Dermatoglyphic Characteristics of Patients with Severe Nerve Deafness

Yu-Hsing Lien Founder of Lien's Dermatoglyphics Think tank macronu@ms45.hinet.net

Chi-Ling Chuang Founder of Yo-Chin Consortium Psychiatric Hospital empyreanorganics@gmail.com

Yow-Ji Chang Senior Consultant of Lien's Dermatoglyphics Think tank macronu@ms45.hinet.ne

Abstract

A total of 104 patients with severe nerve deafness and the parents of 96 patients with deafness. A total of 192 patients participated in this study. The control group was 115 normal children without deafness. In the study, there was a double loops in the IV area of the left palm dermatoglyphics and a loop in the mid-fibular side of the right foot dermatoglyphics. The probability of these two messages in deaf patients was about 23%-26%. In the relatives of patients with normal hearing, these two messages also appeared in a certain proportion; but they were not found in the normal control group. Compared with the normal control group, there are significant differences in the dermatoglyphic group between the positive group of deaf patients, the normal gene-carrying relatives' group.

Keywords: Severe sensorineural deafness, dermatoglyphic features, GJB2related deafness genes, large vestibular aqueduct syndrome

Historical context

Nerve deafness, also known as sensorineural hearing loss, is an ear condition whose symptoms include decreased hearing abilities or total deafness because of inner ear damage. It affects millions of people at varying degrees of severity. The condition can result from defects present at birth, referred to as congenital defects, or symptoms acquired later in life. Most occurrences of nerve deafness are attributed to abnormalities of hair cells located in the inner ear.

The majority of hereditary deafness are single-gene genetic diseases with strong heterogeneity. Among them, non-syndromic deafness is the majority. In the study of hereditary deafness *in Europe and America, several key deafness genes have been found*, such as *GJB2 Deafness caused by gene mutation* (Kelsell 1997, Estivll 1998); *SLC26A4 gene mutation caused Pendred syndrome* (Abe & Usami 1999). However, because of the heterogeneity of genes, *it is still difficult to analyze and determine the*

exact genetic cause of hearing loss (Fetal Pediatr 2019). High-throughput

sequencing analysis of genes related to hearing loss is an effective and economical method, and currently provides a diagnosis rate of about 40% (Ariane Paoloni-Giacobino 2019). In addition, for non-syndromic hearing loss caused by MYO15A gene mutation, *MYO15A is the third most important gene for hereditary sensorineural hearing loss* after GJB2 and SLC26A4. There is currently a meta-analysis of gene mutation frequency (Farjami 2020). According to the study of congenital deafness in Chinese, *the mutation rate of GJB2 can reach 25%, and the detection rate of SLC26A4 gene mutation in patients with large vestibular aqueduct syndrome reaches 97.9%* (Dai Pu et al. 2005).

Dermatoglyphics is a very special feature in humans. *Characteristics, the dermatoglyphics possessed by an individual are unique*, based on the regulation of DGF by NGF (Mobley et al. 1977; Levi-Montalcini 1987), dermatoglyphics is closely related to genes and the nervous system. *Dermatoglyphics has done a lot of research on genetic abnormalities and genetic diseases* (Alter 1966; Schauman & Alter 1976). Therefore, the study of dermatoglyphics in patients with severe sensorineural hearing loss is aimed at patients with clear pathogenic organs. At present, there is also a certain proportion of genetic screening. In addition to exploring whether patients with hereditary deafness have special dermatoglyphic characteristics, the exploration of the correlation between dermatoglyphics and established genes and the nervous system is a breakthrough research. Further expand the analysis The dermatoglyphic characteristics of the parents also hope to explore whether the recessive gene of deafness also has identifiable dermatoglyphic characteristics.

Methods:

There are 104 patients with severe nerve deafness and the parents of 96 patients with deafness, a total of 192 patients participated in this study.

After inquiring about medical history, specialist physical examination, audiology examination and imaging examination, the subjects were all subjected to dermatoglyphic collection and analysis, and peripheral blood was drawn for common deafness gene detection (GJB2, SLC26A4 and mitochondrial gene 12SrRNA).

