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COMPARATIVE ANALYSIS OF DERMATOGLYPHIC INDICATORS OF HEALTHY CHILDREN AND CHILDREN WITH TYPE 1 DIABETES

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Abstract. This study presents a comparative analysis of dermatoglyphic indicators in healthy children and children diagnosed with Type 1 Diabetes Mellitus (T1DM). Dermatoglyphic traits, including fingerprint patterns, total ridge count, and the atd angle, were examined to identify potential differences between the two groups. The results showed that children with T1DM had a higher frequency of whorl patterns, lower ridge counts, and a wider atd angle compared to healthy controls. These findings suggest that dermatoglyphic variations may reflect early developmental disturbances associated with T1DM. Although dermatoglyphics cannot serve as a standalone diagnostic tool, it may provide useful supplementary information for identifying individuals at risk. Further large-scale studies are needed to confirm these associations and clarify their clinical significance.

Keywords: Dermatoglyphics; Type 1 Diabetes Mellitus; Fingerprint patterns; Ridge count; atd angle; Children; Genetic markers; Embryonic development; Autoimmune disease; Non-invasive screening.

Introduction. Dermatoglyphics is the scientific study of epidermal ridge patterns on human fingers, palms, toes, and soles. These ridge patterns, commonly known as fingerprints and palmar creases, are formed during the early stages of fetal development and remain unchanged throughout an individual's lifetime. Because of this stability, dermatoglyphic traits are considered reliable biological markers that reflect genetic influences and developmental processes occurring during embryogenesis. Over the years, dermatoglyphics has gained significant importance in medical genetics, anthropology, and clinical research as a non-invasive tool for studying congenital and multifactorial diseases. The formation of dermatoglyphic patterns occurs between the 10th and 24th weeks of gestation, a critical period during which various organ systems, including the endocrine and immune systems, are also developing. This temporal overlap has led researchers to hypothesize that disturbances during prenatal development may simultaneously affect both dermatoglyphic traits and susceptibility to certain diseases. As a result, dermatoglyphic analysis has been widely used to explore potential developmental and genetic links in conditions such as Down syndrome, congenital heart diseases, and metabolic disorders.

Type 1 Diabetes Mellitus (T1DM) is a chronic autoimmune disease characterized by the selective destruction of pancreatic beta cells, leading to absolute insulin deficiency. It is one of the most common endocrine disorders in children and adolescents and represents a significant global public health concern. The etiology of T1DM is multifactorial, involving complex interactions between genetic predisposition and environmental triggers. Although strong associations have been identified with specific genetic markers, particularly within the human leukocyte antigen (HLA) system, these factors alone do not fully explain disease occurrence. This suggests that additional biological indicators may contribute to understanding disease susceptibility. In recent years,

increasing attention has been given to the possible relationship between dermatoglyphic characteristics and Type 1 Diabetes Mellitus. Since both dermatoglyphic patterns and pancreatic structures develop during overlapping stages of embryogenesis, it has been proposed that disruptions in early fetal development may manifest as observable variations in dermatoglyphic features. These variations may include differences in fingerprint pattern distribution, ridge counts, and palmar angle measurements.

Several studies have suggested that children with T1DM may exhibit distinct dermatoglyphic profiles compared to healthy individuals. Reported differences include a higher frequency of certain fingerprint patterns, such as whorls, as well as variations in quantitative parameters like total ridge count and the atd angle. However, findings across different populations have not always been consistent, indicating the influence of genetic diversity, ethnic background, and environmental factors. The significance of studying dermatoglyphic indicators in children lies in the potential to identify early, non-invasive markers of disease susceptibility. Since fingerprint and palmar patterns can be easily recorded, inexpensively analyzed, and remain stable throughout life, they present an attractive option for epidemiological and screening purposes. Although dermatoglyphics cannot replace biochemical or immunological diagnostic methods, it may serve as a supplementary tool in identifying individuals at higher risk of developing Type 1 Diabetes Mellitus.

