ijlpr 2023; doi 10.22376/ijlpr.2023.13.1.L21-30

International Journal of Life science and Pharma Research

ISSN 2250-0480

Review Article

Medicine for novel therapy

Dermatoglyphics and Its Importance in Oral Precancerous and Cancerous Lesions - Systematic Review

Vinothini Gunasekaran[1], Elavarasi Elangovan[1], Prithiviraj Nagarajan[2*], John Hearty Deepak Jeyaraj [3], Rajan Thangarasu[4]

[1] Assistant Professor, Department of Dentistry, Aarupadai Veedu Medical College & Hospital, Vinayaka Mission's Research Foundation (Deemed to be University),Kirumampakkam, Puducherry - 607403, India

[2] Assistant Professor (Research), Multi-Disciplinary Centre for Biomedical Research, Aarupadai Veedu Medical College & Hospital, Vinayaka Mission's Research Foundation (Deemed to be University), Kirumampakkam, Puducherry- 607403, India*

[3] Reader, Department of Oral Medicine & Radiology, Rajas Dental College and hospital, Kavalkinaru Tirunelveli - 627007, India

[4] Professor & HOD Department of Anatomy, Aarupadai Veedu Medical College & Hospital, Vinayaka Mission's Research Foundation (Deemed to be University), Kirumampakkam, Puducherry; 607403, India

Abstract: Dermatoglyphics, the study of palm prints and fingerprints, is currently a precious method for the early detection of premalignant lesions, dental caries, systemic disorders, hereditary diseases, and syndromes. Palmar dermatoglyphics can indicate the development of potentially malignant lesions and help identify people at high risk of developing oral submucous fibrosis (OSMF) , and oral squamous cell carcinoma (OSSC), which cause panic and hold an excessively high ranking as killers. Another important condition that has become a major public health issue in the South Indian population is oral submucous fibrosis (OSMF), which is correlated with genetic abnormalities and is used in biomedical studies. Similarly, anthropologists and medical professionals have long found the study of the human hand fascinating, as have psychologists, novelists, artists, and chiromancers. Fingerprints do not change when the body decays, just as they change from birth to death. Our systematic review aims to assess the correlation between dermatoglyphics and oral precancerous and cancerous lesions in the human population and to investigate the embryogenesis and topology of dermatoglyphics, emphasizing the many types of research involving dermatoglyphics in many disciplines of medicine and dentistry. In our systematic review, we have collected many review papers using databases including PubMed, Medline, Scopus, Embase, and the Web of Science to determine the importance of dermatoglyphics in oral precancerous and cancerous lesions. The data from our systematic review showed a link between oral precancerous and cancerous lesions, which suggests that dermatoglyphics could be used to find precancerous and cancerous lesions in the oral cavity early.

Keywords: Dermatoglyphics, Fingerprints, Ridge Patterns, Oral Leukoplakia, Oral Submucous Fibrosis and Genetic Alterations

***Corresponding Author**

Prithiviraj Nagarajan , Assistant Professor (Research), Multi-Disciplinary Centre for Biomedical Research, Aarupadai Veedu Medical College & Hospital, Vinayaka Mission's Research Foundation (Deemed to be University), Kirumampakkam, Puducherry- 607403, India

Received On 5 September, 2022
Revised On 28 October, 2022
Accepted On 4 November, 2022
Published On 2 January, 2023

Funding This research did not receive any specific grant from any funding agencies in the public, commercial or not for profit sectors.

Citation Vinothini Gunasekaran , Elavarasi Elangovan , Prithiviraj Nagarajan , John Hearty Deepak Jeyaraj , Rajan Thangarasu , Dermatoglyphics and Its Importance in Oral Precancerous and Cancerous Lesions - Systematic Review.(2023).Int. J. Life Sci. Pharma Res.13(1), L21-30 http://dx.doi.org/10.22376/ijlpr.2023.13.1.L21-30

1. INTRODUCTION

The study of epidermal ridges and their arrangement on the fingers, palms, and soles is known as dermatoglyphics[1]. Cummins and Mid low (1926) created dermatoglyphics (derma = skin) to study of frogs and the ridges themselves[2]. The word dermatoglyphics is derived from the Greek words derm, meaning skin, and glyphics, meaning carving, which came up with the phrase[3,4]. Abnormal ridge arrangements are present in more than just individuals, and people with single-gene diseases also have chromosomal anomalies, as well as in some cases where the genetic cause of the condition is yet unknown[4]. It is also beneficial in identifying premalignant lesions and diseases of the oral cavity, such as tooth decay, periodontitis, head and neck cancer, cleft lip, cleft palate, and malignancy. The dermatoglyphic pattern begins to take shape with the emergence of the fetal pad in the sixth week of pregnancy. It attains its maximum size between the 12th and 13th weeks of development before reaching full maturity in the 24th week of pregnancy [5]. However, the exact method of heredity is still a mystery.

