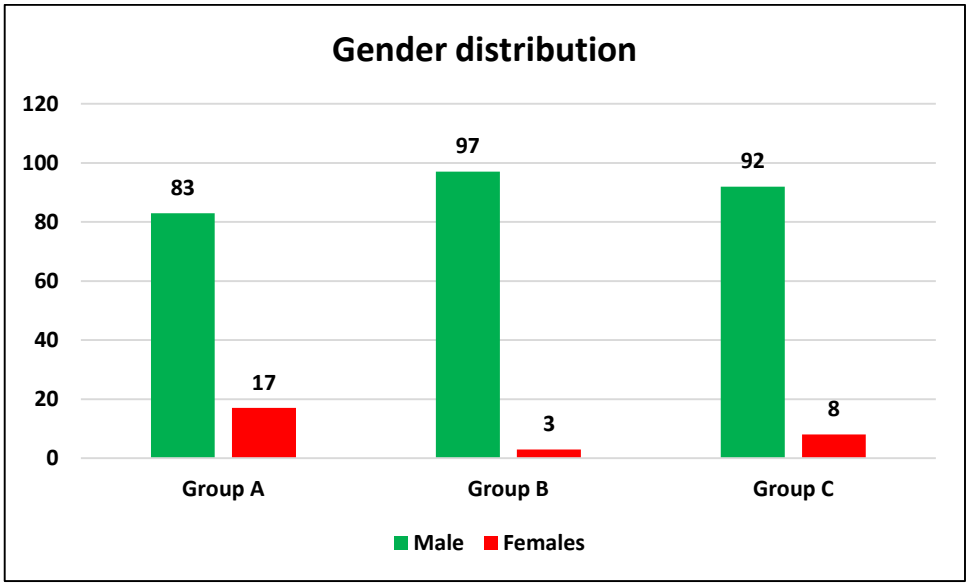


Chart 3: Bar diagram showing the frequency distribution of finger ridge pattern among three groups

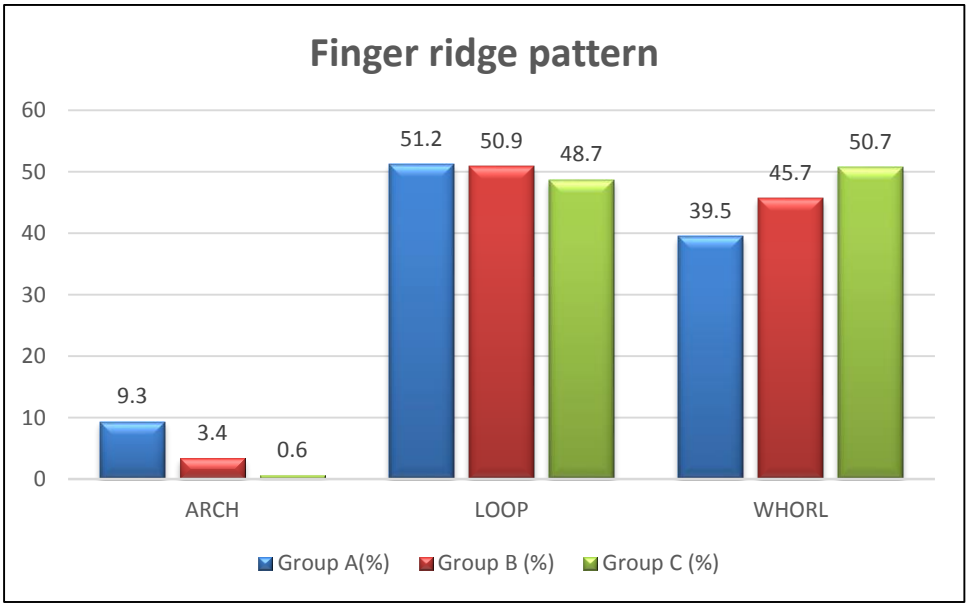


Chart 4: Bar diagram showing the frequency distribution thenar/I1,I2,I3,I4, hypothenar pattern among three groups

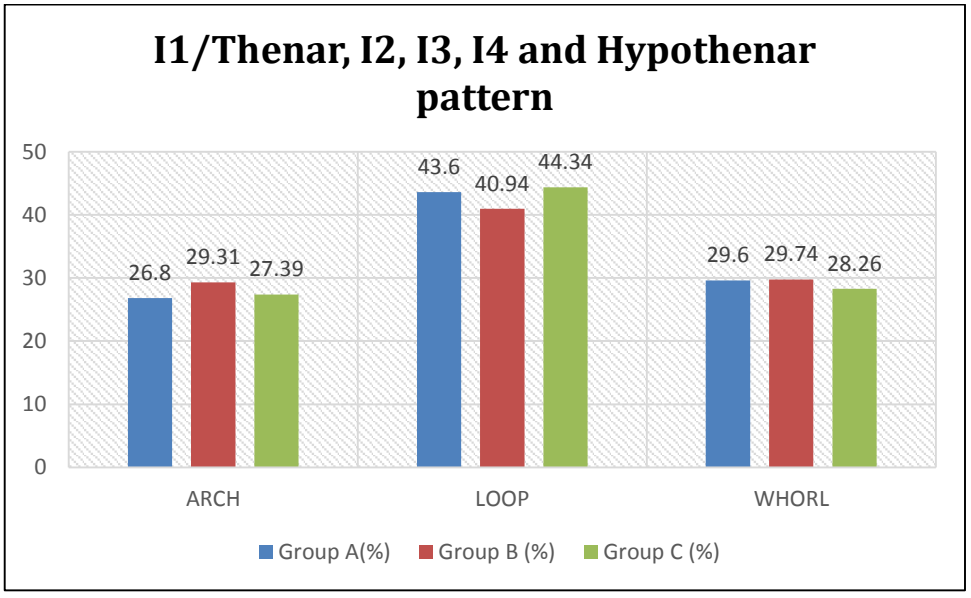


Chart 5: Bar diagram showing the distribution of total finger count between the groups

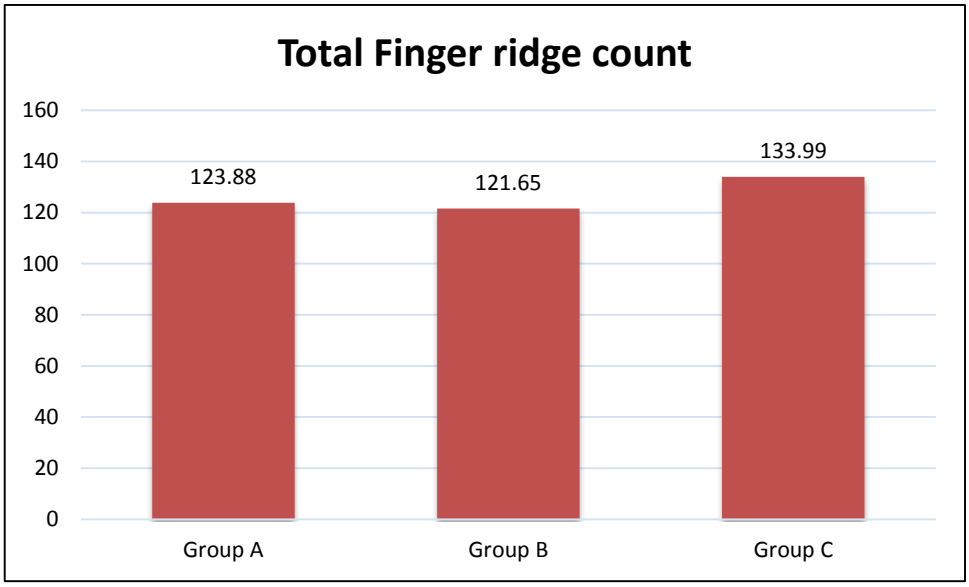


Chart 6: Bar diagram showing the distribution of ATD angle of right hand between the groups

