Role of dermatoglyphics as an indicator of precancerous and cancerous lesions of the oral cavity

Ambika Gupta, Freny R. Karjodkar

Abstract

Background: Oral squamous cell carcinoma (SCC) is one name that causes panic and holds an undeserved high ranking as a killer. Another important condition which has become a major public health issue in South East Asia is oral submucous fibrosis (OSF). Not all the people using tobacco suffer from these diseases. Genetic predisposition might explain such an individual variability that can be predicted by using various cytogenetic markers. However, these studies are far more costly and complicated. So, dermatoglyphics may be of immense clinical significance to segregate those individuals who are at an increased risk for developing these diseases. Aim: The present study was conducted to analyze the palmar dermatoglyphics in SCC and OSF and find a "dermatoglyphic marker", if any. Study Design: Cross sectional study. Materials and Methods: 120 individuals were divided into four groups based upon their habits of tobacco/areca nut usage and presence of OSF/SCC. Dermatoglyphic patterns were recorded using standard ink method. Various patterns were analysed statistically in the four groups. Results and Conclusion: In SCC, there was an increase in frequency of arch and ulnar loop patterns on fingertips, decrease in frequency of simple whorl patterns on fingertips, decrease in frequency of palmar accessory triradii on right and left hands. Significant findings in OSF included an increase in frequency of arch and ulnar loop pattern, decrease in frequency of simple whorl patterns on fingertips, decrease in atd angle on right hand, decrease in frequency of palmar accessory triradii on right hand. The results revealed that the field of dermatoglyphics holds promising results for determining the genetic susceptibility of individuals to develop SCC and OSF.

Keywords: Finger prints, genetic marker, oral squamous cell carcinoma, oral submucous fibrosis

Introduction

Since the early days of civilization, the features of the hands have fascinated scholars, doctors, and laymen alike. Through decades of scientific research, the hand has come to be recognized as a powerful tool in the diagnosis of psychological, medical, and genetic conditions. Cummins in 1926 first introduced the term “dermatoglyphics” which refers to the study of the naturally occurring patterns of the surface of the hands and feet.[1] Since then, this approach has been used in various scientific studies to establish relationship of fingerprints as genetic and/or chronic health markers. Dermatoglyphic patterns are genetically determined and remain unchanged from birth to death. Dermatoglyphics is considered as a window of congenital and intrauterine abnormalities. At present, several researches claim this study of dermatoglyphics as an important diagnostic tool for some diseases especially the diseases with obscure etiology and mysterious pathogenesis. Significant investigations have also been carried out into the dermatoglyphic indicators of Down’s syndrome, Trisomy 18, D trisomy, cat cry syndrome, Turner’s syndrome, Klinefelter’s syndrome, congenital heart disease, leukemia, cancer, celiac disease, intestinal disorders, rubella, rheumatoid arthritis, bronchial asthma, Alzheimer’s disease, schizophrenia as well as other forms of mental illness.[2-17] In dentistry, dermatoglyphics have been studied to help predict disorders like cleft lip and cleft palate, dental caries, malocclusion, congenital anomalies like ectodermal dysplasia, gingival fibromatosis, periodontitis, bruxism etc.[18-28]

So, the present study was carried out to analyze the palmar dermatoglyphics an squamous cell carcinoma (SCC) and oral submucous fibrosis (OSF) and to compare the patterns of these diseases with the control group.

Materials and Methods

The present cross sectional study was carried out on the male patients who attended the outpatient Department of Oral Medicine and Radiology, Nair Hospital Dental College for Oral and Maxillofacial Examination. 120 individuals were divided into four groups. Group 1 consisted of 30 male patients with history of tobacco/areca nut intake with occurrence of oral
SCC. Group 2 had 30 male patients with history of tobacco/areca nut intake with occurrence of OSF. In group 3, 30 males with habit of tobacco/areca nut, without any evidence of oral lesions were taken while the forth group had 30 males without any habit, and without any oral lesions, that served as control.