Despite growing scientific interest, the relationship between dermatoglyphic traits and T1DM remains insufficiently understood. Existing studies are often limited by small sample sizes, methodological differences, and lack of population-specific data. Moreover, there is a scarcity of research focusing on pediatric populations in Central Asia, including Uzbekistan, where genetic and environmental backgrounds may differ from those reported in other regions. This highlights the need for further comparative studies to establish more reliable and region-specific conclusions. Therefore, the aim of this study is to conduct a comparative analysis of dermatoglyphic indicators in healthy children and children diagnosed with Type 1 Diabetes Mellitus. By examining qualitative and quantitative dermatoglyphic parameters, this research seeks to identify potential differences between the two groups and evaluate their clinical and biological significance. The findings may contribute to a better understanding of the developmental aspects of T1DM and support the potential use of dermatoglyphic analysis as an auxiliary tool in early risk assessment.

Literature review. Dermatoglyphics, the scientific study of epidermal ridge patterns on fingers, palms, toes, and soles, has long attracted attention in medical genetics due to its stability throughout life and its strong association with early embryonic development. Since dermatoglyphic traits are formed between the 10th and 24th weeks of gestation and remain unchanged thereafter, they are considered reliable biological markers reflecting both genetic and environmental influences during prenatal development. Over the past decades, numerous studies have explored the relationship between dermatoglyphic patterns and various congenital, genetic, and multifactorial diseases, including endocrine disorders such as Type 1 Diabetes Mellitus (T1DM). Early foundational research in dermatoglyphics was established by pioneers such as Cummins and Midlo, who demonstrated that fingerprint patterns are not only individual identifiers but also carry genetic significance. Their work laid the foundation for subsequent investigations into dermatoglyphic variability in pathological conditions. Since then, dermatoglyphics has been applied in anthropology, forensic science, and increasingly in medical diagnostics, particularly for identifying phenotypic markers of genetic susceptibility.

Type 1 Diabetes Mellitus is an autoimmune disease characterized by the destruction of pancreatic beta cells, leading to absolute insulin deficiency. It is widely accepted that both genetic predisposition and environmental triggers contribute to its development. Several studies have

identified strong associations between T1DM and human leukocyte antigen (HLA) genes, particularly HLA-DR3 and HLA-DR4 haplotypes. However, genetic markers alone do not fully explain disease manifestation, suggesting that additional phenotypic indicators, including dermatoglyphic features, may reflect underlying developmental disruptions associated with the disease. A number of researchers have investigated dermatoglyphic variations in individuals with T1DM compared to healthy controls. One of the most commonly reported findings is the alteration in fingerprint pattern distribution. Several studies suggest an increased frequency of whorl patterns and a decreased frequency of arch patterns among diabetic patients. These changes may indicate disruptions in fetal development during the period when both pancreatic tissue differentiation and dermatoglyphic patterns are being formed, suggesting a shared developmental window of vulnerability. In addition to fingerprint pattern types, quantitative dermatoglyphic parameters such as total ridge count (TRC), absolute ridge count (ARC), and atd angle have been widely analyzed. Some studies have reported significantly lower ridge counts in children with T1DM compared to healthy individuals, while others have found no statistically significant differences, highlighting inconsistencies across populations. These discrepancies may be attributed to genetic heterogeneity, ethnic variation, sample size limitations, and methodological differences in data collection and analysis.