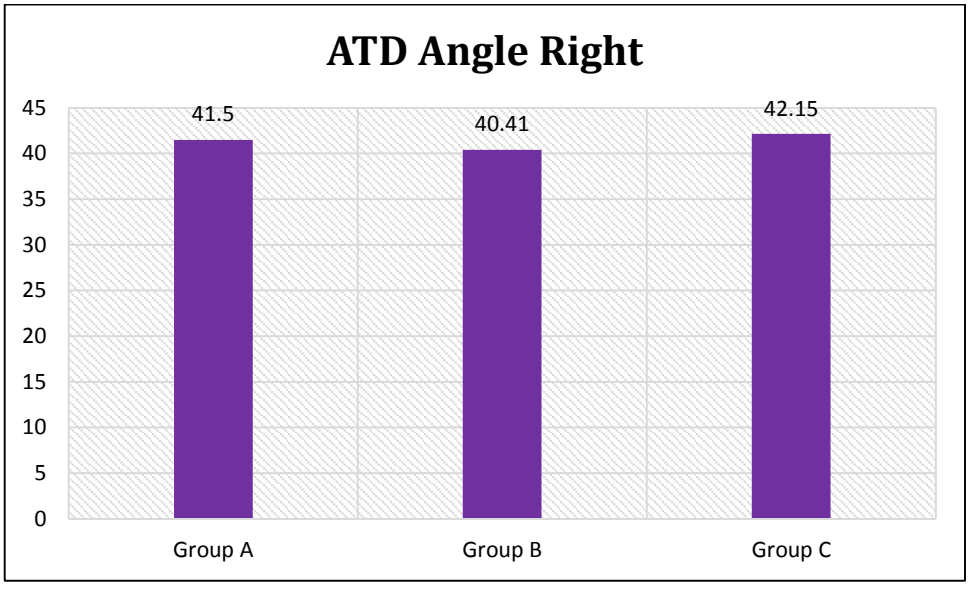
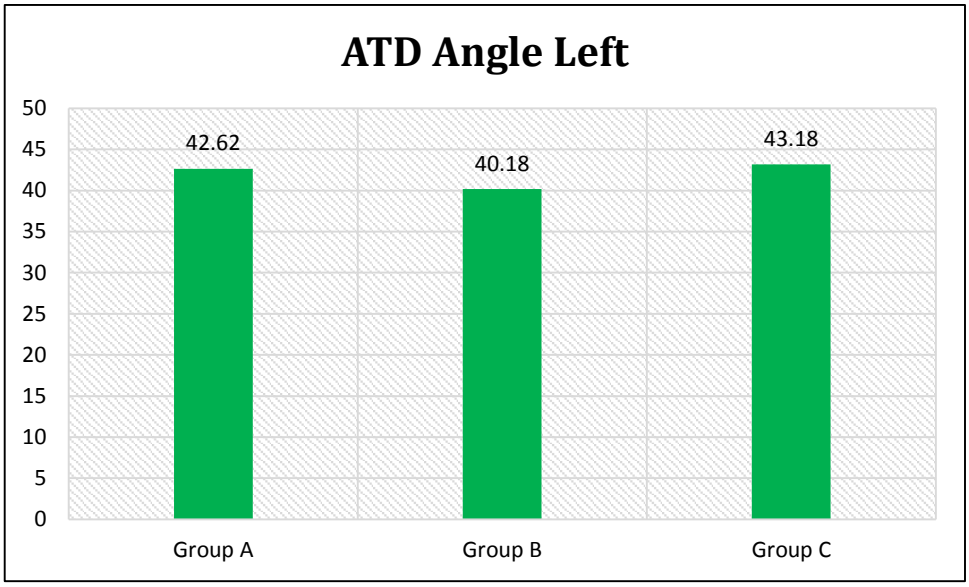


Chart 7: Bar diagram showing the distribution of ATD angle of left hand between the groups



DISCUSSION

OSCC is a widespread disease associated with increased amount of morbidity and mortality rate. It is a major worldwide health problem with rapidly increasing number of sufferer as more and more people embracing deleterious habits such as tobacco chewing, smoking and alcohol abuse. Although the etiology is multifactorial, but regardless of the various accelerating factors, oral cancer is thought to arise clonally from transformed cells which undergone specific genetic and epigenetic alterations in oncogenes or tumor-suppressor genes. The development and progression of SCC and the stages of carcinogenesis is associated with various genetic alteration that have been clearly defined.

An increased risk of oral cancer is associated with various inherited cancer syndromes, including Li-Fraumeni, Fanconi's anaemia and xeroderma pigmentosum. Some studies have suggested that there is an genetic inheritance for sporadic oral cancer. First-degree relatives of people with oral cancer have been reported with the increased risk of developing the disease.

Similarly OL and OSF is a widespread potentially malignant disorder which is more prevalent in South East Asia. Areca nut and tobacco is an important predisposing factor, but not all the patients with long term habits suffer from the disease. Conversely, not all the patients with OSF have a long term history of areca nut or tobacco consumption. It is suggested that genetic susceptibility is responsible for such variations.

The dermal ridges have various important characteristics which make them unique, not only for personal identification, but also in human biology for various

reasons. The dermal ridges and configuration once formed remain unchanged except in dimensions unlike other bodily traits, i.e. they are age stable. The dermal ridges are environmentaly stable and starts to appear from 5th month of embryonic life. Although the patterns formed by ridges vary in size, shape and detailed structures, still they can be classified into definite main types. Thus the dermatoglyphic can be used as "genetic marker" of a disorder.

Various epidemiological studies support the fact that certain genetic alterations may be involved in the pathogenesis of OSCC and OSF. These antenatal disturbances can change the epithelium making it susceptible to various carcinogens. The present study has been done assuming the hypothesis that any such antenatal disturbance, if responsible for a disorder, should manifest in a prenatal event like dermal ridge formation.

Previously, very limited studies were done on the use of dermatoglyphics as a marker for malignant and potentially malignant disorders but in the recent days many studies have been done and published on the use of dermatoglyphics as a marker for OSCC, OSF and OL.

The present study on dermatoglyphic patterns of patients with malignant lesion like OSCC and potentially malignant disorders like OL and OSF revealed some significant parameters which can be used as a "dermatoglyphic markers". Based on the comparison between the groups the following positive parameters were observed in patients with malignant and potentially malignant lesion - Increase in frequency of arch and loop patterns on fingertips, with decrease in frequency of simple whorl patterns on fingertips, decreased total finger ridge count and ATD angle on both right and left hand

The results of frequency distribution of arch pattern in different groups were compared with various studies.

In our study with total population of 300 (100 on each category) the frequency of arch pattern observed in group A patients with OSCC, OSF, and OL is 9.3%, in group B it is 3.4% and group C it is 0.6% .

In a study done by **Elluru Venkatesh et al**[1] with total population of 90 (30 on each group), the frequency distribution of arch pattern in Oral leukoplakia is 19 (06.30%), OSCC is 21 (07.00%) and in Control group is 06 (02.00%).

Veena kulkarni et al[43] in their study with total number of population 150 (50 on each group) showed that the arch frequency in OSF Cases was 029 (05.8%), Control Group With Gutkha was 022(04.40%) and in Control Group Without Gutkha was 07 (01.40%). The result of this study is in accordance with our study.

In our study all the groups have increased frequency of loops in the palmar region with frequency of 43.6%, which was in accordance with the study conducted by **Lakshmanan et al**[3] where the predominant patterns observed in thenar areas of both hands in all the three groups were found to be loops. The predominant pattern observed in I2, I3 and I4 areas of both hands in all the three groups were the loops.

In this study the total finger ridge count was less in patients with malignant and potentially malignant disorder compared to the control group, in group A TFRC is 123.88, for group B it is 121.65, for group C it is 133.99, which was in accordance with the study done by **Lakshmanan et al**.[3] In their study total finger ridge in Group 1 patients with OSCC, the mean was 141.19 as against group 2 patients with

OL and OSF a mean of 144.81 and 152.04 for Group 3 patients with no lesion. In both the studies the study group with lesions have less TRFC compared with the control group.

The results of the study conducted by **Ambika Gupta et al[45]** shows mean ATD angle of OSCC group was 42.07 and OSF group was 39.50 on right hand and on left hand OSCC group and OSF group showed 41.90 and 40.70 respectively, which is in accordance with the present study result with the mean ATD angle of OSCC,OL,OSF group on the right hand was 41.50 and on the left hand it was 42.62 which is compared to the control group. It was almost equal to the previous study result.