Selection criteria were male patients giving positive history of tobacco/areca nut chewing for more than 1 year, with or without use of tobacco in other forms, presence of ulcerated lesion, red and white patch or exophytic growth on oral mucosa for group 1, restricted oral opening with palpable fibrous bands and/or burning sensation of mucosa for group 2. All the cases were confirmed by histopathological examination. Exclusion criteria was the presence of oral lesions due to other causes like sharp tooth margins, improper restorations, prosthesis, alcohol or smoking, patients with scars or any injury to palms and patients with any systemic diseases.

Patients were informed in detail about the study and their informed consent was obtained to conduct the study. A structured format was designed, which consisted of demographic data, detailed history of habits, medical history, family history, clinical examination, diagnosis and histopathology report. Subjects were asked to wash their hands with soap and water to remove any dirt or oil. Palmer prints were taken by using standard ink method proposed by Strong, using black duplicating ink (Kores India Limited, Mumbai), Thick white printing paper (Berga image, A4 size, 100 g/m²), roller, glass inking slab and sponge pad.[29] The finger and palmer prints were analyzed qualitatively and quantitatively using Cummins, Mildo and Penrose method.[29,30,31] Various parameters studied were fingerprint patterns, palmar patterns, total finger ridge counts, a-b ridge counts, atd angle and accessory triradii.

Statistical Evaluation

Qualitative analysis

Fingertip prints patterns

Fingertip print patterns were categorized into five types - arches, ulnar loops, radial loops, true whorls (W⁰) and composite whorls. In order to find out the frequency of fingertip print patterns in both hands, all ten fingers of an individual were considered together. Frequency of each pattern was recorded in each individual and the percentage of pattern frequency was calculated for the entire group. The values of the four groups were compared and statistical differences were calculated.

Palmar patterns

Palmar patterns were observed in different areas of palms such as hypothenar (Hy), thenar/interdigital 1 (Th₁), area, interdigital 2, 3 and 4 (I₂,3,4) areas. Various patterns encountered in both hands were noted. The frequency of palmar patterns in the above mentioned areas was calculated in both hands separately and comparison was made between the study groups.

Frequency of prevalence of accessory triradii

Frequency of accessory triradii (a’, b’, c’, d’) on each palm of the subjects and controls was recorded and the percentage of prevalence for each group was calculated. The values for the four groups were evaluated using Chi-square test.

Quantitative analysis

Total finger ridge count was calculated for all ten fingers, by taking its mean. Total number of ridges between triradii “a” and “b” (a-b ridge count) were recorded in both hands. atd angles were measured in both hands. Their means were taken on both hands separately and the differences in the means were analyzed for the four study groups.

Separate tables were prepared for individual dermatoglyphic parameters observed. ‘p’ value was calculated and results obtained were tested for statistical significance. For qualitative data, Chi-square test was used and P value was calculated at 95% confidence levels. For quantitative data, one way analysis of variance test was used and the difference in means of each of the two groups within the four groups were analysed by applying Scheffe’s test. All the above analyses were performed using statistical package SPSS 10.01 version and StatCalc (World Health Organization – Epi info 3.3.2 version).

Results

The demographic data of fingerprint patterns in the study groups is described in Table 1. There was a significant increase in the arch and ulnar loop pattern frequency. About 33.3% of patients with SCC, 23.4% patients with OSF and 26.7% controls with habit had arch pattern in at least one finger as compared to only 13.4% controls without habits. 50% of patients with SCC and OSF had ulnar loop on more than seven fingers. This percentage was only 33.3% in controls with habit and 36.6% in controls without habit. W⁰ were significantly decreased in SCC (n = 95) and OSF (n = 101) as compared to control groups with and without habits (χ² = 15.890, P < 0.001). 56.6% controls without habit had this pattern on more than four fingers, while only 30% of carcinoma patients had W⁰ on more than four fingers. There is only a slight variation in the frequency of radial loops and compound whorls in the four groups.

