Case Report

Hypertrophic Lichen Planus in a 38-year-old Filipino Male: A Case Report

Janine Bianca M. Acoba[1], Ma. Margarita Isabel C. Tanchiong[2], Maria Jasmin J. Jamora[1,2,3]

[1]Skin and Cancer Foundation, Inc., Pasig City, [2]Department of Dermatology, Makati Medical Center, Makati, [3]Department of Dermatology, Quirino Memorial Medical Center, Quezon City, Philippines

Abstract

Hypertrophic lichen planus (HLP) is a papulosquamous eruption presenting with extremely pruritic hyperkeratotic flat-topped papules, plaques, and nodules. This is a case of 38-year-old male who presented with a 2-month history of generalized erythematous-to-hyperpigmented papules, patches, and plaques topped with white-to-gray oyster shell-like scales on a background of hyperpigmented macules and patches. There was no involvement of the conjunctival, otic, oral, and genital mucosae, and palmar and plantar aspects of the hands and feet. Dermoscopy showed reticular pearly white structures corresponding to the Wickham striae, comedo-like openings, blue-gray dots, brownish-black dots, and scales. Histopathologic examination revealed marked compact hyperkeratosis, wedge-shaped hypergranulosis, irregular saw-toothed epidermal acanthosis, scattered dyskeratotic keratinocytes, and superficial perivascular lichenoid infiltrate of lymphocytes, histiocytes, and melanophages. The patient was managed as a case of HLP. He was started on methotrexate 10 mg per week, bath psoralen photochemotherapy (PUVA) three times a week, betamethasone valerate 1mg/g cream twice a day for 2 weeks alternating with tacrolimus 0.1% ointment twice a day for another 2 weeks, 10% lactic acid, emollients, and sunscreen. After 6 months of treatment, there was almost 80% improvement of lesions and relief of pruritus.

Keywords: 8-methoxsalen, hypertrophic lichen planus, methotrexate, psoralen photochemotherapy

Address for correspondence: Dr. Janine Bianca M. Acoba, Skin and Cancer Foundation, Inc. Unit 1611 Medical Plaza Ortigas, San Miguel Avenue, San Antonio Village, Pasig City, Metro Manila, Philippines.
E-mail: janine.acoba@gmail.com

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Introduction

Hypertrophic lichen planus (HLP) is a papulosquamous eruption presenting with extremely pruritic hyperkeratotic flat-topped papules, plaques, and nodules commonly affecting the wrists, interphalangeal joints, and the anterior lower legs.[1]

Case Report

A 38-year-old male, with hypertension, bronchial asthma, dyslipidemia, and hyperuricemia, presented with a 2-month history of extremely pruritic plaques with scales. He had no history of fever, malaise, photosensitivity, joint pains, and oral or genital ulcers. The patient had been taking amlodipine, febuxostat, ezetimibe, and fenofibrate for the past 3 years. His medications were shifted to a different drug class to rule out the possibility of drug eruption, yet his lesions persisted.

On physical examination, there was almost generalized involvement. Lesions consisted of multiple well-defined

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erythematous-to-hyperpigmented papules, patches, and plaques with white to gray oyster shell-like scales on a background of hyperpigmented macules and patches [Figure 1]. There were no lesions on the conjunctival, otic, oral, and genital mucosae, and palmar and plantar aspects of the hands and feet.

Dermoscopy revealed reticular pearly white structures corresponding to Wickham striae, comedo-like openings, blue-gray dots, brownish-black dots, and scales [Figure 2a and b]. Trichoscopy also revealed the presence of peripilar cast, perifollicular scales, and blue-gray dots, which were consistent with the findings of lichen planopilaris [Figure 2c].

Initial biopsy, which was taken from the lower extremity, showed basket-weave orthokeratosis with mounds of parakeratosis. There was epidermal acanthosis, scattered dyskeratotic keratinocytes seen singly and in groups, and a broad front of vacuolar interface change and lymphocyte exocytosis and satellite necrosis. Within the dermis were superficial and deep perivascular, interstitial, and periappendageal infiltrates of mainly lymphocytes with extravasated erythrocytes. The initial histopathologic diagnosis was vacuolar interface dermatitis consistent with pityriasis lichenoides et varioliformis acuta.

He was started on bath psoralen photochemotherapy (PUVA) three times a week and was prescribed oral doxycycline 100 mg twice a day, loratadine 5 mg and betamethasone 250 μg once a day for 7 days, clobetasol 500 μg/g ointment twice a day for the body, and desonide 500 μg/g cream twice a day for the face. However, there was minimal improvement after 2 months. He was shifted to methotrexate 10 mg per week and folic acid 5 mg once a day, and a repeat biopsy was done on the upper extremity.