Athreya vijayaraghavan et al[44] in their study said that, the mean ATD angle in the control group was 45.70° with a standard deviation of ±1.35, and in the subjects with habit without lesion it was 44.90° with a standard deviation of ±3.56. However, on comparison with the controls, the ATD angle in patients with OSF and oral cancer it was less than 45°. In OSF patients, it was 41.88° with a standard deviation of ±2.21 and in oral cancer patients it was 40.95° with a standard deviation of ±2.46.º which is in concordance with our study result where ATD angle of patients with malignant and potentially malignant disorder was also less than 45º.

SUMMARY

Though various epidemiologic studies suggest that the use of tobacco (chewing or non-chewing) is an important risk factor for the development of malignant and potentially malignant disorder of the oral cavity, not all individuals develop the same. It seems genetic predisposition could be an underlying mechanism.

Hence the assessment of genetic abnormalities also plays an essential role with the diagnostic procedure of PMD (Potentially malignant disorder) and OSCC. It can be determined by various genetic studies. As such these procedures are complex and expensive, dermatoglyphics can be efficiently employed with other clinical signs as a screening procedure as a noninvasive, simple, and inexpensive procedure for the assessment of genetic susceptibility.

We have done this study on patients with oral cancer, oral leukoplakia and oral submucous fibrosis in the Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital.

Total population included in the study was 300. They were categorized into three groups, as mentioned below:

Group A: It includes100 patients with oral leukoplakia (OL), oral submucous fibrosis (OSF), and oral squamous cell carcinoma (OSCC).

Group B: It includes 100 patients with habits (such as tobacco smoking or chewing and consumption of alcohol) and without lesions.

Group C: it includes 100 healthy patients without any habits.

A detailed history of clinically diagnosed cases of OSF, OL, and OSCC was taken and recorded in a case history proforma. Incisional biopsy was done. After histopathologic confirmation they were included in the study. The finger and palm prints of patients with history of tobacco-related habits (smoking/smokeless), pan masala, betel nut chewing (Group A and B), and normal patients of similar age group and sex without any tobacco-related habits (Group C) were taken for the study.

The finger and palm prints were obtained by the Ink method one of the most common method, which was described by Cummins and Midlo in 1943. Patients were asked to wash the hands thoroughly washed with soap before taking prints to remove oil or any dirt. Then the ink was applied on the palms and it was spread evenly on the palms and fingers. Then the patient was asked to keep the finger on white paper and firm pressure was applied on the center of the dorsum of hand, and inter digital areas. The finger prints were recorded by placing it on a white paper with one lateral aspect of the finger and then it is rolled it towards the opposite side. It is then analysed with the use of magnifying lens. The dermatoglyphics patterns were analysed both qualitatively and quantitatively.

The result of our study showed an increase in frequency of arch pattern in group A, when compared to group B and group C and decrease in frequency of whorl pattern in group A, when compared to group B and group C which was statistically significant. Increase in frequency of loops in the palmar region in all the three groups but it was statistically insignificant. The mean total finger ridge count and ATD angle was less in group A which was statistically significant.

CONCLUSION

The dermatoglyphics study is not only useful for diagnosis but also for prevention by predicting a disease. It is not only for defining an existing disease but also for the identification of people with a genetic predisposition to develop the disease.

The main limitation of the study, is that the dermatoglyphic patterns cannot be recorded if the palms are malformed or any disease affecting the pattern.

The present study on dermatoglyphic patterns of patients with malignant lesion like OSCC and potentially malignant disorders like OL and OSF revealed some significant parameters which can be used as a "dermatoglyphic markers". Based on the comparison between the groups the following positive parameters were observed in patients with malignant and potentially malignant lesions.

1) Increase in frequency of arch and loop patterns on fingertips, with decrease in frequency of simple whorl patterns on fingertips
2) Decreased total finger ridge count and
3) Decreased ATD angle on both right and left hand
4) Whorls were the more predominant pattern in the normal/ control group.

BIBLIOGRAPHY

1. Venkatesh E. *Palmar dermatoglyphics in oral leukoplanka and oral squamous cell carcinoma patients* (Doctoral dissertation, RGUHS).
2. Namouchi I. Anthropological significance of dermatoglyphic trait variation: an intra-Tunisian population analysis. International Journal of Modern Anthropology. 2011;1(4):12-27.
3. Lakshmana N, Ravikiran A, Samatha Y, Nayya AS, Vamsi PB, Kartheeki B. Role of digital and palmar dermatoglyphics in early detection of oral leukoplakia, oral submucous fibrosis and oral squamous cell carcinoma patients. Advances in Human Biology. 2016 Sep 1;6(3):136.
4. Masthan KM, Babu NA, Sankari SL, Priyadharsini C. Leukoplakia: A short review on malignant potential. Journal of pharmacy & bioallied sciences. 2015 Apr;7(Suppl 1):S165.
5. Shih YH, Wang TH, Shieh TM, Tseng YH. Oral Submucous Fibrosis: A Review on Etiopathogenesis, Diagnosis, and Therapy. International journal of molecular sciences. 2019 Jan;20(12):2940.
6. Lakshmana N, Nayyar AS, Ravikiran A, Samatha Y, Pavani VB, Kartheeki B. Dermatoglyphics: Revival in oral pre-cancers and cancers, a review. CHRISMED Journal of Health and Research. 2017 Jan 1;4(1):1.
7. Warnakulasuriya S, Johnson NW, Van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. Journal of oral pathology & medicine. 2007 Nov;36(10):575-80.
8. Pindborg JJ, Sirsat SM. Oral submucous fibrosis. Oral Surgery, Oral Medicine, Oral Pathology. 1966 Dec 1;22(6):764-79.

9. Ranganathan K, Devi MU, Joshua E, Kirankumar K, Saraswathi TR. Oral submucous fibrosis: a case-control study in Chennai, South India. J. Oral Pathol. Med.2004;33(5):274-7
10. Rajendran R. Oral submucous fibrosis: etiology, pathogenesis, and future research. Bulletin of the World Health Organization. 1994;72(6):985.
11. Oral submucous fibrosis – cause, symptoms, diagnosis, treatment http://mobilephysiotherapyclinic.in/oral-sub-mucous-fibrosis/
12. Hu Y, Jian X, Peng J, Jiang X, Li N, Zhou S. Gene expression profiling of oral submucous fibrosis using oligonucleotide microarray. Oncol Rep. 2008;20(2):287–294.
13. Khan I, Kumar N, Pant I, Narra S, Kondaiah P. Activation of TGF-β pathway by areca nut constituents: a possible cause of oral submucous fibrosis. PLoS One. 2012;7(12):e51806.
14. Wollina U, Verma S, Parikh D, Parikh A. Oral and extraoral disease due to betel nut chewing. Hautarzt. 2002;53(12):795–797. German.
15. More CB, Gupta S, Joshi J, Varma SN. Classification system for oral submucous fibrosis. Journal of Indian Academy of Oral Medicine and Radiology. 2012;24(1):24.
16. Wollina U, Verma SB, Ali FM, Patil K. Oral submucous fibrosis: an update. Clinical, cosmetic and investigational dermatology. 2015;8:193.
17. Axéll T, Pindborg JJ, Smith CJ, Van der Waal I, an International Collaborative Group on Oral White Lesions. Oral white lesions with special reference to precancerous and tobacco-related lesions: conclusions of an international symposium held in Uppsala, Sweden, May 18–21 1994. Journal of oral pathology & medicine. 1996 Feb;25(2):49-54.