2. HISTORY OF DERMATOGLYPHICS

The first person to test fingerprints in India was William Herschel in 1858.[6] Sir Francis Galton (1892), through his considerable research, showed the hereditary relevance of fingerprints and biological differences between ethnic groups[7]. The term "dermatoglyphics" was first used in 1926 by Cummins and Mid low[3]. The use of dermatoglyphic screening in cases of Down syndrome and other congenital abnormalities was studied by Penrose LS in 1945 [8]. The book "Dermatoglyphics in Medical Disorders" by Schumann and Alters was released in 1976[4]. In Medical dermatoglyphics today; some disorders can now be identified only based on dermatoglyphics. This is because of the current state of the area. Much of the research now emphasizes that hand traits have a high degree of prediction accuracy. The importance of dermatoglyphics can be applied in various fields like congenital disorders, genetic abnormalities, educated fields, human resources, etc., and also possible to identify individuals genetically prone to oral squamous cell carcinoma and oral submucous fibrosis by doing a quantitative and qualitative assessment of their fingers and palm prints[7]. This strategy of using dermatoglyphics as a genetic marker is easy to carry out and more cost-effective than genetic biomarkers, which are already available[8]. An impression made by abrasive ridges that are nearly parallel and have a similar crest-to-crest wavelength is known as a fingerprint. The swirl, loop, arch, and triradii are some of the most prominent primary elements of the pattern[3]. A thorough investigation reveals hundreds of additional imperfections, such as ridge ends, ridge bifurcations, island ridges, etc. The uniqueness of fingerprints is on the type of dislocation type and relative geometry[12]. Some of the characteristics used for personal identification include the uniqueness and consistency of fingerprints throughout life[10]. The genetic components of fingerprint embryogenesis require their correlation with disease status[11]. Dermatoglyphics has also been accepted as a simple and inexpensive means of diagnosing numerous disorders of genetic and nongenetic origin[4]. Since most of the investigations required to confirm the diagnosis of hereditary disorders are complex expensive, and dermatoglyphics can be efficiently employed with other clinical signs as a noninvasive, simple, and inexpensive screening procedure. The question now is to test the predictive possibilities of dermatoglyphics in search of the various oral precancerous and cancerous, as the studies conducted so far have been convincing. However, it is still in its infancy in dentistry, where the correlation of dental conditions with dermatoglyphic patterns is done. Hence, the present systematic reviews focus on dermatoglyphics and work conducted by various authors on the application of dermatoglyphics in dentistry, along with the advantages and patterns of dermatoglyphics.

3. MATERIALS AND METHODS

Databases were searched for study inclusion criteria, including the NCBI Database, Web of Science, CrossRef, Scopus, Medline, PubMed, and Google Scholar. Search procedures were carried out using essential keywords on the subheadings of dermatoglyphics, fingerprints, ridge patterns, oral leukoplakia, oral submucous fibrosis, and genetic alterations. Case reports, abstracts, and editorials were excluded.

3.1 Dermatoglyphic Studies in Oral Leukoplakia

The World Health Organization (WHO) approved the most recent definition of oral leukoplakia in 2005, stating that it should be used to identify white patches of dubious risk after excluding other existing conditions but does not enhance the chance of developing cancer[1].

3.2 Epidemiology

Leukoplakia prevalence was found to be 3.6%, and pre-leukoplakia prevalence was found to be 6.4%. According to reports, 2.9% of leukoplakia was exclusively caused by tobacco use, while 0.7% was idiopathic[13].

Gender and age: The most significant incidence occurs around age 50 and often begins 30 years after onset. The frequency of leukoplakia increases with age, with middle-aged and older men being the most affected. Oral leukoplakia can develop up to five years before oral cancer. The majority of the population is male than female. Leukoplakia is a lesion that frequently develops, especially in adults over the age of 40. 2:1 is the male-to-female ratio[14]. Most studies have a mixed gender distribution, ranging from a considerable male predominance in some areas of India to about 1:1 in the Western world [31]. Bánóczy J conducted 30-year follow-up research with 670 individuals with oral leukoplakia and discovered cancer growth in 40 instances[54]. Leukoplakia is more common in people aged 51- 60, while carcinoma is more common in people aged 61 -70, according to age distribution[11]. In the leukoplakia group, the male-to-female ratio was 3.2:1, but in the carcinoma group, it was 1.9:1[9].

3.3 Variables that cause Leukoplakia [1]

- Use of alcohol and tobacco
- Localized pain from sharp teeth, poor denture fit, and poor restorations
- Immune system weakness
- A personal or family history of cancer
- Areca nut and betel leaf chewing are practiced in various cultures.

There are two primary types[9]

1. Homogenous
2. Non- Homogenous

These two types' differences are purely clinical and based on morphological features such as surface colour and thickness, which affect prognosis.

3.4 Homogenous

Homogenous leukoplakia is a flat, thin-appearing, mostly white lesion that may have small fractures and a uniformly smooth, wrinkled, or corrugated surface. A malignant transformation is a rare event. Although it may be greyish-white, the lesion is primarily white. It makes up about 84% of leukoplakia[14].

3.5 Non- Homogenous

Ulcerative: White and red colours mix together but still appear primarily white.

