Palmar Dermatoglyphics and Idiopathic Epilepsy – A Systematic Review

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Abstract

Dermatoglyphics is the science which deals with the study of dermal ridge configuration on the digits, palms and soles. Etymologically this term is harmonious blend of two words Derma – skin; Glyphe – carve. It gives the impression that something has been carved out of the skin. The entire human body is clothed with the skin which happens to be the largest and most important organ of the body. However, the skin on the ventral sides of the hands and the plantar sides of feet is exclusively designed and is corrugated with the ridges and configurations which are functionally useful as they help in the grasping without which the objects would easily slip away from the hands. Dermatoglyphic traits are genetically determined. Dermatoglyphic abnormalities are due to genetic or other factors that express their effect before the end of 5th month of foetal development. The permanency of finger patterns, the extreme variability from one individual to the other and easy analysis are some of the reasons for its wide application in a variety of conditions. Abnormality in the genetic configurations of parents is inherited by children and is reflected in the dermatoglyphic pattern. Hence dermatoglyphic study proves to be a very useful, easily applicable, inexpensive, indispensable tool as an indicator in the diagnosis of hereditary diseases in patients. The etiology of the epilepsies allows a classification of syndrome features into two groups – idiopathic or cryptogenic epilepsy, which has isolated primary symptoms without apparent cause and is probably hereditary and finger print configurations are inherited with an embryonic origin common to nervous system. Their attractions indicate pleiotropic effects of the genotype responsible for enaphalographic irregularity and convulsive seizures.

Key words: *Dermatoglyphics, Epilepsy, Finger prints.*

Literature

Over the past 150 years, dermatoglyphics has been a useful tool in understanding basic questions in biology, medicine, genetics and evolution, in addition to being the best and most widely used method for personal identification.[4] History of Dermatoglyphics. The scientific study of papillary ridges of hands and feet is credited as the beginning with the work of Joannes Evangelista Purkinje in 1823. William Herschel (1858) was the first to experiment with fingerprints in India. He noticed the use of thumb prints as a form of signature amongst illiterate Indians and clearly established the fact that fingerprints did not change their form over time. Sir Francis Galton (1892) published the book "Finger Prints" and in doing so, significantly advanced the science of finger print identification. He demonstrated the epidermal ridge configurations did not change throughout postnatal life. He divided fingertip patterns into three groups Arches, loops and whorls. Sir Edward Henry (1893) established the modern era of finger print identification. Morris Hawthorne Wilder (1902) pioneered comprehensive studies on methodology, inheritance and racial variation of palmar and plantar papillary ridge patterns, as well as fingerprints. Cummins and Midlo (1926) were the first to coin the term Dermatoglyphics. The main thrust of their research was on Down's syndrome and the characteristic hand formations. Charles Midlo MD (1929) together with

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others published "Fingerprints of palms and soles", a bible in the field of dermatoglyphics. Cummins (1935) proposed, that direction of epidermal edges was determined by growth forces and contour of volar skin at the time of ridge formation. Henry (1937) limited the designation of the term 'whorl' to those configurations having ridges that actually encircle a core. He named more complex patterns as 'composite'.[4] Penzose LS (1945) conducted dermatoglyphic investigations of his research into Down's syndrome and other congenital medical disorders. Galton Center (1965) contributed to the development of dermatoglyphics and formulated the measurement to establish the position of displaced axial tri radius in terms of atd angle, as well as establishing the inheritance of its position in the palm. Sarah Holt (1968) published the book "The genetics of dermal edges" and summarized the statistical distributions of dermatoglyphic patterns in both normal and congenitally affected individuals. Hirsch and J.V. Schweichel (1973) emphasized that the neuroepithelium plays an important part in the development of dermatoglyphic patterns.