18. Warnakulasuriya S, Johnson NW, Van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. Journal of oral pathology & medicine. 2007 Nov;36(10):575-80.
19. Petti S. Pooled estimate of world leukoplakia prevalence: a systematic review. Oral oncology. 2003 Dec 1;39(8):770-80.
20. Feller L, Lemmer J. Oral leukoplakia as it relates to HPV infection: a review. International journal of dentistry. 2012;2012.
21. Waldron CA, Shafer WG. Leukoplakia revisited. A clinicopathologic study 3256 oral leukoplakias. Cancer. 1975 Oct;36(4):1386-92.
22. Brouns ER, Baart JA, Karagozoglu KH, Aartman IH, Bloemena E, Van der Waal I. Malignant transformation of oral leukoplakia in a well-defined cohort of 144 patients. Oral diseases. 2014 Apr;20(3):e19-24.
23. Hansen LS, Olson JA, Silverman Jr S. Proliferative verrucous leukoplakia: A long-term study of thirty patients. Oral surgery, oral medicine, oral pathology. 1985 Sep 1;60(3):285-98.
24. Parlatescu I, Gheorghe C, Coculescu E, Tovaru S. Oral leukoplakia–An update. Maedica. 2014 Mar;9(1):88.
25. Ramaswamy G, Rao VR, Kumaraswamy SV, Anantha N. Serum vitamins' status in oral leucoplakias—a preliminary study. European Journal of Cancer Part B: Oral Oncology. 1996 Mar 1;32(2):120-2.
26. Van der Waal I, Schepman KP, Van der Meij EH. A modified classification and staging system for oral leukoplakia. Oral oncology. 2000 May 1;36(3):264-6.

27. Deliverska EG, Petkova M. Management of oral leukoplakia-analysis of the literature. Journal of IMAB–Annual Proceeding Scientific Papers. 2017 Mar 26;23(1):1495-504.
28. Shah JP, Gil Z. Current concepts in management of oral cancer–surgery. Oral oncology. 2009 Apr 1;45(4-5):394-401.
29. Rivera C. Essentials of oral cancer. International journal of clinical and experimental pathology. 2015;8(9):11884.
30. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral oncology. 2009 Apr 1;45(4-5):309-16.
31. Kumar M, Nanavati R, Modi TG, Dobariya C. Oral cancer: Etiology and risk factors: A review. Journal of cancer research and therapeutics. 2016 Apr 1;12(2):458. Williams HK. Molecular pathogenesis of oral squamous carcinoma. Molecular Pathology. 2000 Aug;53(4):165.
32. Ke LD, Adler-Storthz K, Mitchell MF, Clayman GL, Chen Z. Expression of human papillomavirus E7 mRNA in human oral and cervical neoplasia and cell lines. Oral oncology. 1999 Jul 1;35(4):415-20.
33. Todd R, Donoff RB, Wong DT. The molecular biology of oral carcinogenesis: toward a tumor progression model. Journal of oral and maxillofacial surgery. 1997 Jun 1;55(6):613-23.
34. Hollstein M, Sidransky D, Vogelstein B, Harris CC. p53 mutations in human cancers. Science. 1991 Jul 5;253(5015):49-53.
35. Pavani NP, Srinivas P, Kothia NR, Chandu VC. Recent advances in the early diagnosis of oral cancer: A systematic review. International Journal of Medical Reviews. 2017 Dec 25;4(4):119-25.

36. Prabha LJ, Thenmozhi R. A short review on dermatoglyphics. Journal of Pharmaceutical Sciences and Research. 2014 Apr 1;6(4):200.
37. Sinha CK, Meel M, Bayan B. Using dermatoglyphics pattern to identify the left handed unique pattern and its biological significance-if any. World Applied Sciences Journal. 2012;20(8):1107-13.
38. Dermatoglyphic: Finger Pattern Types (Henry Classification), Total Ridge Count http://eacharya.inflibnet.ac.in/data-server/eacharya-
39. Preus M, Fraser FC. Dermatoglyphics and syndromes. American Journal of Diseases of Children. 1972 Dec 1;124(6):933-43.
40. Abhilash PR, Divyashree R, Patil SG, Gupta M, Chandrasekar T, Karthikeyan R. Dermatoglyphics in patients with dental caries: A study on 1250 individuals. J Contemp Dent Pract. 2012 May 1;13(3):266-74.
41. Ahmed RH, Aref MI, Hassan RM, Mohammed NR. Dermatoglyphic study on patients with dental caries who wearing dental fillings and its correlation to apoptosis that induced by using dental fillings. Nat Sci. 2010;8:54-7.
42. Mathew L, Hegde AM, Rai K. Dermatoglyphic peculiarities in children with oral clefts. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2005 Oct 1;23(4):179.
43. Kulkarni V. Palmar dermatoglyphics among gutkha chewers with and without oral submucous fibrosis. UPDATE. 2014;5:1.
44. Vijayaraghavan A, Aswath N. Qualitative and quantitative analysis of palmar dermatoglyphics among smokeless tobacco users. Indian Journal of Dental Research. 2015 Sep 1;26(5):483.

45. Gupta A, Karjodkar FR. Role of dermatoglyphics as an indicator of precancerous and cancerous lesions of the oral cavity. Contemporary clinical dentistry. 2013 Oct;4(4):448.
46. Vk C, Ss R, Ar S. Qualitative dermatoglyphic analysis of finger tip patterns in patients of oral sub mucous fibrosis. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS) e-ISSN. 2013:2279-0853.

APPENDIX

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AFFLIATED TO THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY,CHENNAI

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The Investigator / Investigating team is advised to adhere to the guide lines given below:

- Should be carried out under the direct supervision of the Guide
- Get detailed informed consent from the patients / participants and maintain confidentiality.
- Carry out the work without affecting regular work and without extra expenditure to the Institution or the Government.
- Inform the IRB in case of any change of study procedure, site, Investigator and Guide.
- Not deviate from the area of work for which applied for clearance.
- Inform the IRB immediately in case of any adverse events or serious adverse reactions. Should abide to the rules and regulations of the institution(s) .
- Complete the work within specific period and if any extension of time is required, should apply for permission again to do the work.
- Submit the summary of the work to the IRB : Students-every 3 months;
  Faculty-every 6 months.
- Should not claim any kind of funds from the institution for doing the work or on completion/ or for any kind of compensations.
- The members of the IRB have the right to monitor the work without prior intimation.
- The investigator and Guide should each declare that no plagiarism is involved in this whole study and enclose the undertaking in dissertation/ thesis.

PATIENT INFORMATION SHEET

I**, Dr.N.Thilagavathi,** I – MDS student, Department of Oral Medicine and Radiology, primary investigator under the guidance of Prof.Dr.G.V.Murali Gopika Manoharan, MDS, Professor, Department of Oral Medicine and Radiology, Tamilnadu Government Dental College and Hospital, have planned to conduct a study titled **"Role of dermatoglyphics in malignant and potentially malignant disorders of the oral cavity: A cross-sectional study"** in Tamilnadu Government Dental College and Hospital, Chennai-3.

Purpose of the study

To evaluate the genetic predisposition of malignant and potentially malignant disorder

Procedures. The dermatoglyphics of these three groups is compared

Group A: 100 patients with oral leukoplakia (OL), oral submucous fibrosis (OSMF), oral lichen planus(OLP) and oral squamous cell carcinoma (OSCC).

Group B: 100 healthy patients with habits (such as tobacco smoking or chewing and consumption of alcohol) and no lesions.

Group C: 100 healthy patients without habits.

Benefits of participation

As PMDs and OSCC have a genetic basis, with the knowledge of dermatoglyphic patterns, individuals who are prone to develop these lesions can avoid the trigger factors.

Participant's rights

Taking part in this study is voluntary. Patients are free to decide whether to participate in the study or to withdraw at any time; patient's decision will not result in any loss of benefits to which you are otherwise entitled. The results of this special study may be intimated to patient at the end of the study period.

Risk of participation

Nil

Confidentiality

The identity of the patients participating in the research will be kept confidential throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Compensation

Nil

Contacts for queries related to the study,

Dr.N.Thilagavathi
I year PG student,
Department of Oral Medicine and Radiology,
Tamilnadu Government Dental College and Hospital,
Chennai - 600003.
Phone no: 9944414880

Queries related to patients Rights,

Dr.B. Saravanan. M.D.S, Ph.D
The chairman, institutional ethical committee,
Tamilnadu Government Dental College and Hospital,
Chennai - 600003

Name of the Patient **Signature / Thumb impression**

Name of the investigator **Signature**

Date

ஆராய்ச்சி பற்றிய தகவல் படிவம்

ஆராய்ச்சி தலைப்பு

திலகவதி.ந ஆகிய நான் மரு.க.வெ.முரளி கோபிகா மனோகரன், MDS அவர்களின் வழிநடத்துதலின் கீழ் வாய் புற்றுநோய் மற்றும் வாய் புற்றுநோயின் முன்னோடி நோய்களில் ரேகையியலின் பங்களிப்பு பற்றிய ஆய்வு.

