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Fingerprint loss in two patients with Sézary syndrome: clinical and dermatoscopic evaluation

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Dear Editors,

Sézary syndrome (SS) is an aggressive leukemic cutaneous T-cell lymphoma variant that is characterized by circulating atypical T cells (Sézary cells), diffuse erythema, and severe pruritus with or without lymphadenopathy [1]. Palmoplantar keratoderma is seen in over 60 % of the patients with SS [2]. We herein report a novel clinical observation of fingerprint loss in two patients with long-standing SS.

In the first patient, a 77-year-old woman with a history of long-standing SS, partially controlled with topical corticosteroids, oral romidepsin and lenalidomide, we noticed bilateral loss of finger dermatoglyphics (Figure 1a). Ink blot of the patient's thumbs showed complete loss of the fingerprints (Figure 1b). Dermatoscopic examination demonstrated effacement of the typical acral skin markings of ridges, furrows and eccrine duct openings and the presence of linear red dots (Figure 1c). Retrospective evaluation of the clinical images, taken during her visits at the cutaneous lymphoma clinic, showed that the dermatoglyphics loss developed in the patient gradually over a period of six years. The patient herself did not notice the change in her fingers dermatoglyphics and did not encounter any daily life difficulty due to this condition; however, she was not required to use any fingerprint-based identification in recent years.

Following this observation, we identified a second patient with SS and fingerprint loss. This 49-year-old man had a long history of SS treated with multiple lines of therapy. He returned to a follow-up visit at our clinic with diffuse erythema while treated with extracorporeal photopheresis and oral bexarotene. Physical examination of his fingers showed loss of the finger dermatoglyphics (Figure 2a). Dermatoscopy showed similar findings as seen in the other patient (Figure 2b). This patient had not noticed any change in his fingerprints nor any related effects.

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Conflict of interest

None.

Acquired adermatoglyphia, or loss of fingerprints, is a rare condition that has been reported as a potential consequence of dermatitis, skin infections (e.g. palmar warts, herpetic whitlow, pitted keratolysis), bullous dermatoses and other certain dermatologic conditions or as a drug-related event [3]. Severe fingerprint quality loss has been reported to be a frequent adverse event during capecitabine treatment [4, 5]. Capecitabine associated fingerprints loss may occur following hand-foot syndrome (HFS) or with no prior HFS [3, 5].

Adermatoglyphia has not been reported to be associated with romidepsin or lenalidomide treatments received by the first patient during the period she lost her fingerprints. We hypothesize that in the two reported patients, fingerprint loss was a result of the frequent cycles of keratoderma and subsequent significant palmar exfoliation, a clinical presentation that is typically seen in patients with SS [2]. The chronic use of topical corticosteroids might have also played a role in the fingerprint loss in these two cases [6].

Our observation of fingerprint loss in two patients with SS highlights the need to further evaluate the possible development of adermatoglyphia in association with SS.

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References

1. Nicolay JP, Felcht M, Schledzewski K et al. Sézary syndrome: old enigmas, new targets. *J Dtsch Dermatol Ges* 2016; 14(3): 256–64. [PubMed: 26972187]
2. Martin SJ, Duvic M. Prevalence and treatment of palmoplantar keratoderma and tinea pedis in patients with Sézary syndrome. *Int J Dermatol* 2012; 51(10): 1195–8. [PubMed: 22994666]
3. Sarfraz N Adermatoglyphia: barriers to biometric identification and the need for a standardized alternative. *Cureus* 2019; 11(2): e4040. [PubMed: 31011502]
4. Chavarri-Guerra Y, Soto-Perez-de-Celis E. Images in clinical medicine. Loss of fingerprints. *N Engl J Med* 2015; 372: 0.
5. van Doorn L, Veelenturf S, Binkhorst L et al. Capecitabine and the risk of fingerprint loss. *JAMA Oncol* 2017; 3(1): 122–3. [PubMed: 27560202]
6. Sergeant A, McPhee N, Holme SA. Acquired loss of fingerprints: do topical corticosteroids play an aetiological role? *Clin Exp Dermatol* 2012; 37(6): 679–80. [PubMed: 22329748]

Figure 1.

Acquired adermatoglyphia in a 77-year-old woman with Sézary syndrome (a). Ink blot of the patient's thumb demonstrates the complete effacement of the fingerprint (b). Dermatoscopic examination under non-polarized light demonstrates linear red dots with loss of the ridges, furrows and eccrine sweat glands' openings that are typically seen in normal acral skin (as demonstrated by the healthy examiner's finger at the bottom) (c).

Figure 2. Adermatoglyphia in a 49-year-old male with Sézary syndrome (a). Dermatoscopic examination shows linear red dots and no ridges, furrows or eccrine sweat glands' openings (b).