Nodular (mottled): Small, circular, red, or white streaks with polypoid outgrowths[30]. The name "erythro leukoplakia" is used primarily for red and white lesions that may be irregularly flat, nodular, exophytic, or verrucous[32]. The presence of a wrinkled or corrugated surface. White spots or nodules on the erythematous foundation are defined as nodular lesions[38].

3.6 Clinical Signs

The most commonly affected areas are the retro commissural region, buccal mucosa, edentulous alveolar ridge, hard palate, tongue, and lips[1]. In Indians, the gums, soft palate, and oral floor are less affected than in western countries[30]. The early stages of leukoplakia are thin, translucent, grey-white plaques that are soft, flat, and sometimes fissured or wrinkled [9].

3.7 Oral Precancerous Lesions and Oral Cancers

In the potentially malignant epithelial lesions (PMEL) group, leukoplakia is one of the most prevalent premalignant lesions most frequently seen in the oral cavity (van der Waal 1997). (Leuko means white; Plakia means patch). According to the World Health Organization (WHO) [13], oral leukoplakia (OL) is a white lesion that cannot be attributed to any other specific oral mucosal disease[15]. According to various studies and locations, OL is one of the most frequent PMELs of oral mucosa, with a malignant transformation rate ranging from 0.6% to 20%[14]. The fatal transformation rate has been reported to be 0.3% yearly. Studies in Western countries have found somewhat higher numbers with a malignant transformation rate of about 1% per year, which may be an acceptable average for all forms of leukoplakia[13]. On the other hand, oral submucous fibrosis (OSF) is another common premalignant condition associated mainly with chewing areca nut, an ingredient of betel quid. It is more prevalent in the South Asian population[20]. OSF is also called diffuse OSF, idiopathic scleroderma of mouth, idiopathic palatal fibrosis, sclerosing stomatitis, juxta-epithelial fibrosisetc.,[16] It is a potentially malignant disorder and a crippling condition of the oral mucosa[17]. Although available epidemiological evidence suggests that chewing gutkha is an important risk factor for the development of OSF, the genetic etiology of OSF has been studied since 1986, suggesting a genetic predisposition[18]. The occurrence of OSF is more or less restricted to Southeast Asia; However, several cases have also been reported in other parts where such an etiology is not seen, suggesting a possible role of genetic factors toward the same[13]. Furthermore most of the available epidemiological evidence indicates that chewing gutkha and using tobacco (smoking and non-smoking) is an important risk factor for OSF; not all individuals develop the same. Patients without any risk factors also develop a malignancy explaining the role of genetic factors as a plausible explanation for such an individual variation[19]. Later in the disease process, fibrosis and hyalinization occur in the lamina propria, leading to upper epithelial atrophy. In the presence of carcinogens, this atrophic epithelium turns malignant. Long-term investigations found OSF had an annual malignant transformation rate of 0.5%[13]. Oral squamous cell carcinoma (OSCC) is the most frequent oral cancer, developing from the stratified squamous epithelium of the oral mucosa in 95% of cases. OSCC is the sixth most frequent cancer worldwide and is a major problem in India[21]. OSCC ranks tenth in the world regarding epidemiological variation in cancer incidence. The third most prevalent cancer in South-Central Asia[22]. The WHO expects OSCC to increase in the next two decades. OSCC accounts for 2-4% of identified malignancies and 8000 deaths annually in the US[4, 5]. 36% of US patients had the local disease at the time of diagnosis, 43% had the regional disease, and 9% had distant metastasis[22]. OSCC causes an average of 500,000 new cases each year, which can be a huge health burden in some areas[23]. Some people are prone to OSCC because of an inherited trait that inhibits their ability to metabolize carcinogens and/or procarcinogens. Others have a reduced ability to repair carcinogen-damaged DNA. Susceptibility to some cancers is due to acquired immunological deficiencies. In some, susceptibility arises from defective genes controlling the fate of chromosomally damaged cells, cell cycle-tumor suppressor genes, and cell signaling genes (proto-oncogenes and oncogenes) [24] . In a case-control study, one evaluated genetic polymorphisms and oral cancer susceptibility to drug - metabolizing enzymes related to the CYP1A1, GSTMI, and GSTT1 genes. Habitual betel nut/tobacco chewers of Indian ethnicity with the null genotypes of GSTMI and GSTT1 are at an exceptionally increased risk of developing OL, precancerous cancer that often turns fatal. Based on these findings, they concluded that it is possible to identify individuals diagnosed with cancer by GSTM1/GST1 genotyping and then take preventive steps to control large-scale mortality[25].