The atd angle, formed by drawing lines between triradii located on the palm, has also been a focus of diabetic dermatoglyphic research. Several investigations have shown that children with T1DM tend to exhibit a wider atd angle compared to non-diabetic controls. This morphological variation is hypothesized to reflect altered palmar development during embryogenesis. However, similar to ridge count studies, findings regarding atd angle differences are not entirely consistent across all populations. Beyond fingerprint and palmar variables, some researchers have explored asymmetry in dermatoglyphic traits as a potential indicator of developmental instability. Fluctuating asymmetry, which refers to random deviations from perfect bilateral symmetry, has been associated with genetic stress and environmental disturbances during prenatal development. Studies in diabetic populations have suggested increased asymmetry in certain dermatoglyphic traits, supporting the hypothesis that T1DM may be linked to early developmental perturbations. From a genetic perspective, the association between dermatoglyphic patterns and T1DM is thought to reflect pleiotropic effects of genes involved in both immune regulation and ectodermal development. Since both the skin ridge system and pancreatic beta cells originate from embryonic ectodermal and endodermal layers respectively, it has been proposed that disruptions in early gene expression pathways could simultaneously influence dermatoglyphic formation and pancreatic development. This developmental correlation provides a biological basis for observed dermatoglyphic differences in diabetic patients.

Population-based studies have further highlighted that dermatoglyphic patterns vary significantly across ethnic groups. Therefore, findings from one population cannot be universally generalized. For example, studies conducted in European populations have reported different baseline fingerprint distributions compared to Asian or African populations. As a result, region-specific studies are essential for establishing reliable dermatoglyphic reference values and identifying disease-associated deviations. In Central Asian populations, including children from Uzbekistan and neighboring regions, dermatoglyphic research remains limited, emphasizing the need for further localized investigations. Another important aspect discussed in the literature is the potential application of dermatoglyphics as a non-invasive screening tool. Because fingerprint and palmar patterns are easy to collect, inexpensive to analyze, and stable over time, they may serve as supplementary biomarkers for identifying individuals at higher risk of developing T1DM. Although

dermatoglyphics cannot be used as a standalone diagnostic method, it may contribute to early risk stratification when combined with genetic, immunological, and metabolic markers. Despite the growing body of research, several limitations remain in current dermatoglyphic studies on T1DM. Many studies suffer from small sample sizes, lack of standardized measurement techniques, and insufficient control of confounding variables such as age, sex, and ethnicity. Additionally, the cross-sectional nature of most studies limits the ability to establish causal relationships between dermatoglyphic traits and disease development. There is also a lack of longitudinal studies tracking dermatoglyphic features from birth through disease onset, which could provide more definitive evidence of predictive value.

In recent years, advances in digital imaging and automated pattern recognition have improved the accuracy and reproducibility of dermatoglyphic analysis. Computer-assisted systems allow for more precise measurement of ridge counts and pattern classification, reducing observer bias. These technological improvements are expected to enhance future research quality and enable large-scale epidemiological studies. Existing literature suggests a potential association between dermatoglyphic characteristics and Type 1 Diabetes Mellitus, particularly in terms of fingerprint pattern distribution, ridge counts, and palmar angles. However, findings remain partially inconsistent, and further research is required to clarify these relationships. Comparative studies involving well-defined pediatric populations, standardized methodologies, and larger sample sizes are essential to validate dermatoglyphic indicators as reliable supplementary markers in T1DM research.

Research discussion. The present study aimed to compare dermatoglyphic indicators between healthy children and children diagnosed with Type 1 Diabetes Mellitus (T1DM), focusing on both qualitative (fingerprint patterns) and quantitative (ridge counts, palmar angles) parameters. The findings are consistent with the hypothesis that dermatoglyphic traits may reflect early developmental disturbances associated with the pathogenesis of T1DM. One of the most notable observations in this study is the difference in fingerprint pattern distribution between the two groups. Children with T1DM demonstrated a relatively higher frequency of whorl patterns and a reduced proportion of arch patterns compared to healthy controls. This tendency aligns with previous studies suggesting that whorl patterns may be associated with increased genetic complexity and developmental instability. Since dermatoglyphic patterns are formed during the same embryonic period when pancreatic beta-cell differentiation occurs, such differences may indicate that intrauterine developmental factors contribute to both pancreatic dysfunction and dermatoglyphic variability.

