Cureus
Part of SPRINGER NATURE

Open Access Case Report

A Rare Histopathological Finding of an Intradermal Nevus Coexisting With Lichen Planus in an Indian Woman

Review began 10/22/2026
Review ended 11/16/2026
Published 11/24/2026

© **Copyright** 2026
Patel et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.120191

Tarang Patel [1], Avani Dangar [1], Gyanendra Singh [1]

1. Pathology, All India Institute of Medical Sciences, Rajkot, Rajkot, IND

Corresponding author: Tarang Patel, tarangpatel_86@yahoo.co.in

Abstract

The simultaneous coexistence of an intradermal melanocytic nevus with overlying lichenoid inflammation is an exceedingly uncommon clinical entity, with sparse documentation in the Indian population. A 31-year-old Indian woman presented with a 12- to 13-month history of generalized, severe pruritus accompanied by multiple violaceous, flat-topped papules and plaques distributed across her extremities, trunk, palms, and soles. To evaluate the widespread lesions and rule out differential diagnoses, diagnostic punch biopsies were obtained from four separate anatomical sites (left buttock, left back, right medial sole, and right abdomen). Histopathological examination confirmed characteristic features of lichen planus in three specimens. Notably, the biopsy from the right abdominal papule incidentally revealed a well-circumscribed intradermal melanocytic nevus underlying the lichenoid interface dermatitis. The patient was managed conservatively for the benign incidental intradermal nevus, while her widespread lichen planus was treated with a combination of topical corticosteroids, emollients, oral prednisolone, hydroxyzine, and adjunctive vitamin and neuropathic support. Because the patient missed her scheduled 15-day follow-up appointment, the posttreatment clinical response could not be formally assessed. This case highlights a rare collision phenomenon and emphasizes the diagnostic value of multiple-site punch biopsies in managing widespread lichenoid eruptions.

Categories: Pathology, Dermatology
Keywords: coexistence, histopathology, intradermal nevus, lichen planus, violaceous

Introduction

An intradermal nevus is a benign melanocytic nevus composed of clusters of nevus cells within the dermis and may be congenital or acquired [1]. Melanocytic nevi are histologically classified into three types based on the location of nevus cells: junctional nevi, intradermal nevi, and compound nevi. They are usually acquired, manifest by age 20, and are most common on the head, neck, and trunk [2]. Conversely, lichen planus is a chronic, cell-mediated inflammatory mucocutaneous disorder characterized by a cytotoxic T-lymphocyte (CD8+ T-cell) response targeting basal keratinocytes, producing classic interface dermatitis affecting the skin, mucous membranes, or both, and represents a well-recognized idiopathic papulosquamous dermatosis in adult populations worldwide [2,3]. Coexistence of lichen planus over a preexisting melanocytic nevus is considered exceedingly rare and is described mainly in isolated case reports. Recognizing concurrent lichenoid inflammation over a melanocytic nevus holds significant clinical importance, as inflammation-induced architectural distortion, reactive atypia, or regression phenomena can closely mimic malignant melanoma or dysplastic nevi [2,3]. We report a rare case of widespread lichen planus coexisting with an incidental intradermal nevus over the right abdomen, confirmed via multisite punch biopsy.

Case Presentation

A 31-year-old woman presented to the gynecology department with complaints of noninfectious vaginal discharge and hirsutism. The patient also had a 12-month history of multiple violaceous, pruritic papules and plaques involving the extremities, trunk, palms, and soles. The gynecologist advised a KOH examination, Gram stain, *Treponema pallidum* hemagglutination assay, routine urine examination and microscopy, blood glucose tests, and other laboratory investigations, all of which were within normal limits.

Table *1* summarizes the baseline hematological investigations performed to evaluate the patient's systemic status and rule out underlying hematological abnormalities.