3.8 Genetic Marker for Oral Submucous Fibrosis

In an oncogene or tumor, the multi-stage progression of OSCC involves accumulating genetic and epigenetic changes[46]. Suppression of tumor suppressor genes that disrupt the cell cycle. Growth inhibitors include Janus kinase/signal transducer and activator of transcription (JAK/STAT3), RasMAPK-ERK (Ras-mitogen-activated protein kinase-extracellular), PI3K/Akt/mTOR (mechanistic target of phosphoinositide 3-kinase/Akt) are included), and PLC-PKC (phospholipase C)-/protein kinase[47]. The tumor suppressor gene p53 is frequently mutated in oral cancer; this change renders it inactive and contributes to the development of OSCC [48]. Notch1 is another tumor suppressor gene; it has been reported that in Chinese patients, 54% of OSCC and 60% of paraneoplastic lesions carry the mutation [49]. For OSCC development, NOTCH1 modifications have been proposed [50]. On the other hand, 25% - 40% of all human cancers have increased MDM2, a proto-oncogene [47]. The tumor suppressor gene p53 has been studied for precancerous regions in OSCC [46]. Co-expression of the proteins p53 and Mdm2 has been proposed as a marker for aggressive tumor behavior in OSCC. In oral cancer cells, the proto-oncogene Akt1 is highly expressed, which promotes cancer cell growth, survival, and metastasis[47]. Poor outcomes are predicted when the PKB/AKT pathway is

activated as it initiates multiple cellular actions, promotes tumor growth, and more [47]. An additional affected Via is EGFR, whose over-expression is associated with an aggressive phenotype and poor prognosis in oral cancer [52]. Additionally, TC21 is a member of the RAS family, which is involved in Erk2, 14-3-3, and PI3-K interactions in multiple cell processes, suggesting that TC21 is a signalling pathway for oral cancer[46]. Furthermore, the earlier stages of OSCC (T1+T2) displayed higher TC21 expression than the later stages (T3+T4); this indicates that the prognosis of OSCC is poor [51]. Prostaglandins are produced from arachidonic acid by the enzyme cyclooxygenase -2 (COX-2) as a target molecule of NF-kB. By increasing the evasion of vascular and cellular proliferation and apoptosis processes, overexpression of COX-2 promotes cancer progression[43]. This suggests that NF-kB activation may reduce patient survival [47]. Mutations can also occur in the genes CDKN2A, CCND1, PIK3CA, PTEN, and HRAS. Initiates and progresses OSCC and contributes to cell cycle deregulation and immortalization [46]. There is a strong association between metastatic grading and lymph node, the WNT/-catenin signaling pathway is frequently mutated, and carcinogenesis. The WNT/-catenin pathway may be a target in anticancer therapy for metastatic disease [53]. The basal cell layer of the normal lingual epithelium, which controls tissue maintenance and regeneration, contains BMI1+ cells. Recent research has shown that the OSCC BMI1+ subpopulation is a group of tumour cells that grow slowly and are responsible for OSCC 46's aggressive growth and spread to other parts of the body. [46].

3.9 Comparison of Dermatoglyphic Patterns in Oral Leukoplakia, OSSC, and Oral Submucous Fibrosis Patients

The patterns of prints on the palm and plantar surfaces of the hands and toes are the subjects of this research[6]. Each person's fingerprints are unique by the 24th week of intrauterine life, they begin to develop. Many genes interact with environmental conditions to form an individual's distinctive patterns[4]. Ridge patterns in fingerprints exhibit anomalous behavior in cases of chromosomal or genetic abnormalities. Finding links between physical and genetic traits and several clinical disorders has recently attracted the attention of many researchers[8]. Fluctuating asymmetry accurately reflects and quantifies developmental instability. Fluctuating heterogeneity is a sign of genetic and environmental stress[26]. Various ridge patterns on the plantar surfaces of the hands can be examined to detect this asymmetry. Therefore, determining the relationship between genetics and disorders using dermatoglyphics is quick, painless, and inexpensive. The genetic basis for the different finger patterns was found by Ramani et al [27]; they become a reliable sign of genetic damage once they develop and are resistant to aging and environmental changes[28]. In light of this knowledge, the current systematic review was conducted to assess, and compare the fingerprint patterns of people with oral leukoplakia and OSMF [29]. The frequency of tobacco use in India is quite high. This problem has resulted in increased cases of oral leukoplakia and submucous fibrosis. Although these lesions are curable, there is a potential that they might develop into cancers if they go undetected. Various genetic, molecular, and chromosomal alterations, as well as the malignant lesions that result, differentiate oral, possibly malignant lesions from other types. Using dermatoglyphics is a quick and easy technique to analyze these lesions. Awasthi D et al. studied OSMF and oral leukoplakia by comparing the dermatoglyphics of healthy participants without habit or lesion and those with a habit but no lesion. They claimed that although the loop pattern is often observed in all populations, the frequency of loops is somewhat higher in pathological diseases, including OSMF and leukoplakia[30]. The most frequently searched pattern was the loop pattern. However, most studies discuss oral leukoplakia and OSMF rather than heavy subjects. Healthy patients exhibit a more loop pattern compared with oral leukoplakia and OSMF. Compared to OSMF patients and gutka chewers, Munishwar and colleagues found that the loop pattern was more prevalent in a healthy group, in which the 15 OSMF patients (43.60%) and the control group (57.60%), an increase in the loop pattern was observed [31]. According to Kulkarni et al., loop patterns are higher in healthy participants (61.0%) than in those with OSMF (57.0%), although they are less in healthy subjects [32]. Jatti D. et al. compared dermatoglyphics with potentially fatal diseases and oral squamous cell carcinoma. He found that in an arch, the pattern is usually found in potentially fatal diseases[33]. In a study by Tamgire Dw et al., comparisons were made between subjects with habits but no lesions and subjects with OSMF. He found a decline in the pattern of whorls was observed compared to the OSMF Gutkha chewers[34]. Gupta and Karjodkar studied the correlation of dermatoglyphics with OSMF and oral squamous cell carcinoma subjects. They reported an increase in loop and arc patterns in the OSMF group but a decrease in the frequency of the winding pattern [35]. Similarly, Kulkarni's study showed that there had been a decrease in the percentage of loop patterns in OSMF patients and an increase in the frequency of healthy patients swirl patterns in oral leukoplakia and OSMF subjects [36]. Satish Kumar et al. examined correlative subjects with no habit or lesion and subjects with OSMF[18] , and he reported that whorl patterns increased among OSMF subjects compared to healthy subjects[37]. In another study by Venkatesh et al on dermatoglyphics in patients with oral leukoplakia and OSCC, the frequency of an arch pattern was higher in oral leukoplakia subjects than in healthy subjects, whereas the frequency of a curved pattern was lower in oral leukoplakia subjects than in healthy subjects [38].

