Dermatoglyphics in Patients with Dental Caries: A Study on 1250 Individuals

PR Abhilash, R Divyashree, Shankar Gouda Patil, Mohit Gupta, T Chandrasekar, R Karthikeyan

ABSTRACT

Aim: This study was undertaken to investigate and analyze the significance of dermatoglyphics in predicting the susceptibility of individuals to develop dental caries.

Materials and methods: This case-control study was conducted on 1250 children in the age group of 5 to 12 years from Chennai Corporation School, Vadapalani, Chennai. Out of 1250 subjects, 625 subjects were in the study group and the remaining 625 subjects were the control group. The study group included children with dental caries in 5 or more teeth based on the DMFT index performed and control group consisted of normal, healthy children without any dental caries.

The finger and palmar prints of both hands were taken using a stamp pad. The fingertip patterns were analyzed according to the classical method and configurational types were classified according to the topological method.

Statistical analysis was performed using nonparametric tests and t-test to compare the dermatoglyphic pattern changes between the study group and the control group and was applied for each variable, to compare the proportions, and p-value.

Results: (1) Dental caries susceptibility of an individual increases with an increase in the incidence of whorl pattern (83% correlation). (2) All the variables show statistically significant value, with a degree of divergence of specific dermatoglyphic patterns among study and control group. (3) The dermatoglyphic patterns are efficient and can predict in assessing the risk of susceptibility to dental caries in study group.

Conclusion: The dental caries susceptibility of an individual increased with incidence of whorl pattern and it decreased with incidence of loop pattern.

Clinical significance: The dermatoglyphic patterns may be utilized effectively to study the genetic basis of dental caries. In a developing country like India, it might prove to be a noninvasive, inexpensive and effective tool for screening.

Keywords: Dermatoglyphics, Fingerprint pattern, Dental Caries, Case-control study.

peg like structures, the dermal papillae, characteristic of the definitive dermal ridges progressively formed. ¹

‘The word dermatoglyphics is literally descriptive of the delicately sculpted skin surface, inclusive of single ridges and their configurational arrangements’. This refers to the friction ridge formations which appear on the palms of the hands and soles of the feet. 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.

Some may not rightfully view dermatoglyphics as an independent field of study, even though it has a body of theory, methods and applications. In many respects, it has been used as an adjunct to other disciplines, serving as a vehicle to resolve broader biomedical problems. Thus, in biology, anthropology, genetics and medicine, dermatoglyphics serve as a tool to describe, compare and contrast, and at times predict occurrences and risks for biomedical events studied by these major disciplinary areas. The ridge formations of the skin of an individual begin to appear during 3rd and 4th month of fetal development. After death, decomposition of the skin is last to occur in the area of the dermatoglyphic configurations. The details of these ridges are permanent. There are notably variable characters that are not duplicated in other people even in monozygotic twins or even in the same person, from location to location.

Significant investigations have been carried out into the dermatoglyphic indicators of congenital heart disease, leukemia, cancer, celiac disease, intestinal disorders, rubella, embryopathy, schizophrenia as well as other forms of mental illness.

Dermatoglyphic analysis is now beginning to prove itself as an extremely useful tool for preliminary investigations into conditions with a suspected genetic basis. On the other hand, modes of the inheritance patterns of dermatoglyphics traits and characters are hereditary. So a study was undertaken to investigate and analyze the significance of dermatoglyphics in predicting the susceptibility of individuals to develop dental caries.

AIMS AND OBJECTIVES

1. To record and evaluate the fingerprint patterns of patients diagnosed with dental caries (study group) and caries free individuals (control group). Total numbers of 1250 individuals were considered for the study and the age group considered was between 5 and 12 years.
2. To observe a prevalent and specific dermatoglyphic patterns in study and control group.
3. To determine a degree of divergence of specific dermatoglyphic patterns among study and control group.
4. To predict the efficacy of dermatoglyphic patterns/imprints in assessing the risk of susceptibility to dental caries in study group.