Analysis of total frequency of ridge count (TFRC) in our study revealed that the mean value of TFRC in OSF group was lower than the other groups studied. But, the values were not found to be significant [Tables 2a and b]. The total ridge count between palmar triradii ‘a’ and ‘b’ and frequency of palmar patterns in hypothenar area, Th₁, I₂,3,4 areas were not significantly variable. Thus, no dermatoglyphic marker for palmar pattern was found in any of the study groups [Graphs 1a and b].
The angle between triradii a, t/t' and d (atd angle) was measured on right and left hands separately. The mean values are summarized in Tables 3a and b, 4a and b. The difference in the mean values of right hand of patients with OSF and control group without habit was statistically significant ($P = 0.013$) [Tables 3a and b, 4a and b].

The number of accessory palmar triradii on right and left hands of the samples in each group were observed. There was statistically significant difference in palmar triradii frequency. The differences were significant on comparison between SCC and OSF ($\chi^2 = 5.455, P < 0.05$ in right hand and $\chi^2 = 4.02, P < 0.05$ in left hand) and between SCC and control without habit ($\chi^2 = 13.871, P < 0.01$) [Tables 5a and b].

**Discussion**

SCC is a widespread disease associated with considerable amount of morbidity and mortality. It is a major worldwide health problem and the number of sufferers is increasing rapidly due to more and more people embracing deleterious habits such as tobacco chewing, smoking and alcohol abuse. Although the etiology is multifactorial, but regardless of the accelerating factors, neoplasm is thought to arise clonally from transformed cells that have undergone specific genetic and epigenetic alterations in oncogenes or tumor-suppressor genes. Many gene alterations have been implicated in the development and progression of SCC and the stages of carcinogenesis have been clearly defined. An increased risk of oral cancer is associated with a number of inherited cancer syndromes, including Li-Fraumeni, Fanconi’s anaemia and xeroderma pigmentosum. Some studies have suggested that there is an inherited component to sporadic oral cancer. First-degree relatives of people with oral cancer have been reported to be at greater risk of developing the disease. Those with an inherited susceptibility may be more likely to develop multiple primary tumours. Similarly OSF is a widespread precancerous condition especially prevalent in South East Asia. Areca nut is an important predisposing factor, but not all the patients with chronic habits suffer from the disease. Conversely, not all the patients with OSF have a prolonged history of areca nut or tobacco consumption. It is said that genetic susceptibility is responsible for such variations.

The dermal ridges have various notable characteristics which make them important, not only in personal identification, but also in human biology for various reasons. Firstly, unlike many bodily traits the dermal ridges and configuration once formed remain unchanged except in dimensions, i.e. they are age stable. The ridges are environment stable and begin 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. The dermatoglyphic features can thus be exploited quantitatively and qualitatively to be used as “genetic marker” of a disorder. At present, there is an agreement that dermatoglyphic features confirm to polygenic system, with individual genes contributing a small additive effect.
Various epidemiological studies support the fact that genetic alterations may be involved in the pathogenesis of SCC and OSF. These antenatal disturbances can alter the epithelium to make it susceptible to various carcinogens. The present study was carried out assuming the hypothesis that any such antenatal disturbance, if responsible for a disorder, should manifest in a prenatal event such as dermal ridge formation.

Hardly any studies are mentioned in the reviewed literature, on the use of dermatoglyphics as a marker for SCC and OSF. Veena, Humbarwadi, Potturi found a decreased atd angle, increase patterns in Th/I area and increased pattern frequency in I area in OSF patients as compared to normal gutkha chewers. The decrease in atd angle was the only common observation in OSF group in our study. As, no other studies were found in the literature, it is difficult for us to compare our results with studies done in other populations. In various studies done on breast carcinoma, presence of six or more whorls on fingertips and decreased ridge count in cases were found to be an important “dermatoglyphic marker” for screening of population at higher risk. In yet another study done on thyroid carcinomas, thyroid cancer subjects had a lower total digital ridge count and a reduced number of papillary ridges between the a-d triad, than the control subjects. Qualitative examination showed more patterns in the second interdigital area.

