Wickham striae on skin appendages: a helpful dermoscopic feature

Laura Fagotto,[1] Laura Gnesotto,[1] Colombina Vincenzi,[2,3] Bianca Maria Piraccini,[2,3] Luigi Naldi,[4] Andrea Sechi[2-4]

[1]Dermatology Unit, Department of Medicine, University of Padova; [2]Dermatology Division, IRCCS University Hospital of Bologna S. Orsola-Malpighi Polyclinic, Bologna; [3]Department of Experimental, Diagnostic and Specialty Medicine, Alma Mater Studiorum University of Bologna; [4]Dermatologic Unit, San Bortolo Hospital, Vicenza, Italy

Abstract

Lichen planus (LP) is a chronic inflammatory disease, clinically characterized by purpuric, itchy papules that typically spread on the trunk and extremities. Other body sites can also be affected, including mucosal membranes, nails, and the scalp. The use of dermoscopy is essential in determining the diagnosis of LP, as it may highlight pathognomonic features such as Wickham striae (WS). WS are thin, pearly white structures arranged in a reticular pattern that is observed over LP lesions and histologically correspond to epidermal hypergranulosis. WS is usually most visible on the oral mucosa but can also cover almost every active LP papule. Here, we report two cases of biopsy-proven LP that show WS on dermoscopy in two specific sites: the scalp and proximal nail fold.

Correspondence: Andrea Sechi, Dermatologic Unit, San Bortolo Hospital, Vicenza, Italy.
E-mail: andrea.sechi@aulss8.veneto.it

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Introduction

Lichen planus (LP) is a chronic inflammatory disorder that can affect various parts of the body, including the skin, mucous membranes, nails, and scalp. The condition is characterized by itchy, purple-colored papules and plaques that typically appear on the wrists, lower back, and ankles. Associated nail changes can include ridging, distal splitting, thinning, subungual hyperkeratosis, pterygium formation, and nail loss. LP of the scalp initially causes hair loss and keratotic follicular papules, which can lead to scarring alopecia if left untreated. LP affects between 0.1% and 4% of the general population and is more common in middle-aged women.[1,2] The pathophysiology of LP involves a T-lymphocyte-mediated reaction, and several factors have been proposed as potential triggers, including viral agents such as hepatitis C virus, contact allergens like dental amalgam, gold, nickel, cobalt, drugs, radiotherapy, and stress.[3] Diagnosis of LP is primarily based on the typical clinical and dermoscopic appearance. Dermoscopy of LP lesions reveals pearly white crossing lines known as Wickham striae (WS), red dots, radial capillaries, and brownish diffuse or deeper dotted pigmentation patterns. WS is pathognomonic for LP and helps in distinguishing this disorder from other scaly dermatoses.[4,5] The most commonly used treatment for LP comprises topical and systemic corticosteroid administration while, in case of refractory disease, topical calcineurin inhibitors or systemic immunosuppressive agents should be used.[6] Nail LP can be challenging to cure, and topical treatment is rarely effective. Thus, intralesional and intramuscular triamcinolone acetonide should be considered primarily to avoid permanent nail destruction. Second-line options include oral retinoids and immunosuppressive medications.[7]

Case Report

Patient 1 was a 61-year-old woman who had been experiencing intensely itchy, purplish papules and plaques for four months that had progressively spread to her trunk and proximal upper and lower limbs. During the physical examination, an alopecic patch was observed on her scalp (Figure 1). Trichoscopy showed perifollicular hyperkeratosis and shiny, white-colored reticulated lines highly suggestive of WS (Figure 2). Histological examination of scalp and skin specimens confirmed the diagnosis of LP, revealing a banded lichenoid infiltrate, leukocyte exocytosis, epidermal hypergranulosis, and focal basal layer vacuolization. The scalp biopsy showed a perifollicular lichenoid infiltrate around the mid-part of the hair follicle with loss of sebaceous glands. The patient received three intramuscular injections of triamcinolone acetonide

40 mg/mL monthly, which led to an almost complete remission of the LP on her trunk and limbs. The single scarring alopecia patch on her scalp persisted without signs of inflammation. Patient 2 was a 42-year-old woman who had been experiencing whitish translucent lesions on the floor of her mouth for three months, along with pain and a burning sensation during meals. She also complained about toenail alterations. The patient reported no previous history of new drug intake, alcohol, or smoking consumption. Crossing whitish striae coalescing in a plaque were noted on the left oral floor. Furthermore, the affected nails were thin and presented longitudinal fissures and initial dorsal pterygium.