The control group consisted of 115 normal children without deafness. All cases participating in the experiment were sampled with dermatoglyphics, including fingerprints, palm prints and foot prints, and then the dermatoglyphics of the cases were classified and quantified according to Penrose's method (Penrose 1965, 1968).

Results:

Among 104 patients with deafness, 37 patients with GJB2-related deafness (GJB2 homozygous or compound heterozygous mutation), 26 patients with large vestibular aqueduct syndrome (SLC26A4 homozygous or compound heterozygous mutation), and 44 patients with negative common deafness gene test. In 104 patients with deafness(figure 1), there was 8.65% of the double loops in the IV area on the left palm, and the probability of the loop in the mid-fibular side of the foot dermatoglyphics (figure 2) was on the left side: 10.6%, and the right: 11.5%; in 37 samples of GJB2 deaf patients, the probability of a double loops in the IV zone on the left was 10.8%, and a loop in the mid-fib of the foot dermatoglyphics, the probability of the loops in the IV zone on the left side: 13.5%, the right side: 13.5%; in 26 cases of patients with large vestibular aqueduct syndrome, the probability of the double loops in the IV zone on the left was 3.8%, and the probability of a loop in the mid-fibular side of the foot dermatoglyphics was on the left : 8.3%, right side: 3.8%; in 43 cases of common deafness gene test negative, the probability of double loops in the IV zone on the left was 9.3%, right Side: 16.3%.

Among the 192 relatives of patients with normal hearing, 64 were GJB2 carriers (GJB2 heterozygous mutation), 44 were SLC26A4 carriers (SLC26A4 heterozygous mutation), and 85 were negative for common deafness genes. The family members of 192 patients found that the probability of double loops in the IV zone on the left palm dermatoglyphics was 4.7%, and the probability of a loop in the mid-fibular side of the foot dermatoglyphics was 5.7% on the left side and 7.3% on the right side; among GJB2 carriers , The probability of a loop in the mid-fibular side of the foot dermatoglyphics was on the left: 4.7%, The right was: 6.3%; among SLC26A4 carriers, the probability of a loop in the mid-fibular side of the foot dermatoglyphics was 4.5% on the left side and 6.8% on the right side. Among those with negative deafness gene test,

the probability of a loop in the mid-fibular part of the foot dermatoglyphics was 8.3% on the left and 8.3% on the right.

The probability of the two messages appearing in patients with deafness was between 23% and 26%. GJB2-related deafness patients (the probability of double loops in the IV area on the left palm dermatoglyphics was 10.8%, and the middle section on the right side of the foot dermatoglyphics) The probability was 13.5%) and the deafness gene test negative group had the highest probability (the probability of double loops in the IV area on the left side of palm dermatoglyphics was 9.3%, and the probability of a loop in the right middle section of the foot dermatoglyphics was 16.3%).

In the relatives of patients with normal hearing, these two messages also appear in a certain proportion (the probability of double loops in the IV area on the left palm dermatoglyphics was 6%, and the probability of a loop in the right middle of the fibular side of the foot dermatoglyphics was 8.3%), but none was found in the normal control group. In addition, the analysis results also showed that the research subjects have unique dermatoglyphic characteristics in hand and foot; GJB2-related deafness and patients with large vestibular aqueduct syndrome had different dermatoglyphic characteristics.

In addition, compared the deaf patient group and the relative group of patients with normal hearing, the common deaf gene test negative patient group and the negative relative group, The group of patients diagnosed with hereditary deafness, the group of relatives carrying the deaf gene, the group of relatives carrying the deaf gene,, and the non-carrying. There were also significant differences in the dermatoglyphic patterns in the deaf gene relative group. Furthermore, there were also significant differences between the deaf gene-carrying relative group and the non-deaf gene-carrying relatives group and the normal control group.