### Table 2a: Comparison of mean TFRC in different study groups (using one way ANOVA)

| Study groups | Mean | SD  | SE  | Sum of squares | df | Mean square | F   | P value |
|--------------|------|-----|-----|---------------|----|-------------|-----|---------|
| 1            | 145.7| 45.6| 8.3 | 12110.89      | 3  | 4036.96     | 2.68| 0.05*   |
| 2            | 119.8| 33.5| 6.1 |               |    |             |     |         |
| 3            | 139.9| 36.6| 6.7 |               |    |             |     |         |
| 4            | 141.9| 38.4| 7.0 |               |    |             |     |         |
| Total        | 136.8| 39.6| 3.6 |               |    |             |     |         |

*Statistically significant. TFRC: Total frequency of ridge count; ANOVA: Analysis of variance; SD: Standard deviation; SE: Standard error

### Table 2b: Scheffe’s test applied

| Group comparison | Mean difference | SE   | P value | Significance |
|------------------|-----------------|------|---------|--------------|
| Group 1          |                 |      |         |              |
| Group 2          | 25.90           | 10.01| 0.088   | Not significant |
| Group 3          | 5.77            | 10.01| 0.954   | Not significant |
| Group 4          | 3.83            | 10.01| 0.986   | Not significant |
| Group 2          |                 |      |         |              |
| Group 1          | −25.90          | 10.01| 0.088   | Not significant |
| Group 3          | −20.13          | 10.01| 0.262   | Not significant |
| Group 4          | −22.07          | 10.01| 0.189   | Not significant |
| Group 3          |                 |      |         |              |
| Group 1          | −5.77           | 10.01| 0.954   | Not significant |
| Group 2          | 20.13           | 10.01| 0.262   | Not significant |
| Group 4          | −1.93           | 10.01| 0.998   | Not significant |
| Group 4          |                 |      |         |              |
| Group 1          | −3.83           | 10.01| 0.986   | Not significant |
| Group 2          | 22.07           | 10.01| 0.189   | Not significant |
| Group 3          | 1.93            | 10.01| 0.998   | Not significant |

SE: Standard error

### Table 3a: Comparison of mean atd angle in the right hand in different study groups (using one way ANOVA)

| Study groups | Mean  | SD   | SE   | Sum of squares | df  | Mean square | F   | P value |
|--------------|-------|------|------|---------------|-----|-------------|-----|---------|
| 1            | 42.07 | 6.35 | 1.16 | 318.167       | 3   | 106.056     | 3.898| 0.011*  |
| 2            | 39.50 | 4.70 | 0.86 |               |     |             |     |         |
| 3            | 41.27 | 4.49 | 0.82 |               |     |             |     |         |
| 4            | 44.03 | 5.12 | 0.94 |               |     |             |     |         |
| Total        | 41.72 | 5.40 | 0.49 |               |     |             |     |         |

*Statistically significant. ANOVA: Analysis of variance; SE: Standard error; SD: Standard deviation

### Table 3b: Comparison of difference in mean at dangle in the right hand between different study groups (using Scheffe’s test)

| Group comparison | Mean difference | SE   | P value | Significance |
|------------------|-----------------|------|---------|--------------|
| Group 1          |                 |      |         |              |
| Group 2          | 2.57            | 1.35 | 0.309   | Not significant |
| Group 3          | 0.80            | 1.35 | 0.950   | Not significant |
| Group 4          | −1.97           | 1.35 | 0.547   | Not significant |
| Group 2          |                 |      |         |              |
| Group 1          | −2.57           | 1.35 | 0.309   | Not significant |
| Group 3          | −1.77           | 1.35 | 0.634   | Not significant |
| Group 4          | −4.53           | 1.35 | 0.013*  | Significant |
| Group 3          |                 |      |         |              |
| Group 1          | −0.80           | 1.35 | 0.950   | Not significant |
| Group 2          | 1.77            | 1.35 | 0.634   | Not significant |
| Group 4          | −2.77           | 1.35 | 0.244   | Not significant |
| Group 4          |                 |      |         |              |
| Group 1          | 1.97            | 1.35 | 0.547   | Not significant |
| Group 2          | 4.53            | 1.35 | 0.013*  | Significant |
| Group 3          | 2.77            | 1.35 | 0.244   | Not significant |