MATERIALS AND METHODS

Source of data: A case-control study comprised a total number of 1250 cases was obtained from Chennai Corporation School, Vadapalani, Chennai. Data was collected from these 1250 children between the ages of 5 and 12 years with no difference between the sexes. Out of 1250 subjects, 625 subjects were grouped into study group and the remaining 625 subjects were considered as the control group. The study group included children with dental caries in 5 or more teeth based on the DMFT index performed and control group consisted of normal, healthy children without any dental caries. A4 size plain paper, cotton, stamp pad, soap, gloves, magnifying lens, scale, protractor, micro tip pencil and eraser, oil, case sheets were used as armamentarium (materials used).

Method of collection of data: Considering the ethical issue and confidentiality of fingerprints of patients, the procedure was explained to the parents of the subjects and permission was obtained through written consent forms before recording the fingerprints. Brief case history with clinical examination and DMFT index was recorded. Subject’s hand were cleaned and dried before imprinting. The finger and palm prints of the subjects were taken using a stamp pad; a thin layer of stamp pad ink was applied to the fingers and palms. An imprint of five fingertips and palm was recorded on an A4 size bond sheet. The same procedure was repeated in relation to the other hand. Prints were dried and studied using a magnifying lens to identify the finger and palm patterns. After taking the imprints of all fingers and palm, ink was removed by using oil, soap and water. The fingertip patterns were analyzed according to the classical method and configurational types were classified according to the topological method.

Evaluation of patterns: The various patterns of fingerprints were analyzed according to the standard guidelines for classification of patterns. The data recorded was entered in Microsoft Excel sheet and applied for statistical analysis. Statistical analysis was performed using nonparametric tests and t-test to compare the dermatoglyphic pattern changes between the study group and the control group and was applied for each variable, to compare the proportions and p-value.

Limitations: The use of stamp pad ink in dermatoglyphic study has got certain disadvantages. The imprint is affected by the amount of pressure exerted while the palm is
recorded. Care must be taken while recording the prints to apply the stamp ink material in adequate amounts. A thin or thick application results in light or dark improper prints.

**Results and observations:** The data obtained by analyzing the fingerprints of study group and control group were entered in a primary data sheet. The two independent quantitative variables were dermatoglyphic variable (Figs 1A to C) (which included plain loop (PL), double loop (DL), arch with loop (AWL), plain whorl (PW), double whorl (DW), arch with whorl (AWW), plain arch (PA), tented arch (TA), central pocket loop (CPL) and accidental (A). Total number of independent quantitative variables = 10) and teeth with dental caries (criteria: 5 or more teeth in an individual were considered under study group; maximum value was 10 and minimum value was 5).

Descriptive statistics and correlation test was performed to determine the p-value for each variable. This included the analysis of mean, median, standard deviation, minimum and maximum values. N = total number of individuals, study group N = 625, control group N = 625. The mean and the SD of whorl pattern (PW + DW + AWW) in study group is (X ± SD) = 7.55 ± 2.03. The mean and the SD of whorl pattern in control group is (X ± SD) = 0.69 ± 1.22. The mean and the SD of loop pattern (PL + DL + AWL) in study group is (X ± SD) = 2.04 ± 0.76. The mean and the SD of loop pattern in control group is (X ± SD) = 8.45 ± 1.80.

In order to describe the characteristics of the large sample size, we had to record the long series of observations appropriately and systematically organize the results. So tabulation, frequency distribution and percentage of individual dermatoglyphic patterns were performed. Frequencies, percentage, valid percentage and cumulative percentage of dental caries were also done. From descriptive statistical analysis and its comparative study we can conclude that SD of whorl and loop pattern are very low in study group and control group respectively. This suggests that our data collected follows the normal distribution curve.

Prevalent and specific dermatoglyphic patterns in study and control group was assessed with a scatter plot diagram and correlation tests. The analysis of the relationship of two characteristics (bivariables) namely, dental caries and whorl pattern, are represented by a point on a graph. This graph is called scatter plot diagram (Graph 1). The configuration of the points on the graph indicates the nature of relationship. Since these points lie clustered, it suggests a correlation or relationship between variables (dental caries and whorl pattern).