Schaumann and Alter (1976) published a book "Dermatoglyphic in medical disorders" which summarizes the findings of dermatoglyphic patterns associated with congenital defects and significant markers of prenatal events. Babler (1987) reported that there is a relationship between the volar pad shape and the epidermal ridge configuration. He also suggested the association between the shape of the distal phalanx and the pattern type.[4] Dr. Alexander Rodewald (2001) diagnosed many congenital abnormalities with 90% accuracy from the features of the hands. Dr. Stowens(2003), claims to diagnose schizophrenia and leukemia with 90% accuracy from the patterns on the hands alone. Cummins[2] (1936) reported the association of unusual dermatoglyphics with Down syndrome.The precise configuration of epidermal ridges and miniature is determined at a very early foetal age of around 10 weeks.[5] Volar pads : Volar pads are the site of epidermal ridge development. Volar pads first appear as discrete elevations on the palm around 6.5 weeks post fertilization, followed on digits by apical pads about 1 week later. The volar pads appear over the 2nd, 3rd and 4th inter-digital areas of the palm. Whipple and Cummins noted secondary pads seen on the hands of adult primates. Secondary pad refers primarily to a large elevation on the palm, first seen around 7th week and disappearing during 7th week. Cummins noted the significance of pad regression and initiation of epidermal ridge differentiation. In addition, recent embryologic studies have suggested that plantar, palmar and digital creases develop concurrently with volar pads[12,13] rather than as a consequence of early flexion movements.[14] Development of epidermal ridges: Epidermal ridge morphogenesis has been reported by Kolliper. The critical period of primary ridge differentiation is between 11 and 17 weeks. This finding has been confirmed by Blechschmidt[25], Penrose and O'Hara[26], Okijima[27] and Babler. Epidermal ridges first appear as localized proliferations in the basal layer of the epidermis during the 10th week post fertilization. Primary ridges proliferate rapidly to keep pace with the increasing separation of adjacent ridges due to general growth of hand. This proliferation produces the branching and islands, the miniature. Hale[23] was able to show that the tendency of ridges to multiply was greatest during the period of maximum difference between the increase in surface area of hand and increase in ridge breadth. Primary ridges increase in width and penetrate deeper into the underlying dermis. Secondary ridges or furrow folds correspond to the furrow of surface ridge. Concomitant with secondary ridge formation is the termination of primary ridge formation. From 17 to 24 weeks, secondary ridges continue to proliferate and develop in a manner similar to that of primary ridges. At 24 weeks the epidermal ridge system has an adult morphology. Hirsch and Schweichel reported that ridge formation initiated along the laterodistal portion of the digit and proceeded in a proximomedial direction with the center of pad initially being free of ridges.[5] Factors influencing ridge configuration: Current research suggests that genetic component of dermatoglyphic traits operates indirectly on ridge configuration through ontogenetic factors, eg: pad topography, growth rates and stress on the epidermis that influence ridge alignment. Epidermal ridge growth : From the initial appearance of primary ridges around 11 weeks, prenatal growth of primary ridges can be divided into 3 basic components –

1) Width of the primary ridges
2) Amount of penetration of the ridge into the dermis

3) Spacing or separation between adjacent primary ridges. Dermatoglyphic patterns form on finger pads prenatally and remain unchanged throughout the life. It can aid to the diagnosis of genetically and nongenetically determined diseases which cause distortion of patterns. Recently Penrose and O'Hara used electron microscopy to study the epidermal ridge development. Okijima and Miller reported the techniques to study surface ridges of the fetus.[6] Personal identification : Finger prints are constant and individualistic and can be the most reliable measure for personal identification. The secretions in the finger print contain residues and various chemicals and can be detected and used for forensic purpose and for criminal identification. It is important in medicolegal cases and disputed paternity positive identification using finger print can be established only if 16 – 20 points of similarity exist in the miniature. Concerning the distribution and degree of heritability of palmar main lines, it should be remembered that the differentiation of dermatoglyphics occurs during 3rd and 4th month of foetal life and that the lines remain constant after birth. Therefore except in size, the dermal configurations do not vary with age and are practically independent of environment effect.[9] Line A is considered to be an important indicator of the general direction of ridges coursing over a large area of palm (Wolt, Bsehane and Reinvein 1964). A-d ridge count has been used in pedigree studies. The mean count on left hand was significantly larger than on the right by approximately nine ridge ($p<.01$). This bilateral asymmetry of line A was evident in both sexes. (Cummins and Midlio 1961) Genes of additive effect ailing without dominance would be expected to give correlation coefficients of 0.5 for both parent child and sib-sib comparison (Fisher 1918). The theoretical correlation coefficient for child and midparent is 0.71 (Penrose 1949).