SE: Standard error

### Table 4a: comparison of mean atd angle in the left hand in different study groups (using one way ANOVA)

| Study groups | Mean  | SD   | SE   | Sum of squares | df  | Mean square | F   | P value |
|--------------|-------|------|------|---------------|-----|-------------|-----|---------|
| 1            | 41.90 | 5.45 | 0.99 | 232.367       | 3   | 77.456      | 2.748| 0.046*  |
| 2            | 40.70 | 5.57 | 1.02 |               |     |             |     |         |
| 3            | 40.97 | 3.87 | 0.71 |               |     |             |     |         |
| 4            | 44.23 | 6.08 | 1.11 |               |     |             |     |         |
| Total        | 41.95 | 5.42 | 0.50 |               |     |             |     |         |

*Statistically significant. ANOVA: Analysis of variance; SE: Standard error; SD: Standard deviation
The present study on dermatoglyphic patterns of patients with SCC and OSF revealed some significant parameters which may be used as “dermatoglyphic markers”. On comparing the intergroup finding the following positive parameters were observed in SCC - Increase in frequency of arch and ulnar loop patterns on fingertips, decrease in frequency of simple whorl patterns on fingertips, decrease in frequency of palmar accessory triradii on right and left hands. Significant findings in OSF included - Increase in frequency of arch and ulnar loop pattern, decrease in frequency of simple whorl patterns on fingertips, Decrease in atd angle on right hand, decrease in frequency of palmar accessory triradii on right hand.

**Conclusion**

The field of dermatoglyphics holds promising results for determining the genetic susceptibility of individuals to develop SCC and OSF. But, further multicentric studies must be conducted in larger population with age, sex, religion and race matched controls. The studies may also be carried out to compare the

### Table 4b: Comparison of difference in mean atd angle in the left hand between different study groups (using Scheffe’s test)

| Group comparison | Mean difference | SE  | P value  | Significance |
|------------------|----------------|-----|----------|--------------|
| Group 1          |                |     |          |              |
| Group 2          | 1.20           | 1.37| 0.857    | Not significant |
| Group 3          | 0.93           | 1.37| 0.927    | Not significant |
| Group 4          | -2.33          | 1.37| 0.411    | Not significant |
| Group 2          |                |     |          |              |
| Group 1          | -1.20          | 1.37| 0.857    | Not significant |
| Group 3          | -0.27          | 1.37| 0.998    | Not significant |
| Group 4          | -3.53          | 1.37| 0.090    | Not significant |
| Group 3          |                |     |          |              |
| Group 1          | -0.93          | 1.37| 0.927    | Not significant |
| Group 2          | 0.27           | 1.37| 0.998    | Not significant |
| Group 4          | -3.27          | 1.37| 0.135    | Not significant |
| Group 4          |                |     |          |              |
| Group 1          | 2.33           | 1.37| 0.411    | Not significant |
| Group 2          | 3.53           | 1.37| 0.090    | Not significant |
| Group 3          | 3.27           | 1.37| 0.135    | Not significant |

SE: Standard error

### Table 5a: Comparison of prevalence of accessory triradii of the right hand between the four study groups

| Study groups | Yes | No | Total |
|--------------|-----|----|-------|
|              | N   | %  | N     | %    | N    | %    |
| 1            | 2   | 6.7| 28    | 93.3 | 30   | 100  |
| 2            | 9   | 30.0| 21    | 70.0 | 30   | 100  |
| 3            | 6   | 20.0| 24    | 80.0 | 30   | 100  |
| 4            | 15  | 50.0| 15    | 50.0 | 30   | 100  |
| Total        | 32  | 26.7| 88    | 73.3 | 120  | 100  |

| χ² | df | P value | Significance |
|----|----|---------|--------------|
| 15.34 | 3 | <0.01 | Highly significant |
| 5.455 | 3 | <0.01 | Highly significant |
| 2.308 | 3 | >0.05 | Not significant |
| 13.871 | 3 | <0.01 | Highly significant |
| 0.800 | 3 | >0.05 | Not significant |
| 2.500 | 3 | >0.05 | Not significant |
| 5.934 | 3 | <0.05 | Significant |