3.10 Basics of Dermatoglyphic Patterns

The dermal ridge configurations are a direct consequence of the surface topography of the fetal hand during the dermal ridge development between the 13th and 19th weeks of prenatal life[39].

The various stages are:
1. Early organ development (4 - 6 weeks)
2. Pad Appearance (between 6 1/2 - 8 weeks)
3. Pad Reversal (10 -12 weeks)
4. Ridge Development (13th week).
5. A Clear Pattern (19th Week).

The dermal ridge configuration directly results from physical and topographic growth factors acting on the volcanic skin, which is polygenically prone to forming parallel dermal ridges. Genes have a direct role in ridge patterning, which is an indirect consequence of the overall shape of the hand as the ridges grow[39]. Congenitally deformed hands were noted as having a ridge configuration by Cummins (1943), who argued that these ridges indicated the orientation of the epidermis. The increased pressure at the time of ridge development and the contour of the volar skin were responsible for determining their shape [3]. According to Penrose's theory from 1965, rapid changes in fluid balance during the embryonic stage cause the

ridges to align at right angles to compression pressures, follow the shortest paths on embryonic surfaces, and have unequal configurations[8]. According to Hirsch et al. (1973), the network of blood vessels and nerve pairs beneath the smooth epidermis is believed to have existed before the epithelial layers. This system is believed to have evolved from the blood vessel-nerve joints that cause wrinkles. In pattern formation, the neuroepithelium is important[40]. Palmar and finger patterns are two different pattern configurations shown in Table 1.

Table 1: Types of palmar and fingertip patterns

Palmar patterns	Fingertip patterns
Thenar area (Th or I1)	Arches
Hypothenar area (Hy)	Loops
Interdigital areas (I2, I3, I4)	Whorls
Palmar creases (DC, PC and TC)	
Atd angle	

The four interdigital areas, the thenar region, the hypothenar part, and other anatomical divisions are all on the palm. No pattern is seen in the sole region [3] (Fig. 1)

3.10.1 The Distal Crease (DC)

It is a gesture that starts from the side of the palm and finishes in the centre of the finger.

3.10.2 Proximal Crease (PC)

This develops from the hypothenar between the thumb and the pointing finger, including the ends.

3.10.3 The Thaner Crease (TC)

It is a fold that often connects to the PC, starts at the palm's base, and runs between the thumb and fingers.

ATD Angle: An ATD angle is the angle formed by lines drawn from a digital triangle (A) to an axial triangle (T) and then from it to a digital triangle (D). The angle atd increases with distance from the locus of t (Figure 2).

Triradius: It is formed when three ridge systems converge. A *triangle point* is a point at which the triangle is geometrically centered. The junction of the three ridges makes an angle of about 120 degrees with each other [3]. All 10 digits of an individual were considered to find fingerprint pattern frequency; both right and left palms were studied for hypothenar, I1, thenar, I2, I3, and I4 interdigital areas. From the triradii to the core, ridges were counted from the little finger on the right hand to the thumb on the left. The count starts on the ridge after the first triradii blank space. The core ridge wasn't counted. If more than one ridge counted, the largest one was considered. TFRC was derived by adding ridge counts on ten fingers. Ab ridge count was done between triradii a and b over the palm in the same way. Figure 3 shows how the widest atd angle was found: lines were drawn from the digital triradius "a" to the triradius "t" and from there to the distal triradius "d."