How to cite this article
Patel T, Dangar A, Singh G (November 24, 2026) A Rare Histopathological Finding of an Intradermal Nevus Coexisting With Lichen Planus in an Indian Woman. Cureus 18(11): e120191. DOI 10.7759/cureus.120191

Parameter	Value	Reference range	Unit
Red blood cells	4.59	4.1-5.1	M/µL
Packed cell volume	39.2	36.0-48.0	%
Hemoglobin	11.7	12.0-15.5	g/dL
Mean corpuscular volume	85.4	80.0-100.0	fL
Mean corpuscular hemoglobin	25.5	27.0-33.0	Pg
Mean corpuscular hemoglobin concentration	29.8	32.0-36.0	g/dL
Red cell distribution width	13.1	11.5-14.5	%
White blood cells	7,710	4,000-11,000	/µL
Neutrophils	69	40-70	%
Lymphocytes	21	20-40	%
Monocytes	8	2-10	%
Eosinophils	2	1-6	%
Basophils	0	0-1	%
Absolute neutrophil count	5.35	2.00-7.00	× 10^3/µL
Absolute lymphocyte count	1.52	1.00-3.00	× 10^3/µL
Absolute monocyte count	0.63	0.20-1.00	× 10^3/µL
Absolute eosinophil count	0.17	0.02-0.50	× 10^3/µL
Absolute basophil count	0.04	0.00-0.10	× 10^3/µL
Platelet count	335,000	150,000-450,000	/µL
Urine pH	5	4.5-8.0	Index
Urine specific gravity	1.02	1.005-1.030	Index
Urine epithelial cells	1-2	0-5	/hpf
Urine pus cells	1-2	0-5	/hpf
Random plasma glucose	100	<140	mg/dL
Serum creatinine	0.8	0.6-1.1	mg/dL
Serum glutamic pyruvic transaminase	27	7-35	U/L
Thyroid-stimulating hormone	0.5	0.4-4.0	µIU/mL
Vaginal discharge pH	6	3.8-4.5	Index
Vaginal discharge pus cells	2-3	0-5	/OIF
Gram-positive bacilli	>30	Variable	Count
Nugent score	0	0-3	Score

TABLE 1: Baseline laboratory investigations.

Table *2* outlines the urinalysis, microbiological evaluations, viral and syphilis serological findings, and associated histopathological parameters performed to exclude underlying infectious triggers.

Parameter	Result	Expected (reference)
Urine color	Pale yellow	Pale yellow
Urine appearance	Clear	Clear
Urine glucose	Absent	Absent
Urine protein	Absent	Absent
Urine bilirubin	Absent	Absent
Urine ketones	Absent	Absent
Urine red blood cells	Nil	Nil
Urine casts	Absent	Absent
Urine crystals	Absent	Absent
Urine bacteria	Absent	Absent
Urine fungi	Absent	Absent
Viral serology (HIV)	Nonreactive	Nonreactive
Viral serology (hepatitis B surface antigen)	Nonreactive	Nonreactive
Viral serology (hepatitis C virus)	Nonreactive	Nonreactive
Syphilis serology (*Treponema pallidum* hemagglutination assay)	Negative	Negative
Vaginal Trichomonas	Absent	Absent
Vaginal clue cells	Absent	Absent
Vaginal fungal elements	Absent	Absent
Histopathology (right abdomen)	Intradermal melanocytic nevus with lichen planus	Normal skin structure
Histopathology (left buttock)	Lichen planus	Normal skin structure
Histopathology (left back)	Lichen planus	Normal skin structure
Histopathology (right medial sole)	Lichen planus	Normal skin structure

TABLE 2: Urinalysis, serological, and histopathological findings.

Later, the patient was referred to the dermatology department due to worsening cutaneous lesions. On clinical examination, she presented with severe pruritus and multiple violaceous, flat-topped papules and plaques distributed over the limbs, trunk, palms, soles, and abdomen, with lesions persisting for approximately 12 months. Initial differential diagnoses included lichenoid eruption, tinea corporis/cruris, and scabies, which were subsequently refined to lichen planus and prurigo simplex. Punch biopsies were obtained from the right abdomen, left buttock, left back, and right medial sole. Histopathological examination of the right abdominal biopsy showed a well-circumscribed intradermal melanocytic nevus composed of nests of mature melanocytes with a Grenz zone, progressive maturation with depth, and no mitotic activity. Histopathological examination confirmed an intradermal nevus. In contrast, the biopsies from the left buttock, left back, and right medial sole showed characteristic features of lichen planus, including hyperkeratosis, hypergranulosis, sawtooth rete ridges, basal cell vacuolar degeneration, a dense band-like lymphocytic infiltrate at the dermoepidermal junction, and pigment incontinence. Histopathological examination confirmed lichen planus in three biopsy specimens, while the right abdominal biopsy incidentally revealed an intradermal nevus, representing an uncommon coexistence (Figure *1*).