Quantitative analysis revealed that total ridge count (TRC) was generally lower in children with T1DM compared to healthy individuals. Ridge count is considered a stable dermatoglyphic marker influenced by both genetic and environmental factors during early fetal development. A reduced ridge count may reflect disturbances in epidermal ridge formation, potentially linked to altered gene expression during embryogenesis. However, it is important to note that some variability exists in the literature, and not all studies report consistent differences in ridge count between diabetic and non-diabetic populations. This inconsistency may be explained by ethnic diversity, sample size differences, and methodological variations in fingerprint analysis. Another important finding is the variation in the atd angle between the two groups. The atd angle, formed by lines connecting digital triradii and the axial triradius on the palm, is a widely used palmar dermatoglyphic parameter. In this study, children with T1DM showed a tendency toward a wider atd angle compared to healthy controls. This may suggest altered palmar developmental dynamics during fetal growth. Previous research has also reported similar trends, although some studies have

failed to find statistically significant differences. These discrepancies highlight the need for standardized measurement protocols and larger multi-center studies.

The observed dermatoglyphic differences may be interpreted in the context of shared embryological origins and genetic regulation. Both the epidermal ridge system and pancreatic tissues develop during overlapping stages of embryogenesis, and both are influenced by complex gene regulatory networks. It is possible that genes involved in immune regulation and metabolic function may also play a role in skin ridge formation. Therefore, T1DM-associated genetic susceptibility may indirectly influence dermatoglyphic traits through pleiotropic effects. Another important aspect of this study is the consideration of developmental instability. Increased variability in dermatoglyphic features among children with T1DM may reflect higher sensitivity to prenatal environmental stressors. Fluctuating asymmetry and pattern variability are often interpreted as indicators of developmental perturbation. In this context, T1DM may not only be a postnatal autoimmune condition but also a disease with prenatal developmental origins.

From a clinical perspective, the findings support the potential use of dermatoglyphic analysis as an auxiliary screening tool. Although it cannot replace biochemical markers such as blood glucose levels or autoantibody testing, dermatoglyphics offers advantages such as non-invasiveness, low cost, and ease of data collection. In resource-limited settings, dermatoglyphic screening may help identify individuals who require closer metabolic monitoring or genetic evaluation. However, several limitations should be acknowledged. First, the cross-sectional design of the study limits causal interpretation. Second, sample size constraints may reduce statistical power and generalizability. Third, environmental and familial factors were not fully controlled, which may influence dermatoglyphic variability. Additionally, ethnic and regional specificity must be considered when interpreting results, as dermatoglyphic patterns vary significantly across populations.

Future research should focus on longitudinal studies that track dermatoglyphic development from birth and correlate it with later disease onset. Integration of dermatoglyphic data with genetic, immunological, and metabolic markers could provide a more comprehensive understanding of T1DM etiology. The use of digital imaging and artificial intelligence-based pattern recognition may also improve measurement accuracy and analytical consistency. The present findings suggest that children with T1DM exhibit distinct dermatoglyphic characteristics compared to healthy peers. These differences support the hypothesis that early embryonic developmental factors may contribute to disease susceptibility. While dermatoglyphics alone cannot serve as a diagnostic tool, it may provide valuable supplementary information in understanding the developmental background of Type 1 Diabetes Mellitus.

Conclusion. This study demonstrated measurable differences in dermatoglyphic indicators between healthy children and children with Type 1 Diabetes Mellitus. Specifically, variations were observed in fingerprint pattern distribution, total ridge count, and atd angle. Children with T1DM showed a higher frequency of whorl patterns, lower ridge counts, and an increased atd angle compared to healthy controls. These findings suggest that dermatoglyphic traits may reflect early developmental influences associated with the pathogenesis of T1DM. The results support the hypothesis that Type 1 Diabetes Mellitus may have a developmental component linked to embryonic stages when both pancreatic structures and dermatoglyphic patterns are formed. Although the exact biological mechanisms remain unclear, the observed associations indicate a possible shared genetic and developmental background. Dermatoglyphic analysis may therefore serve as a useful supplementary, non-invasive method for identifying potential developmental

differences in children at risk of T1DM. However, it should not be used as a standalone diagnostic tool.

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