To detect whether these variables (whorl pattern, loop pattern and dental caries) are interdependent or co-vary, that is, whether they vary together, correlation test was performed. Since, our variables were quantitative and continuous variables, coefficient of linear correlation or also called as Pearson correlation—two-tailed test was performed. It was performed in both study and control group between whorl vs dental caries and loop vs dental caries. Table p-value was considered as < 0.05.
Whorl vs dental caries when N = 1250 (Graph 2); with independent variable: Whorl; and dependent variable: Dental caries, Table 1 showed that 85% correlation existed between whorl and dental caries (p-value of 0.000). Thus, our result shows that there is a significant relationship between whorl pattern and dental caries. Thus, the two variables whorl and dental caries was positively correlated \( r = 0.85 \). Table 1 shows the same result when permutation and combination was done.

Whorl vs dental caries in study group (Table 2) when N = 625 (Graphs 2, 3 and 5); with independent variable: Whorl; and dependent variable: Dental caries, Table 2 showed that 66% correlation existed between whorl and dental caries (p-value of 0.01). Thus, our result shows that there is a significant relationship between whorl pattern and dental caries. Thus, the two variables whorl and dental caries was positively correlated \( r = 0.66 \). Table 2 also shows the same result when permutation and combination was done.

### Table 1: Correlation between whorl vs dental caries when N = 1250

<table>
<thead>
<tr>
<th></th>
<th>Whorl</th>
<th>Dental caries (Total no. teeth)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pearson correlation</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>1250</td>
</tr>
<tr>
<td>Whorl</td>
<td>0.847</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>0.000</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>1250</td>
<td>1250</td>
</tr>
</tbody>
</table>

*Correlation is significant at the 0.01 level (2-tailed)

### Table 2: Correlation between whorl vs dental caries in study group when N = 625

<table>
<thead>
<tr>
<th>Groups</th>
<th>Whorl</th>
<th>Dental caries (Total no. teeth)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pearson correlation</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>625</td>
</tr>
<tr>
<td>Study</td>
<td>0.66</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>0.010</td>
<td>*</td>
</tr>
<tr>
<td></td>
<td>625</td>
<td>625</td>
</tr>
<tr>
<td></td>
<td>0.66</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>0.010</td>
<td>*</td>
</tr>
<tr>
<td>Control</td>
<td>625</td>
<td>625</td>
</tr>
<tr>
<td></td>
<td>Pearson correlation</td>
<td>1.000</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>625</td>
</tr>
<tr>
<td></td>
<td>**</td>
<td>**</td>
</tr>
<tr>
<td></td>
<td>625</td>
<td>625</td>
</tr>
</tbody>
</table>

*Cannot be computed because at least one of the variable (dental caries) is constant in control group
**Correlation is significant at the 01 level (two-tailed)
Thus, with an increase in the whorl pattern, the patient has an increased susceptibility to dental caries.

Loop vs dental caries when N = 1250 (Graph 2) with independent variable: Loop and dependent variable: Dental caries. Table 3 showed that –83% correlation existed between loop and dental caries (p-value of 0.000). Thus, the two variables loop and dental caries were negatively correlated \( r = -0.60 \). Thus, the two variables loop and dental caries were negatively correlated \( r = -0.83 \). Table 3 shows the same result when permutation and combination was done.

Loop vs dental caries (Graphs 2 and 3) in study group (Table 4) when N = 625 with independent variable: Loop and dependent variable: Dental caries. Table 4 showed that –60% correlation existed between loop and dental caries (p-value of 0.013). Thus, the two variables loop and dental caries were negatively correlated \( r = -0.60 \). Table 4 shows the same result when permutation and combination was done.

Thus, with an increase in the loop pattern, the patient has a decreased susceptibility to dental caries (Graphs 3 to 6).

To determine a degree of divergence of specific dermatoglyphic patterns among study and control group, i.e. to find any significant difference exists between study and control group for both whorl and loop variable we used independent t-test to test the hypothesis.