Calculation of the correlation coefficient between relatives for both the total finger ridge count (Holt 1961) and the a-b ridge count (Pons, 1964) have yielded values in close agreement with those expected on theoretical grounds for polymeric systems with genes of additive effect. The A-d ridge count appears to resemble the a-b and finger ridge count in that variation in this feature is largely under genetic control and subject to the influence of genes with additive effect.[10] However conclusions are still contradictory. The results suggest a significant familial correlation (except spouse) indicating the involvement of familial component to the variation of dermatoglyphic traits. Segregation analysis reveals the transmission of genetic effects in the families which follows the Mendalian model. Major gene involvement with Mendalian expectation regarding finger dermatoglyphics is confirmed for all analyzed traits. However there is no evidence of significant support for major gene affect or environment effect on palmar a-b ridge count.[11]

The history of dactylography as a fool proof tool for identity establishment has special significance in India – the first finger print bureau, established for medicolegal purpose in the world was at Calcutta. Today, digital dermatoglyphics form an indispensable and reliable tool for criminal investigation at internal level. Forensic scientists could extend their expertise and experience in dermatoglyphics to aid clinicians in medical diagnostic investigation.[13]

Epilepsy is a chronic brain disturbance of varied and complex origin. Epileptic syndromes are classified mainly by brain location (generalized, partial or focal) and seizure type (clinic, tonic akinetic simple, absence or unconsciousness) which, together with electro-encephalographic studies, define the differential diagnostic (Delgado-Escueta et al 1982, Lancet 1990).[16] The etiology of the epilepsies allows a classification of syndrome features into two groups – idiopathic or cryptogenic epilepsy, which has isolated primary symptoms without apparent cause and is probably hereditary. Second group is symptomatic epilepsy of genetic or acquired origin associated with other clinical features. Symptomatic epilepsy is due to cranial traumatism or as a result of infection, intoxication, brain tumours, hypoglycemia, drug abstinence etc. Jacksonian Seizures displayed only molar manifestation without impairment of consciousness and are typical among focal epilepsies. The genetic investigation carried out by Wenberg (1912) is of special historic interest because it was the first statistical segregational analysis of a human pathological condition. (McKusick1992). Research on genetics of various epileptic syndromes has been reported and models designed for analysis of its familial segregation. (Anderson et al 1982) Isolated

idiopathic epilepsies with Mendelian inheritance do not encompass 5% of hereditary convulsive syndromes (Anderson and Hauser 1993) which are predominantly associated with chromosomic aberrations, metabolic disturbances and neurological deficiency. Other genetic molecular approaches involve neurotransmitters, inhibition of gamma amino butyric acid (GABA) synthesis in gerbils (Gerbillus) susceptible to epileptogenic factors (Fukuyama et al 1979) produced convulsive activity of similar origin to human epilepsy in the temporal lobe. (Osolsen et al 1984) Fere (1905) may have done the pioneer study on finger prints in epileptics. Many investigators reported several studies of finger prints in individuals affected by non-specific types of epilepsy (Portius 1937; Brown and Paskind 1940; Katzenstein – Sutro 1945; Cherrill 1950; Alter 1966; Rosner et al 1967; Razavi 1975; Lopez and Lopez 1977; Karitonov et al 1979; Schaumann and Mayerdorf 1979; Schaumann et al 1982; Cisarik et al 1985; Marinina and Drabktsina, 1988). Results revealed significant differences in loop frequencies in the left hand among male and female whole subjects . Considering separately each finger tip, the comparison between epileptic groups exhibited highly significant differences ($p<0.01$) in loop frequencies in right and in the left finger, the values being higher among generalized epileptics. The largest statistical differences were detected in finger III & I between epileptics and control. This suggests an epigenetic connection between embryonic regions I – III and normal physiology of CNS. Schaumann and Mayersdorf (1979) found an increase in radial loops in white adult patients with idiopathic epilepsy. In the non white group, the tented arch (At) pattern was completely absent in male generalized epilepsy and their control.[16] Previous research among adult males with idiopathic epilepsy showed lower TRC values, although not significant at the 5% level of probability, comparatively to the controls (Schaumann and Mayersdorf 1979, Schaumann et al 1982).[16] As in nearly all populations (Saldanha 1968), the TRC means were significantly higher ($p<0.05$) in male than in female groups for both epileptics and controls.[16] The high evidence of arches on I & IV fingers, of raketoid loops on all fingers and especially IV & V, of the papillary ridges disposition as a dense and very dense network at thenar / I of the finalization of the line T's direction of palm's field 11 and 12 instead of 13 which contribute to enriching the clinical image of epilepsy's dermatoglyphic diagnosis, at least for the patients living in Maldivian.[18]

