

CASE REPORT

Hands hypopigmentation

Abdulghani Mohamed Ali, Department of Medicine, Tikrit University College of Medicine, Tikrit, Iraq. Mobile: +9647701831295, Email: abdulghani.Mohamed@tu.edu.iq; galsamarrai@yahoo.com ORCID: <http://orcid.org/0000-0002-7872-6691>

Address correspondence: Prof. Abdulghani Alsamarai, College of Medicine, Tikrit University, P O Box 45, Alyarmook ST, Tikrit, Iraq. Email: abdulghani.Mohamed@tu.edu.iq; galsamarrai@yahoo.com.

ABSTRACT

Hypopigmentation is a common dermatologic problem of multiple aetiologies. This health problem is associated with social and psychological impact on the patient and his family. Here I report a case of hands hypopigmentation in a year's old, adult male.

Key Words: Hypopigmentation, Skin, Loss of color, Anxiety.

Introduction:

Skin depigmentation is a common dermatologic disorder [1]. This clinical condition is of two types, the permanent lesion which is natural history of clearance and relapses such as vitiligo or not cleared with treatment such as post-inflammatory hypopigmentation. [2]. The second clinical form is the temporary depigmentary lesion which is with spontaneous remission or cleared as a response to treatment such as fungal infection and/or chemical depigmentation [3]. Chemical depigmentation or as may be termed occupational leukoderma is uncommon skin condition, however, 20% to 50% of cases are due to occupational environment [4]. There is a long list of chemicals that induced leukoderma [5-10], however, the aliphatic and aromatic derivatives of catechols and phenols are the largest and best studied chemical groups and are responsible for inducing leukoderma in human being [11-21].

Case Report:

A 32- years old male presented with hypopigmentation of both hands of 3 months duration. The normal color of patient hand before lesion development as shown in Fig 1. After the use of solid soap for about of 3 weeks he developed a hypopigmentation of both hands as shown in Fig. 2. The same condition was developed previously once he used the solid soap. Spontaneous repigmentation occurred after the discontinuation of soap uses for 3 to 4 weeks. This hypopigmentation is not developed when he used a liquid soap. There is no lesion in his body other than his hands and thus the case is diagnosed as stage I chemical leukoderma since the lesion is confined only the site of contact (both hands).



Fig.1. Patient hand normal skin color



Fig. 2. Hands of the patient

Soap constituents

The used soap ingredients are: Sodium palmate kernelate, fragrance, palm kernel acid, sodium chloride, glycerin, tetrasodium EDTA, coumarins and limonene.

Discussion:

Skin color loss is a condition that induced by multiple causes. Chemical depigmentation is one of the clinical cases that reported due to occupational environment. The continuous contact of the skin with the chemical substances leads to the induction of hypopigmentation. The pathogenesis of depigmentation due to contact with chemical substances is not well understood [4] and not all individual exposed to the substance developed hypopigmentation, indicating host susceptibility [2,5,7]. Although there are differences in host susceptibility for development of chemical leukoderma, our explanation is that the melanocyte is the target of the chemical substance irrespective of the depigmentation mechanism of induction. Our diagnosis as that the hypopigmentation is induced due to contact with chemicals that are present in solid soap is confirmed by the spontaneous return on hand normal skin following discontinuation of using solid soap. The depigmentation action of melanocytotoxic materials is influenced by exposure time, substance concentration, and host susceptibility. In this case the review of the used soap constituents indicated that sodium chloride may induced depigmentation through the functions of transporters and ion channels at the plasma membrane of melanocyte [22]. In addition, pigmentation regulated through a melanosomal two pore sodium channel and *that in pigment cells the endolysosomal cation channel TPC2 localizes predominantly to melanosomes, where it mediates a Na⁺-selective current to modulate melanosomal membrane potential, pH and pigmentation.* [23]. Coumarins, glycerin, and limonene are having a bleaching effect and thus may play a role of the induction of hypopigmentary lesion in hands of this case [24-26].

In conclusion, the hypopigmentation of hands may be due to bleaching that is induced by sodium chloride, coumarins, glycerin or/ and limonene. However, this warranted an experimental evaluation in an animal model to clarify which one of these soap constituents is the depigmentation inducer.