Conclusions:

Dermatoglyphic analysis is simple and convenient. Taking nerve deafness and its family members as a case study, some identifiable information was found in the dermatoglyphic characteristics, which is because the formation of dermatoglyphics is regulated by multiple genes. Based on hereditary deafness, most of them are singlegene genetic diseases with strong heterogeneity.

Therefore, it can be seen that hereditary deafness will also have multiple dermatoglyphic characteristics, but if the dermatoglyphic characteristics of recessive genes related to deafness can be discovered, it will be very helpful to facilitate the implementation of further tests for high-risk groups. Patients with severe nerve deafness have special dermatoglyphic characteristics, and are closely related to common deafness genes, which can be used as an aid to deafness screening.

Furthermore, based on the dermatoglyphic system formed by the regulation of multiple genes, this study explored the relationship between dermatoglyphics and neurological structure through the investigation of the correlation between dermatoglyphics and deafness genes, and proposed hypotheses between the two. It can be used for further research and verification.

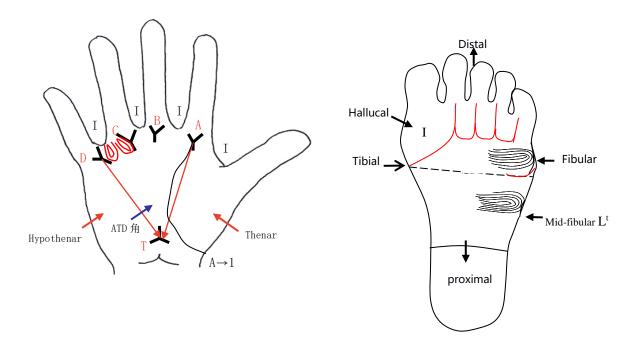


Figure 1: double loops in the IV area (D-L^d) Figure 2: Mid-fibular Loop

(L^t)

References

Abe S., Usami S., Hoover D.M., et al. (1999). Fluctuating sensorineural hearing loss associated with enlarged vestibular aqueduct maps to 7q31, the region containing the Pendred gene. Am J Med Genet, 1999, 82(4): 322-328.

Alter M. (1966). Dermatoglyphic analysis as a diagnostic tool. Medicine, 1966, 46:35 Schauman B, Alter M. (1976). Dermatoglyphics in Medical Disorders, New York Springer-Verlag, 1976

- Ariane Paoloni-Giacobino, et al. (2019). Genetics of hearing disorders in children. Review Rev Med Suisse. 2019 Oct 2;15(665):1740-1745. [Article in French]. PMID: 31580017
- Dai Pu et al. (2005). *Gene diagnosis a major advance in otology*. Chinese Journal of Otology, 2005, 3(1)
- EstivlI X, et al. (1998). Connexin 26 mutations in sporadic and inherited sensorineural deafness. Lancet, 1998, 351: 394
- Farjami M., Assadi R. et al. (2020) The worldwide frequency of MYO15A gene mutations in patients with non-syndromic hearing loss: A meta-analysis. Iran J Basic Med Sci, 2020, 23(7):841-848.
- Fetal Pediatr. (2019) Targeted Mutation Analysis of the SLC26A4, MYO6, PJVK and CDH23 Genes in Iranian Patients with AR Nonsyndromic Hearing Loss. Pathol. 2019 Apr; 38(2): 93-102.
- Kelsell DP, et al. (1997). Connexin 26 mutations in Hereditary non-syndromic sensorineural deafness. Nature, 1997, 387: 802
- Levi-Montalcini R. (1987). *The nerve growth factor 35 years later*. Science, 1987, 237:1154-1162.
- Mobley W. C. et al., (1977). *Nerve growth factor. Parts I,II, and III*. New England J. of Medicine, 1977, 297:1096-1104, 1149-1158, 1211-1218.
- Penrose, L. S. (1965). Dermatoglyphic topology. Nature, 1965, 205:544.
- Penrose, L. S. (1968). *Memorandoum on dermatoglyphic nomenclature*. Birth Defects, 1968, 4(3): 1