ஆய்வின் நோக்கம்

இந்த ஆய்வின் நோக்கமானது, வாய் புற்றுநோய் மற்றும் வாய் புற்றுநோயின் முன்னோடி நோய்களை மரபணு மூலம் முன்கணிப்பது.

செய்முறை

இந்த மூன்று குழுக்களின் ரேகைகள் ஒப்பிடப்படுகின்றன.

குழு 1

வாய் புற்றுநோய் மற்றும் வாய்புற்றுநோய் முன்னோடி நோய்கள் உள்ள 100 நபர்கள்

குழு 2

புகையிலை பழக்கம் உடைய, வாய் புற்றுநோய் இல்லாத 100 நபர்கள்

குழு 3

புகையிலை பழக்கம் மற்றும் வாய் புற்றுநோய் இல்லாத 100 நபர்கள்

நன்மைகள்

வாய் புற்றுநோய் மற்றும் வாய் புற்றுநோயின்முன்னோடி நோய்களை மரபணு அடிப்படையில் ரேகையியல் மூலம் கண்டறிவதால் பாதிப்புக்குள்ளாகும் நபர்கள் அதன் தூண்டுதல் காரணியை புறக்கணிக்கலாம்.

இரகசிய தன்மை

நோயாளிகள் பற்றிய குறிப்புகள் ஆராய்ச்சியை வெளியிடும் போது நோயாளிகளின் தனிப்பட்ட விவரங்கள் எதுவும் பாதிக்கப்படமாட்டாது.

பங்குபெறுவோரின் உரிமை

இந்த ஆராய்ச்சியில் பங்கு பெறுவது நோயாளிகளின் தனிப்பட்ட விருப்பம். மேலும் நோயாளிகள் இந்த ஆய்வில் இருந்து எப்போது வேண்டுமானாலும் விலகிக் கொள்ளலாம். நோயாளிகளின் இந்த முடிவினால் அவருக்கோ அல்லது ஆராய்ச்சியாளருக்கோ எந்தவித பாதிப்பும் கிடையாது. இந்த ஆராய்ச்சியின் முடிவுகள் நோயாளிகளுக்கு ஆராய்ச்சியின் இடையிலோ அல்லது முடிவிலோ தெரிவிக்கப்படும். இதில் ஏதேனும் பின் விளைவுகள் ஏற்பட்டால் அதை சரிசெய்ய சிகிச்சை அளிக்க தகுந்த உதவிகள் செய்யப்படும்.

இழப்பீடு

ஏதுமில்லை

ஆய்வு பற்றிய தகவல் பெற

திலகவதி.ந,
முதலாம் ஆண்டு MDS, முதுநிலை மாணவி,
வாய் நோய் அறிதல் மற்றும் ஊடுகதிர் துறை,
தமிழ்நாடு பல் மருத்துவக் கல்லூரி மற்றும் மருத்துவமனை,
சென்னை-600 003.
தொலைபேசி: 8122659246

.....	.....
நேயாளியின் பெயர்	கையொப்பம்/கைரேகை
.....	.....
ஆராய்ச்சியாளர் பெயர்	கையொப்பம்

தேதி

DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY

INFORMED CONSENT FORM

STUDY TITLE:

"Role of dermatoglyphics in malignant and potentially malignant disorders of the oral cavity: a cross-sectional study"

Name: O.P. No:

Address:

Serial No:

Tel. no: Age / Sex :

I,_____ age_____years

Exercising my free power of choice, hereby give my consent to be included as a participant in the study "Role of dermatoglyphics in malignant and potentially malignant disorders of the oral cavity: a cross-sectional study" I agree to the following:

- I have been informed to my satisfaction about the purpose of the study and study procedures including investigations to monitor and safeguard my body function.
- I agree to give my full participation of the study.
- I agree to cooperate fully and to inform my doctor immediately if I suffer any unusual symptom.
- I agree to report to the doctor for a regular follow–up as and when required for the research.
- I hereby give permission to use my medical records for research purpose. I am told that the investigating doctor and institution will keep my identity confidential.

Name of the patient Signature / Thumb impression

Name of the investigator Signature

Date

ஒப்புதல் படிவம்

ஆராயச்சியின் தலைப்பு

வாய் புற்றுநோய் மற்றும் வாய் புற்றுநோயின் முன்னோடி நோய்களில் ரேகையியலின் பங்களிப்பு பற்றிய ஆய்வு.

பெயர்: புறநோயாளி எண்:

வயது/பால்: ஆராய்ச்சி சேர்க்கை எண்:

முகவரி

தொலைபேசி:

நான்..... வயது ..... என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக் கொள்ள ஒப்புதல் அளிக்கிறேன்.

கீழ்காணப்படும் நிபந்தனைகளுக்குட்பட்டு நான் சம்மதிக்கிறேன்.

- நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் செயல்முறைகள் பற்றி முழுமையாக தெரிவிக்கப்பட்டுள்ளேன்.
- என் உடல்நலம் பாதிக்கப்பட்டாலோ அல்லத எதிர்பாராத வழக்கத்திற்கு மாறான நோய் அறிகுறிகள் தென்பட்டாலோ அதனை விலக்குவதற்கும் முழு உரிமை உள்ளதாக அறிகிறேன்.
- நான் ஏற்கனவே உட்கொண்ட மற்றும் உட்கொள்கின்ற மருந்துகளின் விவரங்களை ஆராய்ச்சியாளரிடம் தெரிவித்துள்ளேன்.
- என் மருத்துவ குறிப்பேடுகள் இந்த ஆராய்ச்சியில் பயன்படுத்திக் கொள்ள சம்மதிக்கிறேன்.
- இந்த ஆராய்ச்சி மையமும் ஆராய்ச்சியாளரும் என்னடைய விவரங்கள் அனைத்தையும் இரகசியமாக வைப்பதாக அறிகிறேன்.

.....	.....	.....
நேயாளியின் பெயர்	கையொப்பம்	தேதி
.....	.....	.....
ஆராய்ச்சியாளர் பெயர்	கையொப்பம்	தேதி

CASE PROFORMA

DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY

TAMIL NADU GOVERNMENT DENTAL COLLEGE AND HOSPITAL, CHENNAI-3

Date: Serial No:

Name: O.P. No:

Age/Sex:

Address:

Phone No:

Occupation: Income:

Religion:

Centre: Department of Oral Medicine & Radiology

Tamilnadu Govt. Dental College & Hospital, Chennai – 600003.

Presenting complaint with duration:

Past Medical History:

Past Surgical History:

Past Dental History: Personal History:

A) Diet:

B) Tooth cleansing habits:

- Cleaning aids used:
- Frequency:

C) Smoking habit:

- Materials used:
- Frequency:
- Duration of the habit:

D) Chewing habit:

- Materials used:
- Frequency:
- Duration of the habit:

E) Other habits (alcohol, snuff):

Marital Status:

Menstrual History:

Family History:

CLINICAL EXAMINATION

Extraoral examination:

Facial symmetry:

Temparomandibular joint

INTRA-ORAL EXAMINATION:

Mouth opening (Interincisal distance):

Size and shape of the mouth:

Jaw movements:

- Teeth:
- Gingiva:
- Alveolar mucosa:
- Labial and Buccal mucosa:
- Hard palate
- Soft palate
- Pillar of fauces and Tonsils
- Tongue
- Floor of the mouth
- Retromolartrigone

Provisional Diagnosis

Investigations:

1) Laboratory investigations:

A) Blood:

RBC count:

Total WBC count:

Differential count:

Haemoglobin%:

Peripheral smear:

Erythrocyte sedimentation rate:

Bleeding time:

Clotting time:

B) Urine:

Glucose:

Clinical Diagnosis:

TRIPARTITE AGREEMENT

This agreement herein after the "Agreement" is entered into on this day……. between the Tamil Nadu Government Dental College and Hospital represented by its **Principal** having address at Tamil Nadu Government Dental College and Hospital, Chennai- 600 003, (hereinafter referred to as, 'the college') And **Prof. Dr.G.V.MURALI GOPIKA MANOHARAN MDS.,** aged 52 years working as **Professor** in Department of Oral medicine and Radiology at the college, (herein after referred to as the 'Principal Investigator') And **Dr.N.Thilagavathi,** aged 26 years currently studying as final year **Postgraduate student** in the Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital, Chennai -3 (hereafter referred to as the 'PG and co- investigator') Whereas the PG student as part of his curriculum undertakes to research on for which purpose the Principal investigator shall act as Principal investigator and the College shall provide the requisite infrastructure based on availability and also provide facility to the PG student as to the extent possible as a Co-investigator. Whereas the parties, by this agreement have mutually agreed to the various issues including in particular the copyright and confidentiality issues that arise in this regard

Now this agreement witnessed as follows:

1. The parties agree that all the Research material and ownership therein shall become the vested right of the college, including in particular all the copyright in the literature including the study, research and all other related papers.
2. To the extent that the college has legal right to do go, shall grant to licence or assign the copyright so vested with it for medical and/or commercial usage of interested

persons/entities subject to a reasonable terms/conditions including royalty as deemed by the college. The Royalty so received by the college shall be shared equally by all the three parties.

3. The PG/Research student and PG/Principal Investigator shall under no circumstances deal with the copyright, Confidential information and know- how- generated during the course of research/study in any manner whatsoever, while shall sole west with the college.
4. The PG student and Principal Investigator undertake not to divulge (or) cause to be divulged any of the confidential information or, know-how to anyone in any manner whatsoever and for any purpose without the express written consent of the college.
5. All expenses pertaining to the research shall be decided upon by the principal investigator/Co-investigator or borne sole by the PG student.(co-investigator)
6. The college shall provide all infrastructure and access facilities within and in other institutes to the extent possible. This includes patient interactions, introductory letters, recommendation letters and such other acts required in this regard.
7. The Principal Investigator shall suitably guide the Student Research right from selection of the Research Topic and Area till its completion. However the selection and conduct of research, topic and area research by the Student Researcher under guidance from the Principal Investigator shall be subject to the prior approval, recommendations and comments of the Ethical Committee of the College constituted for this purpose.
8. It is agreed that as regards other aspects not covered under this agreement, but which pertain to the research undertaken by the PG student, under guidance from the Principal Investigator, the decision of the College shall be binding and final.

9. If any dispute arises as to the matters related or connected to this agreement herein, it shall be referred to arbitration in accordance with the provisions of the Arbitration and Conciliation Act, 1996.
10. In witness where of the parties herein above mentioned have on this the day month and year herein above mentioned set their hands to this agreement in the presence of the following two witnesses.

College represented by its

Principal

PG Student

Student Guide

Witnesses

1.

2.

MASTER CHART

GROUP A – Patients with oral leukoplakia, oral submucous fibrosis and oral squamous cell carcinoma

PATIENT	AGE	GENDER	LESION	FINGER RIDGE PATTERN			I1/THENAR,I2,I3,I4 AND HYPOTHENAR			TOTAL FINGER RIDGE COUNT	ATD ANGLE	
				ARCHES	LOOPS	WHORLS	ARCHES	LOOPS	WHORLS		RIGHT	LEFT
1	63	FEMALE	OSCC	1	9	0	0	2	0	123	58	45
2	55	FEMALE	OSCC	1	6	3	2	2	0	140	40	36
3	29	MALE	OSF	0	9	1	2	0	0	120	48	47
4	60	MALE	OL	0	10	0	0	2	0	130	40	47
5	30	MALE	OSCC	0	8	2	0	1	1	128	44	45
6	45	MALE	OSCC	0	8	2	2	2	0	134	49	38
7	31	MALE	OSCC	1	8	1	0	2	0	139	42	46
8	40	MALE	OSCC	0	9	1	0	0	2	127	55	53
9	37	MALE	OSCC	0	0	10	0	0	2	123	42	39
10	42	MALE	OSCC	0	3	7	0	0	2	118	38	35
11	42	MALE	OSCC	2	4	4	2	0	0	104	39	36
12	58	MALE	OSCC	0	1	9	0	0	2	120	38	41
13	65	MALE	OSCC	0	8	2	2	0	0	124	34	35
14	50	FEMALE	OSCC	2	6	2	1	4	0	133	36	38
15	45	FEMALE	OSF	4	6	0	1	0	0	104	33	34
16	38	MALE	OSF	0	5	5	0	1	0	132	38	41
17	42	MALE	OSCC	0	4	6	0	3	0	132	45	39
18	35	MALE	OSCC	0	6	4	2	1	0	128	35	36
19	42	MALE	OSCC	2	8	0	2	1	1	145	38	41
20	55	FEMALE	OSF	0	7	3	1	0	0	125	34	35
21	34	MALE	OSF	0	3	7	0	0	0	124	40	39
22	56	MALE	OL	0	10	0	1	4	0	128	35	36
23	50	MALE	OL	1	4	5	2	0	4	132	41	42
24	40	MALE	OL	5	3	2	2	1	1	93	42	39
25	56	MALE	OL	5	5	0	0	1	0	142	41	40
26	29	MALE	OSF	0	8	2	0	2	0	124	39	42
27	60	MALE	OSCC	0	3	7	0	0	0	128	41	40
28	60	MALE	OSCC	2	6	2	0	2	0	128	46	40
29	23	MALE	OSF	0	10	0	0	2	0	108	41	37
30	48	MALE	OL	2	8	0	0	0	2	127	38	42

31	42	MALE	OSCC	0	6	4	0	0	0	123	42	45
32	58	MALE	OSF	0	7	3	0	2	0	110	36	41
33	67	MALE	OSCC	0	2	8	0	2	3	142	34	38
34	33	FEMALE	OSCC	0	6	4	0	0	0	112	39	42
35	65	MALE	OSCC	0	4	6	0	4	0	135	41	39
36	40	MALE	OSCC	0	7	3	2	0	0	125	42	43
37	51	MALE	OSCC	0	0	10	0	0	3	134	34	37
38	45	MALE	OL	0	2	8	2	0	0	115	40	42
39	30	FEMALE	OSF	0	0	10	0	3	0	110	43	46
40	40	FEMALE	OSF	0	0	10	1	3	0	119	42	45
41	49	MALE	OL	0	4	6	1	0	2	123	43	39
42	63	FEMALE	OL	0	2	8	0	2	0	106	42	39
43	33	FEMALE	OL	1	8	1	1	0	2	123	45	43
44	28	MALE	OSF	2	8	0	0	4	0	132	46	44
45	56	MALE	OSCC	0	6	4	0	0	2	112	43	39
46	65	MALE	OSCC	0	8	2	2	0	2	132	43	41
47	34	MALE	OSCC	0	3	7	0	2	0	102	43	39
48	65	MALE	OSCC	2	8	0	0	0	0	98	35	38
49	54	MALE	OSCC	2	6	2	2	0	0	121	42	38
50	22	MALE	OSF	0	6	4	2	0	0	135	41	35
51	61	MALE	OSCC	0	6	4	0	0	2	120	40	39
52	53	MALE	OSF	0	4	6	2	0	4	130	39	38
53	48	MALE	OSF	4	6	0	2	0	2	121	40	41
54	29	MALE	OSF	0	8	2	0	0	4	132	50	49
55	62	MALE	OSCC	0	4	6	1	2	0	143	42	46
56	56	MALE	OL	2	2	4	0	2	1	134	47	40
57	45	FEMALE	OSF	0	0	10	1	0	4	126	47	45
58	45	MALE	OSF	0	0	10	2	0	0	138	39	38
59	53	MALE	OSCC	0	6	4	0	2	0	132	40	45
60	42	MALE	OSF	0	6	4	0	2	0	142	44	43
61	57	MALE	OSCC	2	8	0	0	2	0	134	45	47
62	58	MALE	OSCC	0	8	2	0	0	0	129	50	54
63	59	MALE	OSCC	0	7	3	0	2	0	146	40	37
64	57	MALE	OSCC	0	0	10	0	2	2	92	40	45
65	55	FEMALE	0SCC	0	6	4	0	1	2	132	46	39
66	77	MALE	OSCC	0	0	10	1	2	1	121	40	40
67	50	MALE	OSCC	3	7	0	0	1	1	87	40	43
68	55	MALE	OSCC	0	2	8	0	0	0	108	39	46