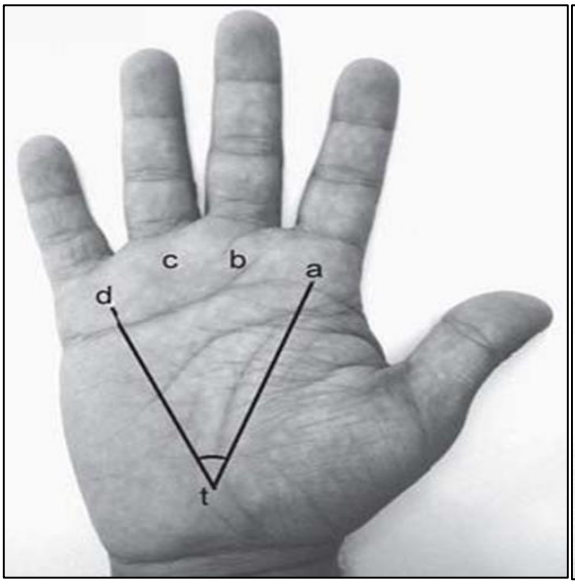

Fig 1: Pattern configurations

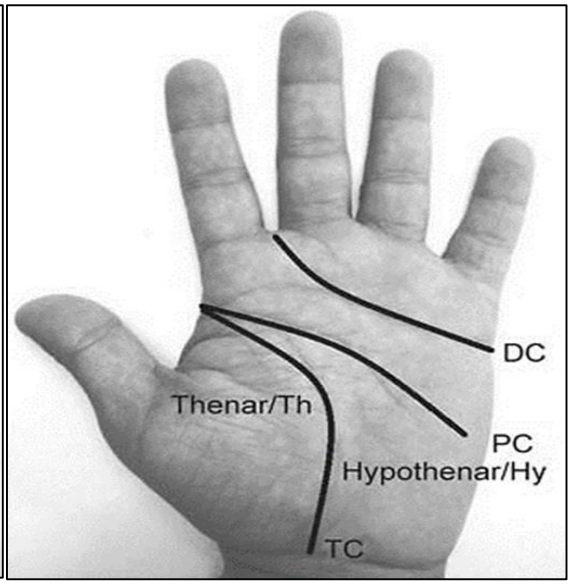

Fig 2: Atd angle

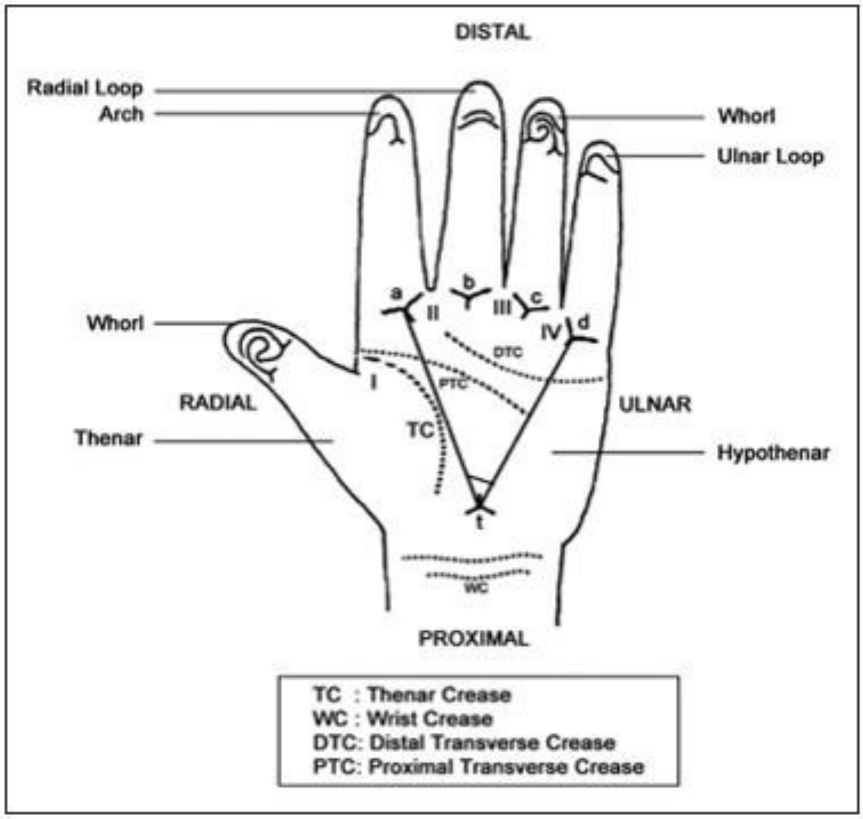

Fig 3: Anatomical areas on the palm

3.11 Anatomical Configurations

Finger patterns were classified by Galton (1892) into three categories [7]: Simple arches, vaulted arch loops, ulnar loops, and radial loops are all types of arches.

3.12 Arches

The simplest design on the fingers is called the arch (A). It comprises a series of roughly parallel ridges that run into the pattern region and come together to form the concave curve closest to the pattern. There are two types of arch patterns:

a. **The simple arch or plain arch** (PA): Composed of ridges extending the fingers from side to side without curling (Figure 4 A).

b. **Tent arc (TA):** It consists of ridges that come together at a point to break up a smooth sweep of ridges. Since ridges often emerge from this location in three different directions, the confluence point is known as the tri-radius. Near the midline axis of the distal phalanx, the triradius is located in the tentacles arch. In most cases, the distal radius of the triradius is upward towards the tip of the finger. The ridges crossing this radiance suddenly rise, known as the "tented arch" because of its tentacle-like appearance (Figure 4 B).