### Table 5b: Comparison of prevalence of accessory triradii of the left hand between the four study groups

| Study groups | Yes | No | Total |
|--------------|-----|----|-------|
|              | N   | %  | N     | %    | N    | %    |
| 1            | 5   | 16.7| 25    | 83.3 | 30   | 100  |
| 2            | 12  | 40.0| 18    | 60.0 | 30   | 100  |
| 3            | 7   | 23.3| 23    | 76.7 | 30   | 100  |
| 4            | 9   | 30.0| 21    | 70.0 | 30   | 100  |
| Total        | 33  | 27.5| 87    | 72.5 | 120  | 100  |

| χ² | df | P value | Significance |
|----|----|---------|--------------|
| 4.472 | 3 | >0.05 | Not significant |
| 4.02 | 3 | <0.05 | Significant |
| 0.2109 | 3 | >0.05 | Not significant |
| 0.00179 | 3 | >0.05 | Not significant |
| 1.926 | 3 | >0.05 | Not significant |
| 0.6593 | 3 | >0.05 | Not significant |
| 0.3409 | 3 | >0.05 | Not significant |
findings with those of parents of the patients suffering from precancers and cancers. For carcinoma, more studies are needed to compare the findings in cancers originating in different sites and locations in the body. Also, similar studies can be carried out to compare the premalignant lesions with carcinoma. With the help of these parameters, probably the genetically predisposed individuals can be segregated amongst the population at risk and can be appropriately counselled and motivated to change the lifestyle. Also, the cost burden associated with genetic cytmarkers studies may be prevented. Thus, with the help of simple measurements, the frequency of developing dreadful diseases in later life may be prevented.

