

To date there have been only about 60 patients diagnosed with CHILD syndrome globally, with nearly 30 unique NSDHL mutations being described. The accumulation of toxic metabolites (3-hydroxy-3-methylglutaryl-coenzyme A and oxysterol) and a deficiency in cholesterol synthesis have been shown to be responsible for the pathogenesis of CHILD syndrome in the epidermis. It causes ipsilateral symptoms with the right side of body affected more often than the left. They have scaly erythematous plaques along the Blaschko's lines and verruciform xanthoma with ptychotropism, without crossing the midline. Histopathology of the skin lesions usually demonstrates orthokeratosis alternating with parakeratosis, hypogranulosis, well-differentiated keratinocytes with irregular acanthosis, and foamy macrophages infiltration at the dermis.

Treatments reported in the literature included topical keratolytic agents, emollient, and oral retinoids which only helped in the thickness of the epidermis.³ Surgical removal of diseased skin followed by skin graft that carries normal alleles has been successful.³ Recent pathogenesis-targeted therapy with topical 2% lovastatin and 2% cholesterol to the lesional area has been shown to be efficacious.^{2,4,5} Awareness of this syndrome should be raised among the clinicians as the current topical treatment is highly promising.

Acknowledgment

The authors thank the Director of Health Malaysia for permission to publish this paper.

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Conflict of interest: None.

Funding source: Woman and Child Hospital, Kuala Lumpur, Malaysia.

doi: 10.1111/ijd.15296

References

- 1 Happle R. Ptychotropism as a cutaneous feature of the CHILD syndrome. *J Am Acad Dermatol* 1990; **23**: 763–766.

- 2 Seeger MA, Paller AS. The role of abnormalities in the distal pathway of cholesterol synthesis in the Congenital Hemidysplasia with Ichthyosiform erythroderma and Limb Defects (CHILD) syndrome. *Biochim Biophys Acta* 2014; **1841**: 345–352.
- 3 du Souich C, Raymond FL, Grzeschik KH, *et al*. NSDHL-Related Disorders. 2011 Feb 1 [Updated 2018 Oct 25]. In: Adam MP, Ardinger HH, Pagon RA, *et al.*, editors. *GeneReviews*® [Internet]. Seattle (WA): University of Washington, Seattle; 1993–2020.
- 4 Paller AS, van Steensel MA, Rodriguez-Martín M, *et al*. Pathogenesis-based therapy reverses cutaneous abnormalities in an inherited disorder of distal cholesterol metabolism. *J Invest Dermatol* 2011; **131**: 2242–2248.
- 5 Bergqvist C, Abdallah B, Hasbani DJ, *et al*. CHILD syndrome: a modified pathogenesis-targeted therapeutic approach. *Am J Med Genet A* 2018; **176**: 733–738.

Supporting information

Additional Supporting Information may be found in the online version of this article:

File S1. Method of genetic test.

Allergic contact dermatitis to eyeglass frame nosepiece caused by tetraethylthiuram disulphide and resorcinol monobenzoate

Dear Editor,

Dermatitis on both sides of the nasal bridge should be suspected of allergic contact reaction to eyeglass frames.

A 41-year-old man, with no personal history of psoriasis or atopic dermatitis, was referred to our Contact Eczema Department complaining of an intermittent and bilateral eczema on both sides of his nasal bridge which had developed over the past 12 months. Regarding information of interest, he reported to wear a pair of glasses (unknown brand) when he taught physical education classes at school. Eczema disappeared or decreased its intensity during the vacation period as well as on weekends. Methylprednisolone aceponate 0.1% cream was applied with improvement of the dermatitis. Recurrence was the rule after wearing the same pair of glasses. Physical examination showed symmetric erythematous and edematous plaques on both sides of his nasal bridge (Fig. 1a), with superficial scaling. Patch tests were performed with the European

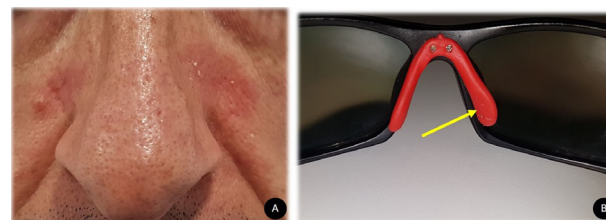


Figure 1 (a) Symmetric erythematous and edematous plaques on both sides of the nasal bridge. (b) Red nosepiece on glasses where shavings were taken to be used in patch testing