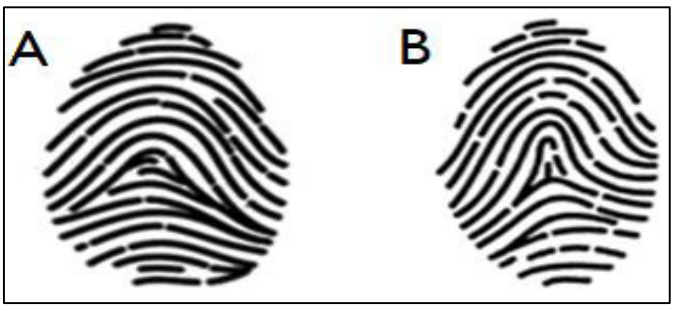

Fig 4: Shows simple and tented arch

3.12.1 Loops

On fingers, this is the most frequent pattern. The ridges enter the pattern area on one side of the points, make a sharp U-turn, and exit on the other side. The ulnar loop is a loop that results in a ridge opening towards the ulnar (Figure 5 A). A radial loop is a ridge that extends near the radial edge (Figure 5 B). A loop has only one triradius, or ridge, confluence point. Triradius is often seen at the edge of the fingertip where the loop closes. The shape and form of the loops can vary greatly. They can be huge or small, short or long, oriented vertically or horizontally, and in either plain loop (PL) or double loop (DL) form. Some transitional ends sometimes resemble whorls or intricate designs.

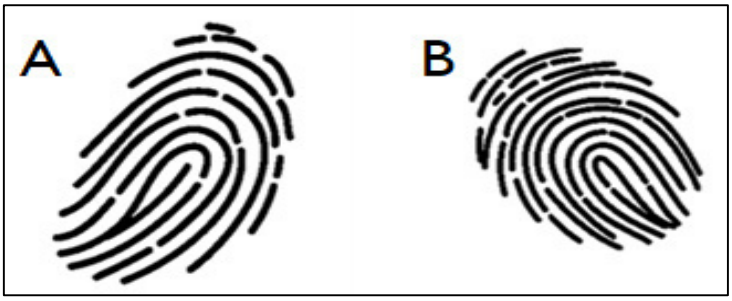

Fig 5 A: Shows the Ulnar loop B: Shows the radial loop

3.12.2 Whorl

Arcs with basic whorl and central pocket whorl. On the fingers, this design is the simplest. Simple arches lack triangles and have finger ridges running from one side to the other. The tented arch has a center triad and is made up of ridges that meet at a point and are interrupted by their smooth contours. They can be found in approximately 25% - 30% of all fingerprint patterns studied. The vortices of the ridge have the effect of forming at least one circuit. Generally speaking, a whorl pattern is any fingerprint pattern with two or more deltas Figure 6. There are six distinct whorls to choose from Mohd. Bhat et al [41].

- **Concentric whorl** - In the case of a concentric whorl, the ridges are organized into concentric rings around the centre.
- **Spiral whorl** - spiral around the centre in either a clockwise or counter-clockwise direction Figure 6 A. In this design, it combines circles and spirals to create a vortex.
- **Central pocket whorl** - a little pocket inside a loop used as a decorative element Figure 6 B. Twin vortices in these ridges are seen emerging from each core and extending to the opposite border of the finger.
- **Unintentional whorls:** These are patterns that are combinations of two or more of the preceding patterns

Fig 6 A. Shows spiral whorl B. Concentric whorl

3.12.3 Printing Methods

Palm printing is quick and inexpensive. (Figure 7A-C).

3.12.4 Inking Method

One of the most popular methods is the ink method. A roller, glass or metal icing slabs, sponge rubber, excellent quality paper with a slightly shiny surface, and printer ink are all necessary pieces of equipment[42].

3.12.5 Faurot Inkless Method

This technique uses a commercially patented product and sensory paper that is carefully prepared[42].

3.12.6 Transparent Adhesive Tape Method

To make prints, dry-colored pigment is applied to the skin and then lifted using transparent adhesive tape. Coloured chalk, dust, ink of India, regular ink, carbon paper, graphite rods or powder graphite, regular oil pastel crayons, etc., can be used as colour agents. Prints are crisp, not blurred, and can be kept for a very long time. [4]

3.12.7 Photographic Method

It is based on the principle of complete internal reflection, which occurs when an object is placed in front of the prism. A Poloroid camera is used to capture the increased picture [8].

3.12.8 New Methods

This epidermal pattern and underlying bone structures (radio dermatography), examination of sweat pores (hygrophotography), or checking the spatial form of a perforated skin patch, for example, enables the testing of the relationship between monkeys (plastic and mould) [4]. Except for infants, the dermal pattern of most people can be seen individually, without magnification, or using a simple hand lens and appropriate lighting.

3.12.9 Numerical Method

Synthesis algorithms are employed for fingerprint prints and are particularly developed for all comprehensible configurations of 'accuracy'. The approach allows the performance of the nuances of mathematical cataloging, the varieties of patterns, and digital fingerprint coding[43].