**Table 3: Correlation between loop vs dental caries when N = 1250**

<table>
<thead>
<tr>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Loop</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dental caries</td>
<td>1.000</td>
<td>*</td>
<td>1250</td>
<td>-0.826</td>
<td>0</td>
<td></td>
<td>1250</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loop</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pearson correlation</td>
<td>-0.826</td>
<td></td>
<td>1250</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>0</td>
<td>*</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 4: Correlation between loop vs dental caries in study group when N = 625**

<table>
<thead>
<tr>
<th>Group</th>
<th>Dental caries (Total no. teeth)</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
<th>Loop</th>
<th>Pearson correlation</th>
<th>Sig. (2-tailed)</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td>Study</td>
<td>Dental caries</td>
<td>1.000</td>
<td>*</td>
<td>625</td>
<td>-0.60</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loop</td>
<td>Pearson correlation</td>
<td>-0.60</td>
<td>0.013</td>
<td>625</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Dental caries</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td></td>
<td>**</td>
<td>**</td>
<td>625</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Loop</td>
<td>Pearson correlation</td>
<td></td>
<td>1.000</td>
<td>625</td>
<td>1.000</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Cannot be computed because at least one of the variable (dental caries) is constant in control group**

**Correlation is significant at the 0.01 level (two-tailed)**
Whorl in study vs control group (Table 5): Showed calculated t-value was 72.333, with a mean difference of 6.85 and p-value was 0.000 for whorl pattern in study vs control group. This signifies that there exists a significant difference between study and control group. 95% confidence interval also supports that there is significant difference between study and control group. (Lower limit, upper limit) = {6.67, 7.04}, standard error difference = 9.48E-02, null value = 0, confidence interval (CI) = 95%.

W = S/C = at 95% CI = {6.67, 7.04}. Whorl (W) in study (S) vs control (C) group at 95% confidence interval was between 6.67 and 7.04 which do not include our null value. Hence, our result was statistically significant.

Loop in study vs control group: (Table 5) (Graphs 4 and 6): Table 5 showed calculated t-value was – 63.654, with a mean difference of – 6.40 and p-value was 0.000 for Loop pattern in study vs control group. This signifies that there exists a significant difference between study and control group in whorl pattern.

95% confidence interval also supports that there is significant difference between study and control group. (Lower limit, upper limit) = {– 6.60, – 6.21}, standard error difference = 0.10, null value = 0, Confidence interval (CI) = 95%.

L = S/C = at 95% CI = {– 6.60, – 6.21}. Loop (L) in study (S) vs control (C) group at 95% confidence interval was between – 6.60 and – 6.21 which do not include our null value. So our result was statistically significant.

To summarize our results, dental caries susceptibility of an individual increased with incidence of whorl pattern and it decreased with incidence of loop pattern. (The analysis of the data was done using SPSS software version 13).

**DISCUSSION**

Dental caries is a microbial disease of the calcified tissues of the teeth, characterized by demineralization of the inorganic portion and destruction of the organic substance of the tooth. Dental caries is the most common chronic disease of childhood and is unequally distributed in the population with most of the disease occurring in 20% of children. Dental caries is a chronic, complex, multifactorial disease for which a multitude of etiologies like host and environmental factors have been proposed. The relative roles of heredity and environmental (nature vs nurture) in the pathogenesis of dental caries has intrigued clinical and basic researchers for decades. There are numerous host resistance and risk factors for dental caries that are genetically determined. It is critical to realize that genes and environment do not act independently of each other and the appearance or magnitude of heritability may differ with various environments.

The pattern of dental caries is similar in members of the same family over several generations and hence, inheritance of this susceptibility is suspected. There are inherited traits that alter the susceptibility to dental caries in humans. Genetic variations in the host factors may contribute to
increased risks for dental caries. Environmental factors, such as diet, oral hygiene habits also play a large role in causing dental caries.