On repeat biopsy, there was marked compact hyperkeratosis, follicular plugging, focal parakeratosis with wedge-shaped hypergranulosis, and irregular saw-toothed epidermal acanthosis. There were many scattered dyskeratotic keratinocytes at all levels of the epidermis. There were superficial perivascular lichenoid infiltrates of lymphocytes, histiocytes, and many melanophages [Figure 3]. The final diagnosis was HLP.

The patient continued receiving methotrexate at a dose of 10 mg per week. The dose was adjusted accordingly based on his response, and laboratory parameters were

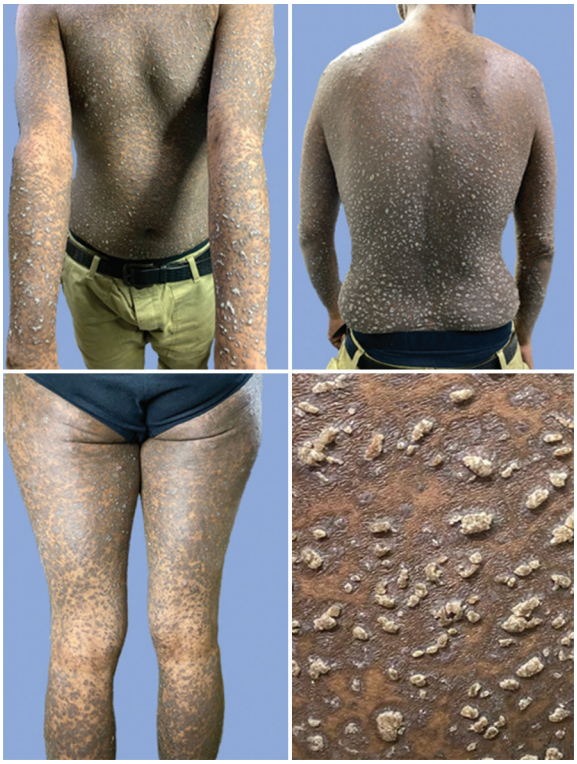

Figure 1: Physical examination revealed multiple well defined erythematous to hyperpigmented papules and plaques with white to gray oyster shell like scales on a background of hyperpigmented macules and patches all over the body

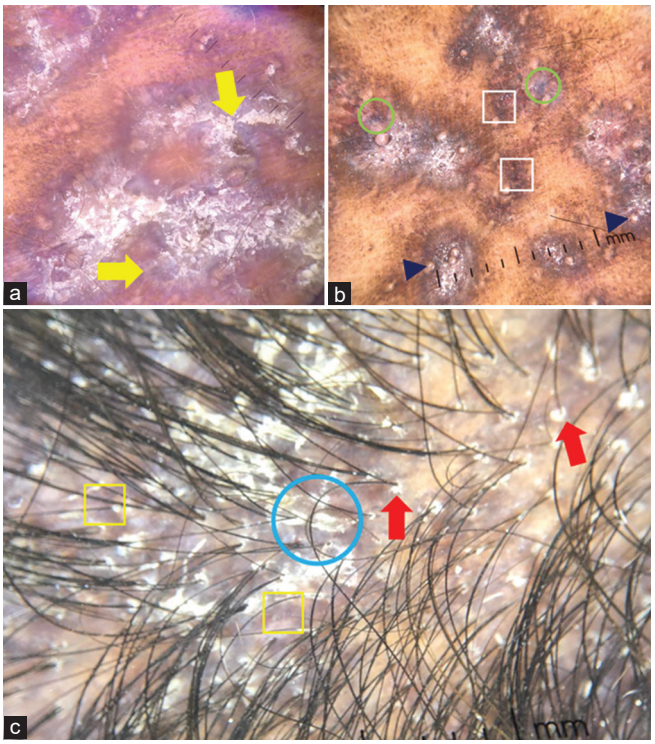

Figure 2: (a) Dermoscopy showing pearly white structures (yellow arrow). (b) Dermoscopy showing comedo-like openings (arrowhead), blue-gray dots (green circle), brownish-black dots (white square), and scales. (c) Trichoscopy showing peripilar cast (blue circle), perifollicular scales (red arrow), and blue-gray dots (yellow square)