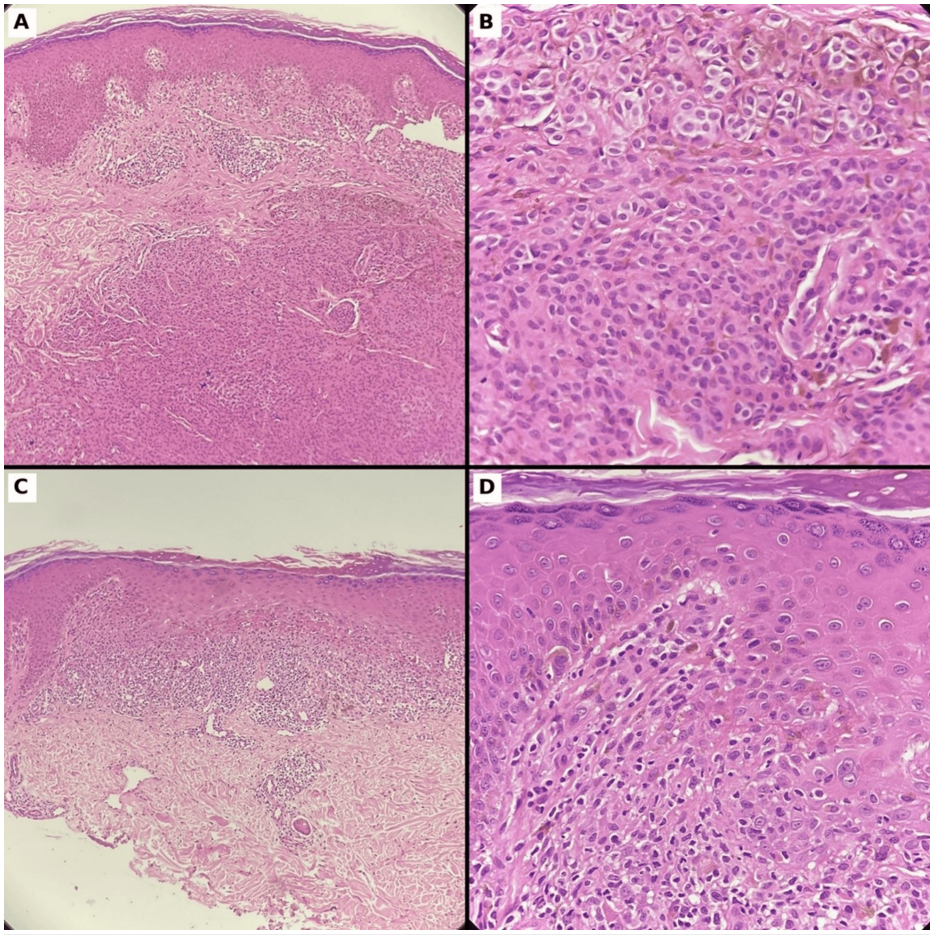


FIGURE 1: (A) Photomicrograph of the skin biopsy stained with H&E showing dermal nests of melanocytes (H&E, 10×). (B) Tumor nests composed of small, uniform cells with scant cytoplasm, regular nuclei, and minimal melanin pigment, consistent with an intradermal nevus (H&E, 40×). (C) The epidermis shows acanthosis, hyperkeratosis, sawtooth elongation of the rete ridges, and a dense band-like lymphocytic infiltrate at the dermoepidermal junction (H&E, 4×). (D) The inflammatory infiltrate obscures the junction, with basal cell squamatization, consistent with lichen planus (H&E, 40×).