The type of fingerprints is unique and is based on the genetical characteristics of each individual. These dermal patterns once formed remain constant throughout life. Till now, only one study has been conducted in a very small group comprising only 24 patients by Metin Atasu (1992) to analyze the dermatoglyphic patterns in dental caries. We designed and undertook this study to evaluate and analyze the dermatoglyphic patterns in patients with dental caries. From our results we can conclude that the dermatoglyphic patterns varied significantly among the patients with dental caries and healthy individuals. Our study results were similar to other studies like Cummins et al. on Down’s syndrome and Bierman et al. on breast cancer, who noted significant variations in whorl and loop patterns.

Our results also showed that with an increase in the whorl pattern, the patient had an increased susceptibility to dental caries. This result could be compared to Engler et al. (1982), who had analyzed dermatoglyphic patterns in breast cancer patients, and concluded that the presence of six or more whorls on the fingertips of a person could indicate a high risk of obtaining breast cancer.

There is a statistically significant difference between study and control group in loop and whorl pattern similar to Metin Atasu (1992). Thus, we found a definite correlation between the dermatoglyphic patterns and patients with dental caries.

In comparison with the control group, 83% positive correlation was found between whorl and dental caries at a p-value = 0.000. This is highly significant so, we analyze the possible reason for this significance. Dermal ridge differentiation takes place early in the fetal development. It is known that finger and palm prints are formed during the first 6 to 7 weeks of the embryonic period and are completed after 10 to 20 weeks of gestation. Abnormalities in these areas are influenced by combination of hereditary and environmental factors. These abnormalities are expected to appear only when the combined factors exceed a certain level. This threshold theory is now generally accepted and has been extrapolated by the studies of Carter (1969) and Mastunga (1977).

Basically, the pattern of the skin lines on the finger is formed in the second trimester of the fetus and it does not change for each individual during the life. The dermal ridges develop in relation to the volar pads, which are formed by the 6th week of gestation and reach maximum size between 12 and 13th week. The epidermal ridges of the fingers and the palms as well as facial structures like lip, alveolus, palate and tooth bud are also formed from the same embryonic tissue (ectomesenchyme) during the same embryonic period (6-9 weeks). The genetic message in the genome whether normal or abnormal is deciphered during this period and is reflected by dermatoglyphics. Thus, with genetic susceptibility and added environmental factors the proneness for caries due to abnormality in the tooth structures like alterations in dental hard tissues like structure of dental enamel, tooth eruption and development may be reflected in the dermatoglyphics namely whorl and loop patterns.

Hence, dermatoglyphics could indicate a genetic susceptibility to dental caries. In the recent decades, a considerable improvement has been achieved in the concept of correlation between the types of pattern of lines on the fingers and some individual disorders. The pattern of lines on the hand finger has been documented in medicine as a method of diagnosis.

Numerous studies have described a potential genetic contribution to the risk for dental caries. There are numerous familial, pedigree and twin studies on dental caries. Studies on twins have provided strong evidence for the role of inheritance. So, the most convincing data on the role of genetics in the pathogenesis of dental caries have been developed by analyzing the caries incidence in monozygotic and dizygotic twins. It was also suggested by different studies that the children showed a remarkable similarity in dental caries to the susceptibility of the parents.

The pathogenesis of the caries process is rather well understood today, and caries attack rate in humans is a consequence of various attributes. Genetically, regulated processes identified as contributing to caries incidence include tooth eruption, tooth morphology, density or structural integrity of the enamel, composition of the secretions of the salivary glands and salivary flow, the immune response and reduction in the clearance of the bacteria. Bordoni concluded from his study that there is a ‘strong genetic component in primary teeth which affects the incidence of caries’.

Individuals with high resistance to dental caries had a specific immunoglobulin within saliva conveying immunity by lysing the cariogenic bacterial cells. It was suggested that this phenotype was inherited and transmitted as an autosomal dominant trait. Several reports and studies have also shown significant heritability for several microorganisms, including streptococci. Thus, genes and genetic abnormalities that leads to abnormal structural organization of teeth and its environment leads to increased susceptibility to dental caries.