69	58	MALE	OSCC	0	6	4	0	2	0	143	47	45
70	47	MALE	OSCC	0	5	5	0	2	2	124	35	45
71	48	MALE	OSCC	0	6	4	0	0	0	145	45	42
72	43	MALE	OL	3	2	5	0	2	0	128	55	45
73	40	MALE	OSF	0	10	0	1	1	0	110	45	47
74	64	FEMALE	OSCC	1	9	0	0	2	0	101	41	37
75	56	FEMALE	OSCC	1	6	3	2	2	0	116	47	46
76	29	MALE	OSF	0	9	1	2	0	0	124	41	48
77	61	MALE	OL	0	8	2	0	2	0	141	45	46
78	32	MALE	OSCC	0	10	0	0	1	1	120	49	43
79	43	MALE	OSCC	0	10	0	2	2	0	131	56	54
80	34	MALE	OSCC	1	0	9	0	2	0	111	42	38
81	42	MALE	OSCC	0	3	7	0	2	0	129	39	42
82	38	MALE	OSCC	0	4	4	0	0	2	133	35	37
83	43	MALE	OSCC	0	6	4	0	0	2	138	37	35
84	44	MALE	OSCC	1	4	5	0	0	2	112	44	39
85	56	MALE	OSCC	0	3	7	0	0	2	127	35	36
86	65	MALE	OSCC	2	6	2	2	0	0	103	37	40
87	50	FEMALE	OSCC	0	7	3	1	4	0	121	40	40
88	43	FEMALE	OSF	0	8	2	1	0	0	119	34	35
89	37	MALE	OSF	0	1	9	0	1	0	125	42	43
90	41	MALE	OSCC	1	5	4	0	3	0	132	41	38
91	43	MALE	OSCC	4	6	0	2	1	0	92	42	41
92	57	MALE	OSCC	0	9	1	2	1	1	133	45	41
93	63	FEMALE	OSCC	0	8	2	1	0	0	132	42	38
94	51	MALE	OSF	0	8	2	1	0	0	128	40	36
95	44	MALE	OSF	1	2	7	2	1	1	128	43	44
96	38	MALE	OL	0	4	6	2	0	4	131	41	38
97	41	MALE	OSCC	0	1	9	2	1	1	121	35	36
98	46	MALE	OSF	2	8	0	0	1	0	106	44	40
99	43	MALE	OL	0	1	9	0	2	0	123	33	37
100	54	MALE	OSCC	0	5	5	0	1	2	122	39	38

GROUP B - Patients with habits without lesions

PATIENT	AGE	GENDER	FINGER RIDGE PATTERN			I1/THENAR,I2,I3,I4 AND HYPOTHENAR			TOTAL FINGER RIDGE COUNT	ATD ANGLE	
			ARCHES	LOOPS	WHORLS	ARCHES	LOOPS	WHORLS		RIGHT	LEFT
1	52	MALE	0	9	1	2	0	0	129	43	41
2	54	MALE	0	2	8	2	2	0	135	48	43
3	53	MALE	0	4	6	2	2	0	142	47	43
4	20	MALE	0	8	2	2	0	2	132	44	37
5	33	MALE	1	9	0	0	1	0	113	46	44
6	36	MALE	0	9	1	0	3	0	129	40	43
7	34	MALE	0	0	10	0	2	0	128	37	38
8	24	MALE	0	10	0	2	2	0	134	48	44
9	50	MALE	0	0	10	1	0	0	126	47	40
10	60	MALE	0	0	10	0	2	0	131	38	41
11	64	MALE	0	2	8	0	2	0	119	38	35
12	40	MALE	3	5	2	0	2	0	110	30	33
13	57	MALE	0	3	7	0	0	2	121	47	40
14	63	MALE	0	8	2	0	0	4	128	46	49
15	54	MALE	3	7	0	1	1	0	123	45	42
16	37	MALE	0	3	7	0	0	4	139	42	47
17	62	MALE	2	8	0	1	1	1	109	45	48
18	37	MALE	0	6	4	2	0	0	142	38	44
19	62	MALE	0	4	6	0	2	0	123	48	55
20	59	MALE	0	10	0	0	3	0	121	43	42
21	49	MALE	0	8	2	0	0	2	101	45	40
22	58	MALE	0	10	0	2	0	0	102	40	40
23	56	MALE	0	0	10	0	1	0	139	32	41
24	46	MALE	0	2	8	0	1	1	121	33	40
25	47	MALE	0	0	10	1	2	1	149	45	44
26	40	MALE	0	0	10	1	2	0	110	45	44
27	51	MALE	0	4	6	0	0	2	121	35	40
28	48	MALE	0	3	7	0	2	2	118	40	48
29	71	MALE	0	4	6	2	0	0	123	45	45
30	48	MALE	0	0	10	2	0	0	117	33	42
31	60	MALE	0	7	3	0	2	2	108	38	42
32	33	MALE	0	0	10	0	1	1	141	37	41
33	40	MALE	2	8	0	0	2	0	102	35	39

34	35	MALE	1	4	5	1	1	0	135	37	40
35	34	MALE	0	6	4	0	0	2	129	38	39
36	37	MALE	0	2	8	0	0	0	138	37	43
37	59	MALE	0	6	4	2	0	0	121	43	39
38	55	MALE	0	3	7	2	0	2	135	40	44
39	48	MALE	2	8	0	2	0	2	121	35	38
40	63	MALE	0	6	4	0	2	0	123	40	39
41	46	FEMALE	0	0	10	0	0	4	142	39	45
42	70	MALE	0	0	10	0	0	2	138	43	43
43	55	MALE	0	4	6	1	1	1	121	38	45
44	56	MALE	0	0	10	0	0	2	132	40	40
45	46	MALE	0	8	2	0	2	0	129	42	43
46	72	MALE	0	6	4	2	4	0	129	47	44
47	52	MALE	0	3	7	2	0	0	139	35	34
48	55	MALE	0	8	2	2	0	1	110	43	43
49	65	MALE	2	8	0	2	2	0	123	37	39
50	56	MALE	0	6	4	0	0	2	102	43	46
51	50	MALE	0	4	6	0	3	0	97	36	44
52	63	MALE	0	4	6	4	0	2	129	42	43
53	59	MALE	0	4	6	0	0	2	146	47	47
54	46	MALE	0	6	4	2	0	2	126	40	40
55	49	MALE	0	7	3	0	2	1	123	38	40
56	40	MALE	0	6	4	0	1	0	137	42	44
57	30	MALE	2	4	4	2	0	0	128	40	41
58	44	MALE	0	9	1	0	1	0	124	39	39
59	39	MALE	0	4	6	0	2	0	125	35	37
60	60	MALE	0	8	2	0	1	0	105	38	40
61	51	MALE	0	4	6	0	2	0	101	36	42
62	64	MALE	0	3	7	0	2	0	111	42	41
63	45	MALE	0	10	0	0	2	1	92	35	35
64	50	MALE	0	6	4	0	0	0	116	44	42
65	52	MALE	0	10	0	2	0	0	101	38	45
66	60	MALE	0	2	8	0	1	0	125	38	40
67	69	MALE	0	8	2	0	0	1	99	38	40
68	50	MALE	0	5	5	1	1	1	135	35	38
69	51	MALE	0	5	5	0	0	0	113	40	40
70	45	MALE	1	4	6	0	2	0	103	50	49
71	36	MALE	0	2	8	0	1	2	143	42	44