References

1. Alsamarai AGM. Prevalence of skin diseases in Iraq. Int J Derm 2009; 48(7):734-9.
2. Lpeere H, Boone B, De Schepper S, Verhaeghe E, Van Geel M, Ongenae K, et al. Chapter 75. Hypomelanoses and Hypermelanoses. In: Goldsmith LA, Katz SI, Gilchrest BA, Paller AS, Leffell D, Wolff K (Eds.). Fitzpatrick's Dermatology in General Medicine, 8e. 2011. McGraw-Hill Education.
3. Al Samarai AM. Prevalence of skin diseases in Samara, Iraq. Middle East J Internal Med 2009; 2: 15-19.
4. Bonamonte D, Vestita M, Romita P, Filoni A, Foti C, Angilini G. Chemical leukoderma. Dermatitis 2016; 27(3):90-100.
5. Wattanakrai P, Miyamoto L, Taylor JS. Occupational pigmentary disorders. In: Wahlberg JE, Kanerva L, Elsner P, et al, eds. Handbook of Occupational Dermatology. Berlin, Germany: Springer; 2000:280-294.
6. Bonamonte D, Angelini G. Disordini pigmentari da contatto. Ann Ital Dermatol Allergol 2001;55:1-15.

7. Boissy RE, Manga P. On the etiology of contact/occupational vitiligo. *Pigment Cell Res* 2004; 17:208-214.
8. Ghosh S. Chemical leukoderma: what's new on etiopathological and clinical aspects? *Indian J Dermatol* 2010; 55:255-258.
9. Miyamoto L, Taylor JS. Chemical leukoderma. In: Hann SK, Nordlund JJ, eds. *Vitiligo: A Monograph on the Basic and Clinical Science*. Oxford, England: Blackwell Science Ltd; 2000:269-280.
10. Rietschel R, Fowler JJ. Contact leukoderma (vitiligo) hyperpigmentation and discolorations from contactants. In: Rietschel R, Fowler JJ, eds. *Fisher's Contact Dermatitis*. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:571-579.
11. Kahn G. Depigmentation caused by phenolic detergent germicides. *Arch Dermatol* 1970; 102:177-187.
12. Malten KE, Seutter E, Hara I. Occupational vitiligo due to paratertiary butylphenol and homologues. *Trans St Johns Hosp Dermatol Soc* 1971; 57:115-134.
13. James O, Mayes RW, Stevenson CJ. Occupational vitiligo induced by p-tertbutylphenol, a systemic disease? *Lancet* 1977; 2(8050):1217-1219.
14. Gellin GA, Possick PA, Davis IH. Occupational depigmentation due to 4-tertiarybutyl catechol (TBC). *J Occup Med* 1970; 12:386-389.
15. Gellin GA, Possick PA, Perone VB. Depigmentation from 4-tertiary butyl catechol experimental study. *J Invest Dermatol* 1970; 55:190-197.
16. Bajaj AK, Gupta SC, Chatterjee AK. Contact depigmentation from free paratertiary- butylphenol in bind adhesive. *Contact Dermatitis* 1990; 22:99-102.
17. Rodermund OE. Letter: occupational vitiligo caused by paratertiary butylphenol. *Arch Dermatol* 1976; 112:554-555.
18. Calnan CD, Cooke MA. Leukoderma in industry. *J Soc Occup Med* 1974; 24:59-61.
19. Malten KE. Paratertiary butylphenol depigmentation in a "consumer". *Contact Dermatitis* 1975; 1:181-182.
20. Malten KE. Dermatological problems with synthetic resins and plastics in glues. Part I. *Derm Beruf Umwelt* 1984; 32:81-86.
21. Horio T, Tanaka K, Komura J. Depigmentation due to paratertiarybutyl catechol. *Int Arch Occup Environ Health* 1977;39:127-133
22. Bellano NW, Oancea EV. Ion transport in pigmentation. *Arch Biochem Biophys* 2014;365:35-41.
23. Bellano NW, Escobar IE, Oancea EV. A melanosomal two-pore sodium channel regulates pigmentation. *Sci Rep* 2016; 6:26570; doi: 10.1038/srep26570 (2016).
24. American Pharmaceutical Association. *Handbook of Non-prescription Drugs*. 1996.
25. Filipsson AF, Bard J, Karlssons S. Concise International Chemical Assessment Document 5. Limonene. WHO, Geneva, 1998.
26. WHO- International Agency for Research on Cancer. *IARC Monograph* 2000; 77:193-225.