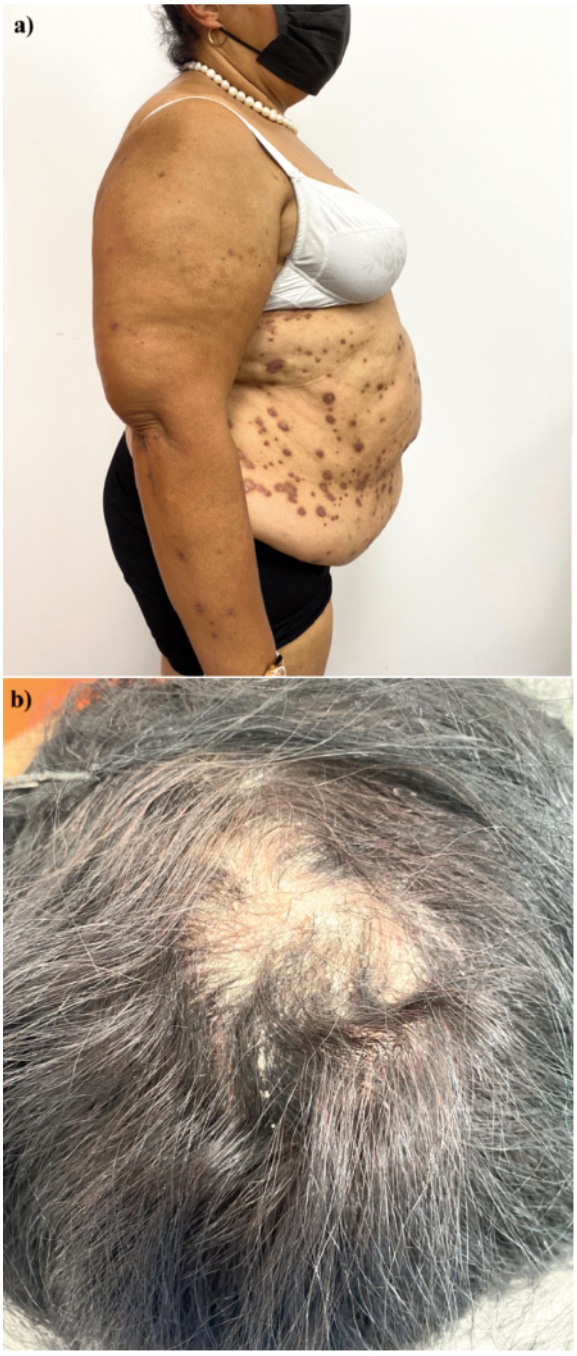

Figure 1. a) Scattered pruriginous hyperpigmented papules, mostly affecting the trunk; b) Alopecic scaling patch on the vertex.

Dermoscopy detected reticular white lines at the proximal nail fold, congruent with WS (Figure 3). An oral biopsy was performed, showing hyperkeratosis, mild epithelial acanthosis, a lichenoid band-like infiltrate in the submucosa, and vacuolization of the basal

Figure 2. Dermoscopy with a liquid interface of the alopecic patch showed perifollicular scale (yellow arrow) and thin, pearly white striae arranged in a reticular configuration, suggestive for Wickham striae. Note the coexistence of scratch-induced erosion in the middle part.

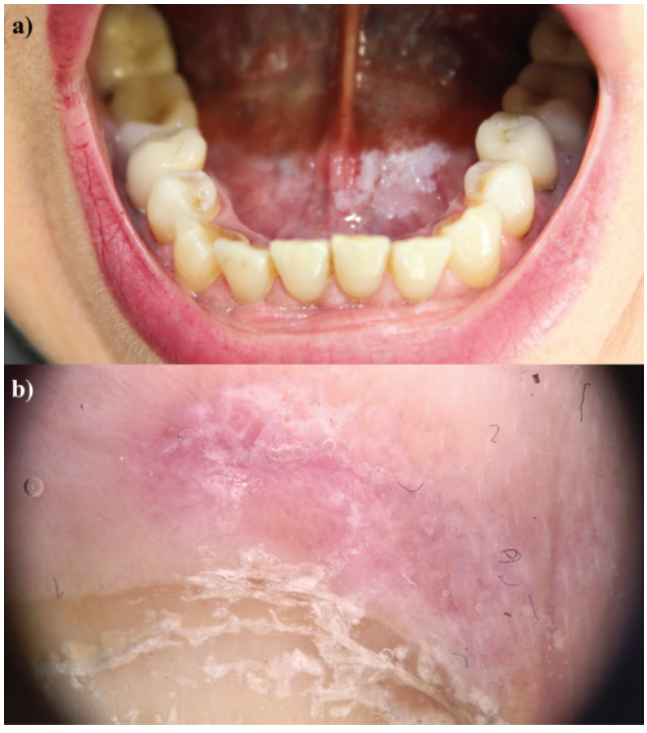

Figure 3. a) Lichen planus of the oral cavity with Wickham striae coalescing in a white plaque on the left oral floor; b) Dermoscopy with a liquid interface of the proximal nail fold of the second toenail showing initial dorsal pterygium and thin whitish striae arranged in a reticular pattern, consistent with Wickham striae.

layer, confirming the diagnosis of LP. The patient received three intramuscular injections of triamcinolone acetonide, which led to a progressive resolution of mucosal and cutaneous symptoms. The proximal part of the toenails showed normal regrowth and was followed up and treated with further intralesional steroid injections.