72	41	MALE	4	4	2	0	2	0	94	40	47
73	37	MALE	0	8	2	0	0	0	99	40	43
74	43	MALE	0	9	1	0	2	0	110	40	40
75	60	FEMALE	0	8	2	0	2	0	119	40	42
76	52	MALE	0	3	7	2	0	0	113	45	42
77	45	MALE	0	3	7	0	2	0	110	38	40
78	40	MALE	0	6	4	0	0	2	105	43	43
79	39	MALE	0	10	0	0	0	0	121	38	42
80	58	MALE	0	2	8	0	0	2	139	43	47
81	46	MALE	0	2	8	0	0	0	135	35	35
82	50	MALE	0	4	6	2	0	0	112	38	38
83	40	MALE	0	6	4	1	1	0	125	42	44
84	30	MALE	0	8	2	0	3	0	100	37	40
85	49	MALE	0	4	6	0	2	0	121	38	42
86	54	MALE	0	5	5	0	1	2	98	37	42
87	61	MALE	1	9	0	2	0	0	104	38	38
88	51	MALE	0	2	8	2	0	0	132	35	40
89	70	MALE	0	8	2	0	1	1	116	43	42
90	51	MALE	0	8	2	0	0	2	103	38	35
91	58	MALE	0	7	3	0	0	2	121	38	42
92	50	MALE	0	2	8	0	0	2	132	40	42
93	45	MALE	0	4	6	0	2	0	118	40	40
94	53	MALE	0	9	1	2	0	0	126	42	40
95	55	MALE	0	2	8	2	2	0	134	47	44
96	54	MALE	0	4	6	2	2	0	141	45	41
97	21	MALE	0	8	2	1	0	0	133	42	38
98	34	MALE	1	9	0	2	0	2	115	38	39
100	37	MALE	0	9	1	0	2	0	128	47	45

GROUP C - Patients without any habits

PATIENT	AGE	GENDER	FINGER RIDGE PATTERN			I1/THENAR,I2,I3,I4 AND HYPOTHENAR			TOTAL FINGER RIDGE COUNT	ATD ANGLE	
			ARCHES	LOOPS	WHORLS	ARCHES	LOOPS	WHORLS		RIGHT	LEFT
1	54	MALE	0	0	10	0	0	1	143	38	38
2	65	MALE	0	6	4	0	1	0	137	48	50
3	53	MALE	0	4	6	0	2	0	128	45	46
4	58	MALE	0	5	5	0	0	2	142	49	44
5	40	MALE	0	8	2	2	2	0	129	48	45
6	62	MALE	0	1	9	1	2	1	148	40	45
7	68	MALE	0	2	8	2	2	0	139	44	40
8	75	MALE	0	5	5	1	2	0	146	35	37
9	49	MALE	0	6	4	0	1	0	128	39	40
10	47	MALE	0	7	3	2	0	3	142	37	47
11	52	FEMALE	0	0	10	0	2	0	131	30	30
12	50	MALE	0	9	1	0	2	0	126	41	38
13	52	MALE	0	0	10	0	0	0	119	40	50
14	53	FEMALE	0	2	8	2	0	0	126	42	39
15	40	FEMALE	0	3	7	1	2	1	131	42	44
16	55	MALE	0	6	4	2	2	0	149	37	40
17	50	MALE	0	7	3	1	0	0	152	32	37
18	60	MALE	0	3	7	0	0	0	147	42	35
19	60	MALE	0	2	8	2	2	0	138	37	40
20	61	MALE	0	2	8	0	0	0	129	38	41
21	48	MALE	0	2	8	0	0	2	143	43	40
22	56	MALE	0	3	7	0	0	2	129	44	42
23	50	MALE	0	6	4	0	0	2	135	47	45
24	54	MALE	0	1	9	3	3	0	137	47	47
25	55	MALE	0	8	2	0	0	2	124	43	45
26	30	MALE	0	5	5	0	2	0	134	51	48
27	50	MALE	0	6	4	0	1	0	135	40	48
28	55	MALE	0	7	3	2	0	0	145	48	42
29	51	MALE	0	8	2	0	0	1	117	40	40
30	56	MALE	0	9	1	2	2	0	138	47	39
31	44	FEMALE	0	0	10	2	0	4	147	40	38
32	49	MALE	2	2	6	1	2	1	135	42	41
33	69	MALE	0	6	4	0	2	0	132	47	38

34	43	MALE	0	8	2	2	2	0	136	39	39
35	68	MALE	0	2	8	1	0	0	129	40	39
36	60	MALE	0	7	3	2	0	0	135	49	45
37	55	MALE	0	8	0	0	0	0	129	42	45
38	38	MALE	0	5	5	1	0	0	141	39	41
39	60	MALE	0	2	8	2	2	0	134	42	46
40	72	MALE	0	8	2	0	0	0	121	45	42
41	63	MALE	0	1	9	0	0	0	128	40	43
42	35	MALE	0	10	0	1	1	0	134	42	42
43	65	MALE	0	0	10	1	3	0	129	43	40
44	63	MALE	0	0	10	1	0	2	132	40	40
45	52	MALE	0	3	7	2	0	0	143	38	35
46	66	MALE	0	0	10	0	2	0	128	43	44
47	67	MALE	0	10	0	0	2	0	137	38	48
48	40	MALE	0	2	8	1	2	0	129	38	41
49	57	MALE	0	7	3	0	2	0	138	42	43
50	57	MALE	0	10	0	0	2	0	128	42	40
51	45	MALE	0	10	0	0	2	1	118	40	45
52	51	MALE	1	6	3	0	0	2	129	35	35
53	33	MALE	0	8	2	0	1	0	132	35	40
54	69	MALE	0	0	10	1	0	2	145	43	40
55	40	MALE	0	8	2	0	2	0	129	43	46
56	59	MALE	0	0	10	0	2	3	138	40	45
57	42	MALE	0	0	10	0	2	0	142	33	37
58	70	MALE	0	10	0	0	2	0	139	35	35
59	64	MALE	0	6	4	1	0	2	142	42	49
60	42	MALE	0	7	3	0	2	0	138	40	48
61	52	MALE	0	8	2	1	2	0	129	37	40
62	56	MALE	0	6	4	0	0	2	131	37	38
63	64	MALE	0	3	7	0	4	0	129	42	45
64	60	MALE	0	4	6	0	0	2	138	40	41
65	56	MALE	0	3	7	0	1	1	143	40	39
66	43	MALE	0	10	0	0	0	2	123	48	50
67	47	MALE	0	4	6	0	0	2	134	40	35
68	65	MALE	0	10	0	0	0	2	128	44	44
69	53	MALE	1	8	1	0	2	0	131	47	48
70	55	MALE	0	0	10	2	0	0	118	37	38
71	66	MALE	0	6	4	0	1	0	144	47	49

72	54	MALE	0	4	6	0	2	0	135	44	45
73	57	MALE	0	1	9	0	0	2	136	48	45
74	41	MALE	0	8	2	3	3	0	123	47	45
75	43	MALE	0	5	5	0	0	2	138	45	41
76	63	MALE	0	2	8	0	0	2	134	34	36
77	67	MALE	0	5	5	0	0	2	128	38	39
78	74	MALE	0	6	4	0	0	0	142	38	48
79	48	MALE	0	9	1	2	2	0	130	31	31
80	46	MALE	0	0	10	0	0	0	142	42	39
81	44	FEMALE	0	7	3	2	2	0	128	42	49
82	53	MALE	0	6	4	0	0	0	137	43	40
83	51	MALE	0	2	8	1	0	0	142	43	45
84	59	MALE	0	5	5	0	0	2	130	38	41
85	61	FEMALE	0	6	4	0	0	1	148	33	38
86	48	FEMALE	0	7	3	0	2	0	123	41	34
87	57	MALE	0	3	7	0	2	0	133	38	41
88	52	MALE	0	2	8	0	0	0	127	37	42
89	56	MALE	0	6	4	2	0	3	120	44	41
90	54	MALE	0	7	3	0	1	0	124	45	41
91	57	MALE	0	6	4	1	2	0	119	46	46
92	31	FEMALE	0	1	9	2	2	0	125	42	44
93	52	MALE	0	8	2	0	0	2	132	52	49
94	54	MALE	0	5	5	2	2	0	141	41	49
95	52	MALE	0	8	2	0	0	2	129	39	38
96	51	MALE	0	7	3	2	2	0	143	42	41
97	54	MALE	0	6	4	1	2	1	136	37	34
98	56	MALE	0	5	5	0	2	0	149	42	39
99	62	MALE	0	8	2	0	1	0	130	31	35
100	71	MALE	0	1	9	0	0	1	143	42	43