Hence, we can also conclude susceptibility to dental caries has genetic control and this control could be multifactorial in nature.
Studies reveal that HLA DR6-1, 2, 3 had a significant relationship to dental caries, with increased susceptibility to dental caries, enamel defect, as well as to low dose response to Streptococcus mutans antigens. HLA DR 5, 7 with decreased enamel defect and dental caries.2,25,27

Two different lines of investigation have proved that genes in the HLA complex are associated with altered enamel development and increased susceptibility to dental caries. Specific allelic variants of these genes could be used as a potential marker to assess the increased dental caries risk.2,28,32

Although conclusions could be drawn based on this study, digital dermatoglyphics may have a future role in identifying people either with or at increased risk for dental caries so that either risk reduction measures or earlier therapy may be instituted. We also have some evidence from this study to suggest that specific fingerprint patterns may be used as a potential noninvasive anatomical tool which could be used for screening for dental caries and for guiding future research. This relatively noninvasive technique can reasonably be used in selective nonsymptomatic patients (those with positive family history) as a part of definite risk assessment strategy with an ability to detect the earliest changes associated with cariogenesis, many years before the appearance of clinical lesion. This may allow the introduction of more preventive, early diagnosis and effective treatment strategies in patients with dental caries.33-51

SUMMARY

Thus from the our observations and study, it can be summarized that:

1. Dental caries susceptibility of an individual increases with an increase in the incidence of whorl pattern (83% correlation).
2. All the variables show statistically significant value, with a degree of divergence of specific dermatoglyphic patterns among study and control group.
3. The dermatoglyphic patterns are efficient and can predict in assessing the risk of susceptibility to dental caries in study group.

CONCLUSION

The dermatoglyphic patterns may be utilized effectively to study the genetic basis of dental caries. In a developing country like India, it might prove to be a noninvasive, inexpensive and effective tool for screening. These patterns may represent the genetic make up of an individual and therefore his/her predisposition to certain diseases.

Given the expenses involved in conducting the analysis of the chromosomes themselves, dermatoglyphics can prove to be an extremely useful tool for preliminary investigations. The pattern seems to be appearing wherein a definite approach in the form of ‘dermatoglyphics’ might play a significant role in the near future not only for the purpose of screening but also for studying the behavior of dental caries.

Since, dermatoglyphics is still an inexact science at the present time, further extensive research and studies in this field have to be done in order to determine, ascertain and to evaluate the significance of these variations in the dermatoglyphic features of patients with dental caries.

REFERENCES

24. Fogle T. Using dermatoglyphics from down syndrome and class populations to study the genetics of a complex trait. Association for Biology Laboratory Education 1990;11:129-50.
27. Balgir RS. Dermatoglyphics in cleft lip and cleft palate anomalies. PMID: 8365784.

ABOUT THE AUTHORS

PR Abhilash
Assistant Professor, Department of Oral Pathology, NSVK SV Dental College and Hospital, Bengaluru, Karnataka, India

R Divyashree
Assistant Professor, Department of Orthodontics, NSVK SV Dental College and Hospital, Bengaluru, Karnataka, India

Shankar Gouda Patil
Assistant Professor, Department of Oral Pathology, KLE Societies Institute of Dental Sciences and Hospital, Bengaluru, Karnataka, India

Mohit Gupta
Assistant Professor, Department of Orthodontics, New Horizon Dental College, Bilaspur, Chhattisgarh, India

T Chandrasekar
Professor and Head, Department of Oral Pathology, Sathyabama University, Dental College and Hospital, Chennai, Tamil Nadu, India

R Karthikeyan
Reader, Department of Oral Pathology, Surendra Dental College and Research Institute, Sri Ganganagar, Rajasthan, India

CORRESPONDING AUTHOR
PR Abhilash, Assistant Professor, Department of Oral Pathology, #108, Sai Poorna Heights Apartment, 27th Main, Somasundara Palya, 2nd Sector, HSR Layout, Bengaluru-560102, Karnataka, India, Phone: (+91) 9845088563, e-mail: drabhilashpr@gmail.com