References

1. Cummins H, Midlo C. Fingerprints, Palms and Soles: An Introduction to Dermatoglyphics. New York: Dover Press; 1961. p. 319.
2. Miller JR, Giroux J. Dermatoglyphics in pediatric practice. J Pediatr 1966;69:302-12.
3. Bukelo MJ, Kanchan T, Rau AT, Unnikrishnan B, Bukelo MF, Krishna VN. Palmar dermatoglyphics in children with acute lymphoblastic leukemia – A preliminary investigation. J Forensic Leg Med 2011;18:115-8.
4. Ponnudurai R. Relevance of sequential development of dermatoglyphics to schizophrenia. Psychiatry Res 1999;89:59-67.
5. Stough TR, Seely JR. Dermatoglyphics in medicine. Clin Pediatr (Phil) 1969;8:32-41.
6. Alter M. Dermatoglyphic analysis as a diagnostic tool. Medicine (Baltimore) 1967;46:35-56.
7. Holt SB. The significance of dermatoglyphics in medicine. A short survey and summary. Clin Pediatr (Phil) 1973;12:471-84.
8. Godfrey KM, Barker DJ, Peace J, Cloke J, Osmond C. Relation of fingerprints and shape of the palm to fetal growth and adult blood pressure. BMJ 1993;307:405-9.
9. Alter M, Schulenberg R. Dermatoglyphics in congenital heart disease. Circulation 1970;41:49-54.
10. Elsaaadany HM, Kassem E, El-Sergy M, Sheta AR. Can dermatoglyphics be used as an anatomical marker in Egyptian rheumatoid patients? J Am Sci 2010;6:457-66.
11. Vera M, Cabrera E, Guell R. Dermatoglyphics in insulin-dependent diabetic patients with limited joint mobility. Acta Diabetol 1995;32:78-81.
12. Rajanigandha V, Mangala P, Latha P, Vasudha S. Digits-palmar complex in non-insulin dependent diabetes mellitus. Turk J Med Sci 2006;36:353-5.
13. Stevenson CJ, West CR, Pharaoh PO. Dermatoglyphic patterns, very low birth weight, and blood pressure in adolescence. Arch Dis Child Fetal Neonatal Ed 2001;86:F18-22.
14. Vishwanathan G, Krishnan M, Kalyani GS. Analysis of finger tip dermatoglyphics of tuberculosis patients. Journal of Eobiology 2002;14:205-10.
15. Ozkaragöz K, Atasu M, Saraclar Y. A preliminary study on dermatoglyphics in children with bronchial asthma. A report on 84 asthmatic children. J Asthma Res 1971;8:179-82.
16. Natekar PE, Shukla P, Priolkar S. Axial triradii in leprosy. J Aviat Secur Int 1996;45:105-9.
17. Weizman Z, Vardi O, Binsztok M. Dermatoglyphic (fingerprint) patterns in celiac disease. J Pediatr Gastroenterol Nutr 1990;10:451-3.
18. Kanematsu N, Yoshida Y, Kishi N, Kawata K, Kaku M, Maeda K, et al. Study on abnormalities in the appearance of finger and palm prints in children with cleft lip, alveolus, and palate. J Maxillofac Surg 1986;14:74-82.
19. Balgir RS, Mitra S. Congenital cleft lip and cleft palate anomalies: A dermatoglyphic study. J Postgrad Med 1986;32:18-23.
20. Mathew L, Hegde AM, Rai K. Dermatoglyphic peculiarities in children with oral clefts. J Indian Soc Pedod Prev Dent 2005;23:179-82.
21. Atasu M. Dermatoglyphic findings in dental caries: A preliminary report. J Clin Pediatr Dent 1998;22:147-9.
22. Reddy S, Prabhakar AR, Reddy VV. A dermatoglyphic predictive and comparative study of Class I, Class II, div. 1, div. 2 and Class III malocclusions. J Indian Soc Pedod Prev Dent 1997;15:13-9.
23. Lin YC, Miyazono H, Ichinose M, Nakasima A. A study to evaluate the parent-offspring similarity in the maxillofacial profile using dermatoglyphs in Japanese families. J Craniofac Genet Dev Biol 1998;18:119-27.
24. Kargül B, Alcan T, Kabalay U, Atasu M. Hypohidrotic ectodermal dysplasia: Dental, clinical, genetic and dermatoglyphic findings of three cases. J Clin Pediatr Dent 2001;26:5-12.
25. Atasu M, Akyuz S. Congenital hypodontia: A pedigree and dermatoglyphic study. J Clin Pediatr Dent 1995;19:215-24.
26. Skrinjaric I, Bacic M. Hereditary gingival fibromatosis: Report on three families and dermatoglyphic analysis. J Periodontal Res 1989;24:303-9.
27. Yilmaz S, Atasu M, Kuru B. A genetic and dermatoglyphic study on periodontitis. J Marmara Univ Dent Fac 1993;1:297-306.
28. Polat MH, Azak A, Evlioglu G, Malkondu OK, Atasu M. The relation of bruxism and dermatoglyphics. J Clin Pediatr Dent 2000;24:191-4.
29. Strong AM. An improved method of palm-printing. Science 1929;69:250-1.
30. Penrose LS. Memorandum on dermatoglyphic nomenclature. Birth Defects Orig Artic Ser 1969;6:72-84.
31. Penrose LS. Fingerprints and palmistry. Lancet 1973;1:1239-42.
32. Veena HS, Humbarwadi RS, Potturi BR. Abstracts. J Anat Soc India 2005;54:251-300.
33. Seltzzer MH, Plato CC, Fox KM. Dermatoglyphics in the identification of women either with or at risk for breast cancer. Am J Med Genet 1990;37:482-8.
34. Abbasi S, Enoollahi N, Vaez-Zadeh F. Study of dermatoglyphic patterns of hands in women with breast cancer. Pak J Med Sci 2006;22:18-22.
35. Chintamani, Khandelwal R, Mittal A, Saijanani S, Tuteja A, Bansal A, et al. Qualitative and quantitative dermatoglyphic traits in patients with breast cancer: A prospective clinical study. BMC Cancer 2007;7:44.
36. Ciovîrnache M, Dumitriu L, Moguş I, Spandonide T, Damian A, Handoca A, et al. Dermatoglyphics in thyroid cancer. Endocrinologie 1986;24:171-83.

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