Only One Family Member Born without Fingerprints

Amal R Nimir
Faculty of Medicine, Perdana University, Malaysia

Abstract
All of us are born with fingerprints and everyone’s are unique. Therefore; they are the world’s oldest means of individual identification. However, people with adermatoglyphia or what is called the immigration delay disease, do not have fingerprints from birth. To date, only few cases have been described with additional clinical features in most of them. A case of adermatoglyphia in a 23-year-old Iraqi male who resides in Malaysia for 21 years is reported here. A comparison of this case with previous reports in the literature has been discussed and the fashion of inheritance of this disorder has been debated. Because of the problem in personal identification, this disorder causes the patient significant difficulties.

Keywords
Skin disorder; Autosomal dominant; Gene mutation

Case Report
Adermatoglyphia is characterised by the lack of epidermal ridges of the palms and soles, which results in the absence of fingerprints. It is also associated with a decreased number of sweat gland openings and reduced sweating of palms and soles [1]. We report a case of a noticeably rare disorder, adermatoglyphia in a 23-year-old Iraqi male who resides in Malaysia for 21 years. He is a good-looking young man with muscular body built. He does not have any manifestation of skin disease or any other illness.

On examination, epidermal ridges were completely missing from his fingers, while the toes are normal. His skin and hair appeared quite normal, with only mild hyperkeratosis of the hands and calluses on weight-bearing areas. He had no history of blistering, no subjective problems of the skin of his hands or feet and was not more sensitive to heat or cold than unaffected persons. A sweat test of the hands revealed reduced ability for transpiration. Touch discrimination tested by two-point discrimination was normal as compared with controls. This patient refused to do a fingertip skin biopsy to identify the number of sweat gland openings.

He is worried about his travel since he has been recently graduated as an aircraft engineer. The candidate accidentally discovered that he has no fingerprints when he wanted to issue a new passport from Iraqi embassy in Kuala Lumpur. After trying on both hands, the embassy officer asked whether the patient has eczema or any skin disease as this well-educated officer knows that some of the skin diseases can cause absence of fingerprints [2]. As our patient denies, the officer told him that it would be very difficult to get a new passport. Few days later, he faced the same problem when he wanted to open a current account in Citi Bank for his new job offer. The patient got a letter from Japatan Pendaftaran Negara (National Registration Department) to officially confirm his disorder. Both hands and the siblings (both genders) do not have this disorder. Asking the patient about the relatively late discovery, he mentioned that previously, fingerprint was not required to issue a passport or identification card. This will raise the possibility that some of the family previous generations might have adermatoglyphia passed unnoticed.

Discussion
By contrast, adermatoglyphia doesn’t come with any side effects besides a minor reduction in the ability to sweat. In general, people with the disease are otherwise completely healthy. The lack of fingerprints can cause a problem of personal identification specially when travelling which are greatly amplified because few people have heard of the condition, that’s why it is also called immigration-delay disease [3].

It is a real occurrence for at least four extended families throughout the world affected by this rare disease. There are some similarities in the presentation of this case and the 29-year-old woman reported by Burger et al. in 2011 [3]. Burger also mentioned in his report that absence of fingerprints was inherited in an autosomal dominant fashion, with 10 affected individuals over 4 generations.

Fingertips are not the only part of the body that leaves identifiable prints. The surfaces
in the palms of the hands and the soles of feet are covered by friction skin. Friction skin is covered with papillary ridges that assist in the ability to grasp and hold onto objects. The patterns formed in these ridges are very important since they are determined by the fourth month of gestation and remain fixed throughout life. Only severe mutilation or skin disease can cause them to change [4].

Hence, the argue here is that if this disorder is inherited as autosomal dominant, how can we explain that the patient’s parents have normal fingerprints? On the other hand, there is no possibility that it is Y-linked or X-linked since his brother has normal fingerprints. The two possibilities here are; it is either a de novo gene mutation, or the disorder might be inherited as autosomal recessive in this case of studied 3 unrelated families with adermatoglyphia. In a 4-generation Swedish family, all affected members had absence of fingerprints from birth. In a 4-generation family of Austrian ancestry, all patients reported absence of recognizable epidermal ridges and difficulty in grasping and holding items with their hands. The male proband of an American family lacked epidermal ridges and also reported plantar caluses, palmoplantar hypohidrosis, difficulty in grasping and holding and fingernail pterygia.

In an effort to find the cause of the disease, sequenced the DNA of 16 members of one family with adermatoglyphia in Switzerland [1,3]. Seven had normal fingerprints and the other nine did not. After investigating a number of genes to find evidence of mutation, the researchers came up empty-handed. However, further analysis revealed a heterozygous splice site mutation in the skin-specific SMARCAD1 short isoform that segregated with disease in the family.

In 3 unrelated families with adermatoglyphia, sequenced the SMARCAD1 gene and identified 3 different heterozygous splice site variants, 1 of which involved the same nucleotide as the mutation identified in the Swiss family by [1,5]. All 3 mutations were predicted to abolish a conserved donor splice site adjacent to the 3-prime end.
of a noncoding exon unique to the skin-specific SMARCAD1 isoform. In the Swedish family, the only family for which multiple DNA samples were available, the mutation segregated completely with disease; none of the 3 mutations were found among 8,000 individual sequences from the 1000 Genomes Project and NHLBI Exome Sequencing Project databases.

Other inherited diseases that result in a lack of fingerprints are Naegeli syndrome and Dermatopathia Pigmentosa Reticularis (DPR). These conditions may not seem life-threatening, but in reality, it can be extremely hazardous. The inability to sweat turns every hot day or active play into a potential for heatstroke. Researchers at the Technion-Israel Institute of Technology found that the same genetic mutation causes both Naegeli syndrome and DPR. The mutation produces a defect in the protein keratin 14 (KRT14) and causes skin cells to be inappropriately targeted for programmed cell death [6]. The finding suggests that the two disorders, which were previously thought to be distinct, might, in fact be one.

Adermatoglyphia was used as a key plot point in Series 4 Episode 7 of BBC crime comedy Death in Paradise, when a woman with the condition was murdered; citing evidence that the condition was only inherited through the maternal line, the detectives proved that one suspect’s claim to be the victim’s long-lost son was a deception and he killed his ‘mother’ to avert her from learning the truth.

Conclusion

Genes’ investigations and DNA sequencing are strongly requisite in this case to perceive the mutant gene that causes adermatoglyphia in this case and to describe the possible fashion of inheritance.

References