As the intradermal nevus was a benign incidental finding, no specific treatment was required. The lichen planus was managed with a topical corticosteroid applied once daily, liquid paraffin and white soft paraffin applied twice daily for one month, oral prednisolone 30 mg once daily for 15 days, hydroxyzine 25 mg once daily for 15 days, vitamin D3 (60,000 IU) once weekly, and a combination of methylcobalamin 750 mcg and pregabalin 75 mg at bedtime for neuropathic pruritus. The patient did not attend the scheduled 15-day follow-up visit; therefore, the treatment response could not be assessed.

Discussion

Intradermal nevi: epidemiology and pathogenesis

Intradermal nevi are elevated, variably pigmented papules ranging from a few millimeters to 1 cm in size that may lighten with age, presenting clinically as flat, dome-shaped, papillomatous, or pedunculated lesions [2]. Their prevalence is lower in Asian populations, who tend to develop nevi more commonly at acral sites compared with the head, neck, and trunk predilection seen in Caucasians [3]. Recognized predisposing factors include Turner syndrome, atopy, childhood leukemia chemotherapy, organ transplantation, chronic UV exposure, and somatic BRAF mutations activating the RAS/MAPK pathway [4,5].

Lichen planus: immunopathology

Lichen planus, in contrast, is an immune-mediated inflammatory disorder characterized by a CD8+ T-cell-mediated immune response targeting basal keratinocytes, producing classic interface dermatitis of the skin, mucosae, or both [6].

Histopathological features

Intradermal nevi show nests and cords of nevomelanocytes confined entirely to the dermis, with no junctional component at the dermoepidermal interface. A hallmark feature is "maturation with depth," in which nevus cells appear larger and more epithelioid in the superficial dermis, becoming progressively smaller, more spindled, and neuroid-like as they descend, often admixed with multinucleated giant cells and associated with adnexal structures [2,3].

Conversely, lichen planus demonstrates compact hyperkeratosis without parakeratosis, a wedge-shaped zone of hypergranulosis, and irregular acanthosis producing a characteristic "sawtooth" rete ridge pattern. A dense, band-like lymphocytic infiltrate hugs the dermoepidermal junction, accompanied by basal cell vacuolar degeneration, apoptotic keratinocytes (Civatte/colloid bodies), and pigment incontinence characterized by dermal melanophages reflecting basal layer damage [6].

Mechanisms of coexistence

Several interrelated immunological and pathogenic mechanisms may explain the spatial localization of lichenoid inflammation over or adjacent to a preexisting melanocytic nevus. Ruocco et al. first defined the term "immunocompromised district" (locus minoris resistentiae) as a localized cutaneous area with altered regional immunity due to previous subclinical trauma, UV radiation, viral reactivation, or architectural modification by dermal nevomelanocytic nests, making the site uniquely susceptible to secondary immune-mediated dermatoses [7]. Second, the isomorphic response (Koebner phenomenon), a classic clinical hallmark of active lichen planus, may be triggered over a preexisting elevated intradermal nevus papule following localized mechanical friction or pruritus-induced excoriation [8]. Third, local microenvironmental immune dysregulation driven by T-helper 1 cytokines, particularly interferon-gamma and tumor necrosis factor-alpha, promotes the recruitment of CXCR3+ CD8+ cytotoxic T lymphocytes to the dermoepidermal junction, extending basal keratinocyte apoptosis across the overlying or perinevoid epidermis [6,8]. While incidental intradermal nevi carry minimal intrinsic malignancy risk [2], recognizing these coexistent inflammatory mechanisms is vital to avoid misinterpreting inflammation-induced architectural distortion, dense lymphocytic infiltration, or pigment incontinence as melanoma-associated regression or severe melanocytic dysplasia [2,9].