Discussion

Louis Frédéric Wickham first described the fine white lines that cover cutaneous papules and oral mucosal lesions of LP in 1895, coining the term "Wickham striae".[8] WS are a hallmark sign of LP and are useful in differential diagnosis. In cutaneous LP, WS are typically seen as white streaks in a reticular pattern, but other shapes have also been reported, including circular, radial streaming, linear, globular, veil-like, leaf venation, and starry sky/white dots. Oral WS typically manifests bilaterally as lacy networks or in tree-like arrangements.[9,10] Several theories have been proposed to explain WS formation. Darier *et al*.[11] attributed them to an increased thickness of the granular cell layer, while Summerly and Wilson-Jones associated WS development with a focal rise in disease activity,[12] as evidenced by granulosis, marked localized acanthosis, and enhanced formation of colloid bodies. Ryan suggested a third pathological factor,[13] arguing that the lack of dermal vessels in the area of LP may contribute to WS formation. WS disappears in treated lesions, and for this reason, they may have significant prognostic value.[14] Dermoscopy aids in the clinical diagnosis of LP, along with the lesions' distinctive shape. Nevertheless, atypical appearances may require histopathologic confirmation.[15] WS in cutaneous LP may resemble scaly lesions of drug-induced skin reactions, guttate psoriasis, discoid lupus erythematosus, pityriasis rosea, and graft-versus-host disease. Conversely, WS in oral LP should be distinguished from leukoplakia, frictional keratosis, and oral lichenoid eruptions.[9] While WS has been widely described on skin and oral mucosal lesions in LP, literature concerning dermoscopic features in other sites is scarce. To the best of our knowledge, the description of WS on the scalp and proximal nail fold has not been previously reported. Both of our patients showed a good response to systemic triamcinolone acetonide.

Conclusions

In conclusion, our report highlights that WS may also appear in unusual areas, such as skin appendages. Dermoscopy is a simple and non-invasive technique that can aid in the diagnosis of LP, improving the recognition of unapparent WS. Literature data concerning the localization of WS on skin appendages are limited, and physicians should not overlook these anatomic sites when examining patients with suspected LP.

References

1. Boyd AS, Neldner KH. Lichen planus. J Am Acad Dermatol 1991;25:593-619.
2. Usatine RP, Tinitigan M. Diagnosis and treatment of lichen planus. Am Fam Physician 2011;84:53-60.
3. Alrashdan MS, Cirillo N, McCullough M. Oral lichen planus: a literature review and update. Arch Dermatol Res 2016;308:539-51.
4. Lallas A, Zalaudek I, Argenziano G, et al. Dermoscopy in general dermatology. Dermatol Clin 2013;31:679-94.
5. Lallas A, Kyrgidis A, Tzellos TG, et al. Accuracy of dermoscopic criteria for the diagnosis of psoriasis, dermatitis, lichen planus and pityriasis rosea. Br J Dermatol 2012;166:1198-205.
6. Atzmony L, Reiter O, Hodak E, et al. Treatments for Cutaneous Lichen Planus: A Systematic Review and Meta-Analysis. Am J Clin Dermatol 2016;17:11-22.
7. Iorizzo M, Tosti A, Starace M, et al. Isolated nail lichen planus: An expert consensus on treatment of the classical form. J Am Acad Dermatol 2020;83:1717-23.
8. Steffen C, Dupree ML. Louis-Frédéric Wickham and the Wickham's striae of lichen planus. Skinmed 2004;3:287-9.
9. Sachdeva S, Sachdeva S, Kapoor P. Wickham striae: etiopathogenesis and clinical significance. Indian J Dermatol 2011;56:442-3.
10. Rouai M, Litaiem N, Hammami H, et al. Dermoscopic features of mucosal lichen planus. Int J Dermatol 2021;60:1368-72.
11. Rivers JK, Jackson R, Orizaga M. Who was Wickham and what are his striae? Int J Dermatol 1986;25:611-3.
12. Summerly R, Wilson Jones E. The Microarchitecture of Wickham's Stirae. Trans St Johns Hosp Dermatol Soc 1964;50:157-61.
13. Ryan TJ. The direction of the growth of the epithelium. Br J Dermatol 1966;78:403-15.
14. Roja Renuka S, Manoharan K. Dermoscopy-a simple diagnostic and prognostic tool in lichen planus. QJM 2022;115:109-10.
15. Papageorgiou C, Apalla Z, Lazaridou E, et al. Atypical case of lichen planus recognized by dermoscopy. Dermatol Pract Concept 2016;6:39-42.