Table 1 Potential allergenic compounds in eyeglasses frames

Frame material	Contact allergen
Plastics and additives as plasticizers	Cellulose acetate
	Cellulose propionate
	Tricresyl phosphate
	Triphenyl phosphate
	Diethylhexyl phthalate
	Diethyl phthalate
	Dibutyl phthalate
	Glyceryl triacetate
	Toluene sulfonamide
	Butyl acrylate
New plasticizer	Triethylene glycol bis 2-ethylhexanoate ⁵
Metals	Copper
	Nickel
	Cobalt
	Chromium
	Palladium
	Aluminum
	Silver
	Gold
	Platinum
	Titanium
Acrylate	Solders
	Polymethyl methacrylate
Join memory metals	Methyl methacrylate
	Cyanoacrylate
Rubber components	Thiurams
	Silicone
	Polyvinyl chloride (F-PVC)
Metal coating	Neoprene
	Epoxy resin
Polyamides	Nylon
	Grilamid (Nylon 12)
UV stabilizers/UV inhibitors	Resorcinol
	Resorcinol monobenzoate
	Phenyl salicylate
	Carbon fiber
	<i>p</i> -Phenylenediamine
Composites	Disperse orange 3
	Disperse Red 17
	Disperse yellow 3
	Solvent orange 60
	Solvent Red 179
Polishes	Colophony
	Abietic acid
	Turpentine
	Iron oxide
Thermoplastic material	Acrylonitrile butadiene styrene
Surface metal and plastic spectacle frames	Polyurethane
Solvents	Acetone
	Ethylene acetate
	Ethylene glycol
Others	Tosylamide formaldehyde resin
	Bone
	Formaldehyde (synthetic horns)
	Leather (chamois leather)
	Wood

Comprehensive Baseline Series (Chemotechnique Diagnostics, Vellinge, Sweden), rubber additives series (Chemotechnique Diagnostics), plastic and glues series (Chemotechnique Diagnostics), disperse Red 1 and 17 1% pet, Basic Red 46 1% pet, Reactive Red 123 and 228 1% pet, Acid Red 118 and 359 5% pet (Chemotechnique Diagnostics), and shaving from the red nosepiece of the glasses “as is” (Fig. 1b, yellow arrow). The results were interpreted according to the criteria of the International Contact Dermatitis Research Group. Patch tests were read on day (D) 2 and D4. The patient showed positive patch test reaction to thiuram mix 1% pet, tetraethylthiuram disulfide 1% pet, resorcinol monobenzoate 1% pet, and methylisothiazolinone (MI) 0.02% aq. The nosepieces of the glasses “as is” (red piece) showed no patch test reaction. A solubilized extract of the red nosepiece was obtained with an ultrasonic probe device (UP400S, Hielscher, Teltow, Germany). Extract was performed in Finn Chamber and patch testing. The patient showed positive patch test reaction with ultrasonic extract on D2 and D4. Avoidance of wearing this pair of glasses resolved the problem. Patch test in 20 dermatitis patient controls showed no reaction to the same extract.

The main reviews have been published by Smith *et al.*,¹ Nakada *et al.*,² and more recently from Walsh *et al.*³ Potential allergenic compounds in eyeglasses frames are included in Table 1. TETD is an accelerator, activator, stabilizer, and vulcanizing agent for different rubber products. It is also used as fungicide, seed disinfectant, and alcohol deterrent (disulfiram). TETD is sensitive to light, the reason for which it requires UV-light absorbent additives to avoid deterioration, such as resorcinol monobenzoate. The MI role as a preservative could be considered.⁴ Personal care products were investigated, but MI was not identified in any of them. The relevance was considered unknown in our case.

Ultrasonic-assisted extraction with an ultrasonic probe device should be considered to study the nosepiece glasses when we cannot contact with the manufacturers, and the nosepieces of the glasses “as is” showed no patch test reaction.

Acknowledgments

We would like to acknowledge all staff working in the Contact Eczema and Immunoallergic Diseases Department, especially the specialized nurse Victor Alfredo Cassini Gómez de Cádiz.

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Conflict of interest: None.

Funding source: None.

doi: 10.1111/ijd.15105

References

- 1 Smith EL, Calnan CD. Studies in contact dermatitis. XVII-spectacle frames. *Trans St Johns Hosp Dermatol Soc* 1966; **52**: 10–34.
- 2 Nakada T, Maibach HI. Eyeglass allergic contact dermatitis. *Contact Dermatitis* 1988; **39**: 1–3.
- 3 Walsh G, Wilkinson SM. Materials and allergens within spectacle frames: a review. *Contact Dermatitis* 2006; **55**: 130–139.
- 4 El-Houri RB, Christensen LP, Persson C, *et al*. Methylisothiazolinone in a designer spectacle frame- a surprising finding. *Contact Dermatitis* 2016; **75**: 310–312.
- 5 Andersen KE, Vestergaard M, Christensen LP. Triethylene glycol bis(2-ethylhexanoate) - a new contact allergen identified in a spectacle frame. *Contact Dermatitis* 2014; **70**: 112–116.