It has been opined that any epidermal ridge alteration in individuals prone to epilepsy may have a distinctive dermtoglyphic feature (Schaumann & Alter 1976). Idiopathic epilepsy of primary generalized epilepsy type is a tendency to have seizures when there is no structural abnormality in the brain. The primary cause could be genetic and a number of genes have been mapped Baulac et al (2001)3 and Brismar (2000)[4] The dermatoglyphic traits, which presented a significant difference, were a-b ridge count, lateral deviation, palmar pattern and finger tip pattern. Mean values of a-b ridge count were more in epileptic patients, especially in left hand, than controls. The ratio of ulnar and radial deviations in control was 1:3 while in epileptics it was 1:5. Arch type of palmar patterns were showing a very significant difference between controls and cases. Frequency of loops was much more and vestiges were absolutely absent in cases. The frequency of Arch type of finger tip pattern was more in controls.[17] A first and very important deviation at the level of affected people's palm is a very strong reduction of models frequency in interdigital spaces IV & III. The strong decrease of distance a-b much below the average value was recorded in Romanian population. A higher frequency of cases was observed in which the papillary ridges from Thenar / I are arranged as a dense or very dense network.[20]

Abnormalities of early development including genetic disorders. On literature review, it was noticed that hardly few studies have reported the association between dermatoglyphics and idiopathic epilepsy. Authors have found two significant variables in males diagnosed with idiopathic epilepsy. (i) decreased 'a-b' ridge count on both palms ($p<0.1$) and (ii) more frequent existence of transverse suclus. The mean age of onset of epilepsy was 15.8 years in males and 13.9 years in females. 18.4% of the male patients and 10.2% of female patients were products of consanguineous marriage; 12% of male and 22% of female patients had family history of epilepsy. Various studies have shown different variations in the qualitative and quantitative parameters of palmar dermatoglyphics in Epilepsy patients.

Priya Ranganatha found no significant difference in the a-b ridge count between patients and controls in males and females. Difference in 'atd' angle between patients and controls in males and females was not found to be significant.[19] A Study on finger tip patterns on comparison of epileptics with controls, in males, with hands combined, loops and arches were increased and whorls were decreased ($p<0.05$). In females, with hands combined, arches and whorls were increased and loops were decreased ($p<0.03$). Significant differences have not been observed for the patterns in hypothenar area / interdigital area and flexion creases.[21] The authors distinguished correlation between dermatoglyphic features and form of epilepsy, type of course and pathogenic forms.[22] Dermatoglyphics characteristic in epileptic patients varied in groups with different clinical characteristics (age at the onset of disease and the duration of the latter, the daily development of paroxysms at the same time, a tendency toward a stable course, the presence of psychic states and the resistance to therapy). Statistical task solving rules for diagnosing epilepsy in children were elaborated. The correct computer aided diagnosis on the basis of dermatoglyphic examination was made in 70% of epileptic patients.[23] A dermatoglyphic study of adult Caucasian males with a confirmed diagnosis of epilepsy was carried out. A multivariant analysis was employed on an enlarged patient sample. Variables found to be significant were decreased a-b ridge count on both left and right palms ($p<0.001$). Tests of eigenvalues showed only one value to be significant and accounting for 71.8% of the intergroup variation.24 The data are important for medical genetic consultation and provide information for the theory of genetic issues in epilepsy.[25] Significant findings in qualitative analysis of male epileptic patients showed increase in frequency of radial loops on finger tips of right and left index fingers and decrease in frequency of whorls on right thumb, index, ring and left ring fingers. In female epileptics there was increase of inter-digital fourth pattern VIII in both hands. Significant findings in quantitative analysis of epileptic patients included increase of total finger ridge count in male and female epileptics.28 Authors reported a k289m mutation in GABAA receptor γ2-subunit gene (GARG2) that segregates in a family with a phenotype closely related to GEFS+, an autosomal dominant disorder associating febrile seizures and generalized epilepsy previously linked to mutations in sodium channel genes, thus providing the first genetic evidence that a GABAA receptor is directly involved in human idiopathic epilepsy.[29] The comparison of palmar pattern types revealed significant difference between symptomatic and controls for right second inter digital area and left hypothenar area. Digital pattern intensity index was higher in epileptics than controls.[31] The significantly increased prevalence of dermatoglyphic abnormalities in epileptics and in their parents suggests a polygenic mode of inheritance. Thus the view that convulsions are inherited by a single dominant gene with incomplete penetrance needs to be reexamined.[32]

Conclusion

One of the most important parameter of dermatoglyphics is the inheritance. Understanding of development of dermatoglyphic traits requires to view it as a living history of prenatal development. Hence it is important in the medicolegal cases of disputed paternity. It has very crucial role in the diagnosis of Monozygotic and dizygotic twins. Degree of affection of investigated epileptics is correlated with the presence in the palmar dermatoglyphics of significant variations.

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