

Non-pruritic lichen planus in sickle cell anemia: Is there a role for hydroxyurea?

M Niveditha¹, Betsy Ambooken¹, K Devi¹, Asokan Neelakandhan²

¹Department of Dermatology, Venereology And Leprosy, Government Medical College, Thrissur, India, ²The Principal, Government Medical College, Kozhikode, India

Corresponding author: M. Niveditha, MD, E-mail: nivedithapknm@gmail.com

Sir,

Herein, we report a case of non-pruritic lichen planus in a 21-year-old male who had been on treatment with hydroxyurea for sickle cell anemia since nine years of age.

A 21-year-old male was admitted with multiple non-pruritic, hyperpigmented, raised lesions in a generalized distribution of three weeks' duration. He was diagnosed to have had sickle cell anemia since nine years of age and had been on treatment with hydroxyurea 500 mg daily and iron and folic acid supplements since then. On examination, he had pallor, bilateral pitting and pedal edema, and there were generalized, bilaterally symmetrical, well-defined, violaceous, and hyperpigmented discrete and confluent papules and plaques of sizes varying from 0.5 x 0.5 cm to 2 x 2 cm with adherent grayish-white scales and Koebner's phenomenon (Figs. 1a and 1b). The mucous membranes and nails were normal. The differential diagnosis considered were acute generalized lichen planus and lichenoid eruption to hydroxyurea.

Peripheral blood smear revealed microcytic hypochromic anemia with poikilocytes such as elliptocytes, pencil-shaped cells, target cells, and some sickle cells. Sickling test was positive. Serology for syphilis, HIV, hepatitis B, and hepatitis C were negative. Histopathological examination of the lesional skin in low power showed acanthosis, focal hypergranulosis, saw toothing of rete ridges, basal cell degeneration, and band-like lymphoplasmacytic infiltration in the dermo-epidermal junction (Fig. 2a). Civatte bodies and pigment incontinence were seen in high power view

(Fig. 2b). The diagnosis of acute generalized lichen planus was made. Due to the generalized and extensive nature of the disease, systemic steroids were initiated. As the sickle cell disease was active and histopathology showed no features of lichenoid eruption, hydroxyurea was not discontinued. The skin lesions resolved with hyperpigmentation and there was no recurrence on stopping systemic steroids (Fig. 3).

There are several reports of lichenoid eruption to hydroxyurea [1]. However, in this case, there were no clinical or histopathological features to suggest lichenoid eruption. Furthermore, there was no exacerbation or recurrence of skin lesions in spite of continuing hydroxyurea. Itching is one of the



Figure 1: (a) Generalized, bilaterally symmetrical, well-defined, violaceous and hyperpigmented, discrete and confluent papules and plaques of sizes varying from 0.5 x 0.5 cm to 2 x 2 cm with adherent grayish-white scales and Koebner's phenomenon on the trunk. (b) Bilaterally symmetrical, well-defined, violaceous and hyperpigmented, discrete and confluent papules and plaques of sizes varying from 0.5 x 0.5 cm to 2 x 2 cm with adherent grayish-white scales on the legs.

How to cite this article: Niveditha M, Ambooken B, Devi K, Neelakandhan A. Non-pruritic lichen planus in sickle cell anemia: Is there a role for hydroxyurea? Our Dermatol Online. 2025;16(1):121-122.

Submission: 02.06.2024; **Acceptance:** 29.10.2024

DOI: 10.7241/ourd.20251.30

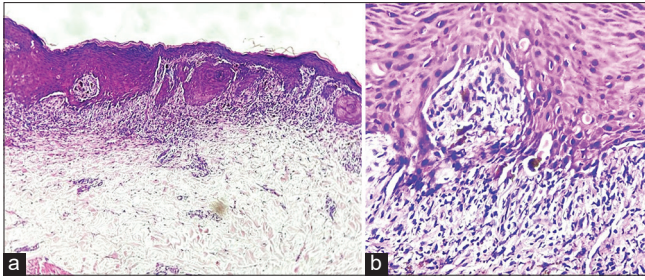


Figure 2: (a) Epidermis showing acanthosis, focal hypergranulosis, saw tootching of rete ridges, basal cell degeneration, and band-like lymphoplasmacytic infiltration in the dermo-epidermal junction (H&E, 40x). (b) Epidermis showing acanthosis, focal hypergranulosis, saw tootching of rete ridges, basal cell degeneration, and band-like lymphoplasmacytic infiltration in the dermo-epidermal junction with Civatte bodies and pigment incontinence (H&E, 100x).



Figure 3: Skin lesions subsided with hyperpigmentation.

important symptoms of lichen planus. It is produced when pruritogenic stimuli activate primary afferent C fibers. Endogenous pruritogens, such as histamine, kinins, proteases, neurotrophins, some opioids, and cytokines produced by keratinocytes, leukocytes, mast cells, fibroblasts, endothelial cells, and cutaneous nerves directly activate the itch-sensitive C-fibers or indirectly induce the release of pruritogenic mediators and modulators from other cells. Some of the newer

pathogenetic factors for itch in lichen planus include increased levels of IL-31 and TNF- α ; increased expression of IL-31 receptors and protease activated receptors; and the activation of μ -opioid receptors and toll-like receptors (TLRs) [2].

Hydroxyurea has anti-inflammatory action by decreasing the levels of TNF- α and IL-6. Treatment with hydroxyurea leads to a significant decrease in neutrophil-to-lymphocyte ratio, which is positively correlated with serum levels of CRP, TNF- α , and IL-6 [3]. In our patient, hydroxyurea could have made the lichen planus non-pruritic by decreasing the levels of the chemical mediators of itch. This needs to be confirmed by further studies. Drugs such as hydroxychloroquine, dapsone, PUVA, phenytoin, and isotretinoin known to cause lichenoid eruption or trigger lichen planus have found a place in the treatment of the same [4,5]. Whether hydroxyurea like these drugs have a therapeutic role (for pruritus) in lichen planus also needs to be explored.

REFERENCES

1. Daoud MS, Gibson LE, Pittelkow MR. Hydroxyurea-induced dermatopathy. *J Am Acad Dermatol.* 1997;36:178-82.
2. Welz-Kubiak K, Reich A. Mediators of pruritus in lichen planus. *Autoimmune Diseases.* 2013;2013:1-4.
3. Zahran AM, Nafady A, Saad K, Hetta HF, Abdallah AEM, Abdel-Aziz SM, et al. Effect of hydroxyurea treatment on the inflammatory markers among children with sickle cell disease. *Clin Appl Thromb Hemost.* 2020;26:1076029619895111.
4. Cribier B, Frances C, Chosidow O. Treatment of lichen planus. *South Med J.* 1942;35:918-20.
5. Boch K, Langan EA, Kridin K, Zillikens D, Ludwig RJ, Bieber K. Lichen planus. *Front Med.* 2021;8:1-17.

Copyright by M. Niveditha, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Source of Support: This article has no funding source.

Conflict of Interest: The authors have no conflict of interest to declare.