Unilateral idiopathic acquired dermal melanocytosis on a palm: An extremely rare entity in a young woman

Dear Editor,

Dermal melanocytoses (DMs) are characterized by bluish-gray hyperpigmented macules. DMs are generally observed at birth and rarely in adolescence or adulthood and are called acquired dermal melanocytosis (ADM). ADM is extremely rare in the acral regions.^{1,2} Here, we present a 20-year-old Turkish woman diagnosed with ADM on the right palm.

A 20-year-old female patient presented to the dermatology outpatient clinic with discoloration on the right palm for 2 years. She was a student and ambidextrous. There were no triggering factors like inflammation, severe sunlight exposure, trauma, inflammatory dermatosis, chemical substance, metal contact, or use of drugs. Her family history was unremarkable, and she did not benefit from hydroquinone treatment. The lesions were asymptomatic. She had Fitzpatrick skin type III. On dermatological examination, bluish gray-colored hyperpigmented macular lesions were observed in a 2 × 2 cm area in diameter where the thenar crease and proximal transverse crease converged on the right palm. It was observed that gray-colored hyperpigmented macules with diameters approximately between 2 and 5 mm were scattered irregularly located in this localized area and concentrated on the thenar crease and proximal transverse crease (Fig. 1). No hyperpigmented lesions were observed on skin and mucous membranes of other parts of the body. On histopathological examination, dendritic melanocytes were observed in the dermis, especially concentrated around vascular structures (Fig. 2). With these findings, the patient was diagnosed with ADM.

Acquired dermal melanocytoses are very rare lesions characterized by hyperpigmented bluish-gray macules, usually seen in younger individuals.³ Dendritic, spindle-shaped melanocytes are found in the dermis and do not tend to form nests.¹ The color of the lesions varies according to the anatomical location where melanocytes are located in the dermis. ADMs are rarely seen in acral regions. This case seems to be the third case of unilateral palmar ADM in the known literature and first case from Europe.

Considering the cases of ADM involving the hands reported to date, it is observed that most of the cases are Asian, there is no gender difference, the lesions occur at young ages, and there is a tendency to appear on the dorsum of the hand and fingers. In addition, significant hyperpigmentation in palmar creases is remarkable in palmar region involvement.^{3,4} Hyperpigmentation concentrated in the palmar creases was detected in our case, in which dorsum of the hand and finger involvement was not observed.

The etiopathogenesis is not known. The lesions may occur as a result of the activation of melanocytes that remain latent in the dermis and cannot reach the epidermis while migrating from the neural crest during the embryological period. Severe sunlight exposure, some medications, and hormones may be responsible for the activation of latent melanocytes.⁵ The fact that it starts at a young age may suggest hormonal etiology. According to a proposed theory, dropping of epidermal melanocytes to the dermis, melanocyte migration from the hair follicle, or the reactivation of melanocytes remaining latent in the dermis may play a role in the formation of lesions.⁶ An increase in the

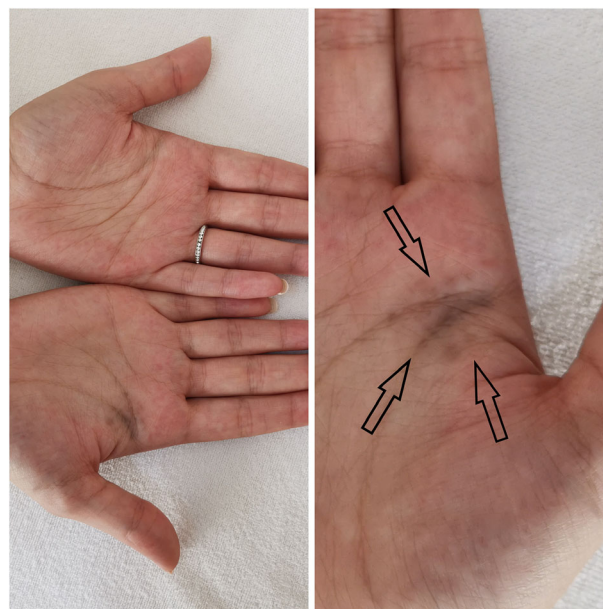


Figure 1 Bluish grey-colored macules on right palm. Arrows show hyperpigmented macules concentrated in palmar creases