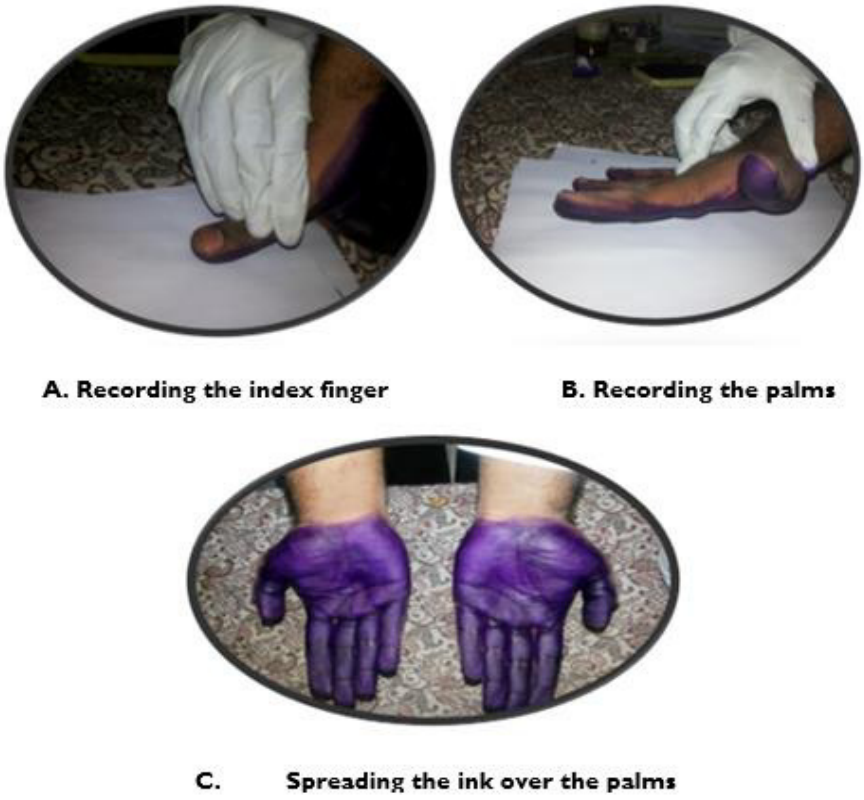

Fig 7: Different methods of printing

3.13 Indications

- One of the most valuable applications of dermatoglyphics is diagnosing and predicting substance misuse diseases such as alcoholism. These problems include mental retardation, autism, schizophrenia, and Alzheimer's disease, among others [44].
- It may also be used to assess a person's genetic susceptibility to dyslexia or hyperactivity, and it can be used as a diagnostic marker for several other trisomy types [45].
- It has been investigated for potential use as a biometric identification system; several dermatological studies have been conducted in various areas.
- Dermatoglyphics has gained popularity in dentistry. It can now diagnose and treat oral diseases such as dental caries, oral cancer, injuries, and anomalies such as cleft lip and palate. It can also be used to denote periodontal disease and dental fluorosis, as well as for medicinal purposes and forensic odontology in inquiry [44].

3.14 Advantages

There are many advantages to this procedure:

1) It is affordable, quick, and may be completed in clinics without for hospitalization or trauma.
2) It requires less equipment and data storage space and can be kept up and running forever [44].

3.15 Limitation

1) The limitation of the dermatoglyphic pattern is that it is difficult to determine if it is clinically meaningful when a patient has gross organ problems.
2) When recording prints, care should be taken to ensure that an adequate quantity of ink material is applied. Improper prints are caused by either too thin or too thick application [44].

4. CONCLUSION

Dermatoglyphics is one of the world's oldest and most valuable procedures, used for thousands of years. As a result of the hereditary nature of premalignant illnesses and oral squamous cell carcinoma, people prone to developing these lesions might avoid trigger factors by becoming familiar with their dermatoglyphic patterns. Fingerprints are stable and individualistic and make the most reliable criteria for identification. Fingerprint patterns are genetically determined and remain unchanged from birth to death. Identification using fingerprints can be established only when 16 to 20 points of similarity are present in the subtlety. As dermatoglyphics are genetically controlled characteristics, any deviation in dermatoglyphics patterns indicates a genetic difference between the control group and the abnormal population. Though dermatoglyphics is considered an inexact science, it has moved from obscurity to acceptability as a diagnostic tool. Extensive research in this field is required to determine its validity.

5. ACKNOWLEDGMENT

We sincerely thank to acknowledge the Aarupadai Veedu Medical College & Hospital, Vinayaka Mission's Research Foundation (Deemed to be a university), Kirumampakkam, Puducherry-607403, India for their support.

6. AUTHORS CONTRIBUTIONS STATEMENT

Vinothini Gunasekaran designed and did the Systematic review study, Prithiviraj Nagarajan (Corresponding author) wrote the entire manuscript and edited manuscript; Elavarasi Elangovan, John Hearty Deepak Jeyaraj, and Rajan Thangarasu were also

helped in designing the study and reviewed the manuscript. All authors contributed, and accepted the entire manuscript

7. CONFLICT OF INTEREST

Conflict of interest declared none.

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