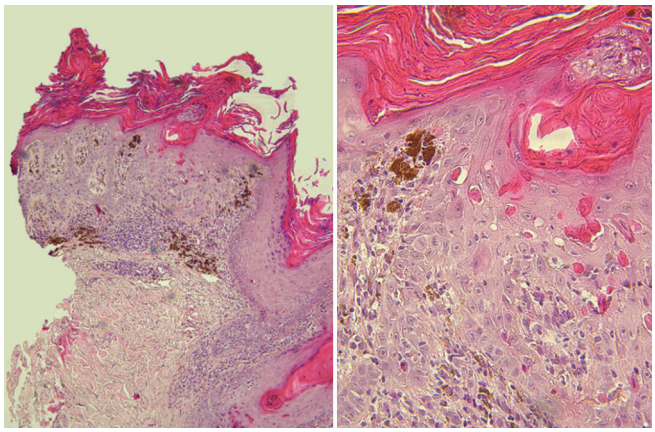

Figure 3: Repeat biopsy revealed marked compact hyperkeratosis, follicular plugging, focal parakeratosis with wedge-shaped hypergranulosis, irregular saw-toothed epidermal acanthosis, scattered dyskeratotic keratinocytes at all levels of the epidermis, and lichenoid infiltrates of lymphocytes, histiocytes, and melanophages

monitored regularly. He continued bath PUVA, where he was soaked in a tub with 4cc 8-methoxsalen (8-MOP) and then exposed to UVA three times a week. His topical medications included betamethasone valerate 1 mg/g cream twice a day for 2 weeks alternating with tacrolimus 0.1% ointment twice a day for another 2 weeks, 10% lactic acid lotion twice a day, emollients, and sunscreen. The patient was monitored monthly for his response. After 6 months of treatment, there was almost 80% improvement. Most of the lesions cleared, leaving deeply hyperpigmented macules and patches [Figure 4]. There was also relief of pruritus, leaving the patient with an improved quality of life.

Discussion

Lesions of HLP are usually distributed in a symmetrical pattern and are often characterized by its resemblance to the extrusive forms of an igneous rock.[1] Its prevalence is largely unknown.[2]

Methotrexate, a synthetic analog of folate, may be used as a first-line systemic therapy for HLP.[3] It showed better results, fewer side effects, and lower relapse rates compared to systemic corticosteroids.[3] It is an effective and safe option for patients as long as biochemical and hematological parameters are regularly monitored.[3]

Systemic corticosteroids can be used as a second-line or third-line treatment.[3] Mini-pulse therapy with either oral betamethasone or intravenous methylprednisolone is preferred over moderate daily dosing.[3] However, its use may be limited by the potential for relapse posttreatment and its associated adverse effects.[3]

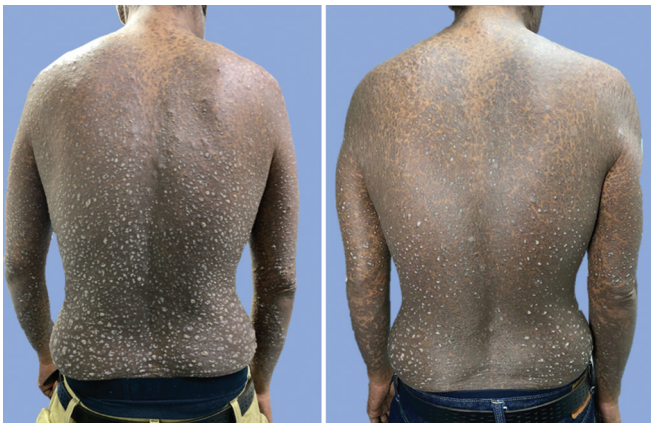

Figure 4: Comparison of lesions on the back during the initial consultation (left) and after 6 months of treatment (right) showed some areas of clearing leaving deeply hyperpigmented macules and patches

Other systemic medications that can be used in HLP include systemic retinoids such as acitretin or alitretinoin.[3] In patients who do not respond to these medications, low molecular weight heparin may also be considered.[3]

While narrow-band ultraviolet B (NBUVB) remains the preferred phototherapeutic option for cutaneous LP in general, there are still no randomized controlled trials that compare its efficacy with PUVA.[4] A study also found that patients with LP had a better initial clinical response to oral PUVA than NBUVB.[5] However, long-term follow-up showed no significant difference between NBUVB and PUVA with regard to the overall response rate.[5] Only two trials included patients with HLP. Based on these studies, patients with HLP did not respond to NBUVB and had a partial response to PUVA.[6]

In terms of topical interventions, class I and class II topical corticosteroids are routinely used by clinicians as the first-line topical therapy for LP despite the lack of strong evidence that supports its use.[4] Topical calcineurin inhibitors may also be given as a steroid-sparing agent.[4]

The treatment of HLP is challenging because no treatment option has been found to be universally effective.[7] A combination of systemic, phototherapeutic, and topical management has been used in this case due to the extent of lesions and its impact on the quality of life of the patient.

Malignant transformation of HLP to squamous cell carcinoma has also been reported in the literature.[2] Hence,

it is prudent to closely monitor the patient even after therapy to screen for any suspicious lesions.

Conclusion

Low-dose methotrexate combined with bath PUVA, topical corticosteroids, and topical calcineurin inhibitor as a steroid-sparing agent may be used as a treatment regimen in patients with HLP.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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