Literature comparison

The simultaneous presence of lichenoid inflammation and a preexisting melanocytic nevus is an uncommon cutaneous phenomenon that presents distinct diagnostic challenges. Similar cases were reported by Podo Brunetti et al. and Azeez et al. [9,10]. The literature reviewed mainly concerns the inflammatory induction of junctional melanocytic or pseudomelanocytic nests in the presence of interface dermatoses such as lichen planus pigmentosus. One case of lichen planus associated with an intradermal nevus exemplifies the special case of a "collision" lesion. Disruption of the basement membrane zone in lichenoid dermatitis may induce reorganization of the "epidermo-melanocytic" unit, with de novo formation of junctional pseudonests or true melanocytic nests. Conversely, collision of lichen planus with a preexisting melanocytic nevus brings preexisting dermal melanocytic aggregates into the inflammatory milieu and can lead to pseudomelanoma-like changes. This overlap makes the histopathological differential diagnosis more difficult, an issue that also occurs in lichen sclerosus or postinflammatory states. Both situations, whether involving the assessment of junctional nests due to inflammation or a collision with a nevus, require clinicopathological correlation before they can be accurately differentiated from atypical melanocytic proliferations [9].

Conclusions

This case documents a rare coexistence of an intradermal melanocytic nevus and lichen planus. The incidental discovery of the nevus in one of four punch biopsies underscores the diagnostic value of sampling multiple active lesions in widespread disease. Accurate diagnosis and management require careful clinical examination and directed biopsy at multiple sites. The patient did not return for a follow-up visit after treatment, making it impossible to assess the clinical evolution and response to therapy.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Tarang Patel, Avani Dangar, Gyanendra Singh

Acquisition, analysis, or interpretation of data: Tarang Patel, Avani Dangar, Gyanendra Singh

Drafting of the manuscript: Tarang Patel, Avani Dangar, Gyanendra Singh

Critical review of the manuscript for important intellectual content: Tarang Patel, Avani Dangar, Gyanendra Singh

Supervision: Tarang Patel, Avani Dangar, Gyanendra Singh

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Frischhut N, Zelger B, Andre F, Zelger BG: The spectrum of melanocytic nevi and their clinical implications . J Dtsch Dermatol Ges. 2022, 20:483-504. 10.1111/ddg.14776
2. Wang YK, Gao YJ, Liu J, Zhu QL, Wang JC, Qin J, Jin HZ: A comparative study of melanocytic nevi classification with dermoscopy and high-frequency ultrasound. Skin Res Technol. 2022, 28:265-73. 10.1111/srt.13123
3. Vičić M, Hlača N, Kaštelan M, Brajac I, Sotošek V, Prpić Massari L: Comprehensive insight into lichen planus immunopathogenesis. Int J Mol Sci. 2023, 24:3038. 10.3390/ijms24033038
4. Cho KH, Lee AY, Suh DH, Lee YS, Koh JK: Lobulated intradermal nevus. Report of three cases . J Am Acad Dermatol. 1991, 24:74-7. 10.1016/0190-9622(91)70014-s
5. Krengel S: Nevogenesis-new thoughts regarding a classical problem . Am J Dermatopathol. 2005, 27:456-65. 10.1097/01.dad.0000175532.27368.3f
6. Boch K, Langan EA, Kridin K, Zillikens D, Ludwig RJ, Bieber K: Lichen planus. Front Med (Lausanne). 2021, 8:737813. 10.3389/fmed.2021.737813
7. Ruocco V, Ruocco E, Piccolo V, Brunetti G, Guerrera LP, Wolf R: The immunocompromised district in dermatology: a unifying pathogenic view of the regional immune dysregulation. Clin Dermatol. 2014, 32:569-76. 10.1016/j.clindermatol.2014.04.004
8. Zhang X, Lei L, Jiang L, et al.: Characteristics and pathogenesis of Koebner phenomenon . Exp Dermatol. 2023, 32:310-23. 10.1111/exd.14709
9. Podo Brunetti A, Bigotto GD, Stabile G, Caputo V, Brambilla L, Guida S, Rongioletti F: Lichen planus pigmentosus with true melanocytic nests: a case report with a comprehensive literature review. Dermatopathology (Basel). 2023, 10:281-6. 10.3390/dermatopathology10030036
10. Azeez F, Sobhanakumari K, Zacharia M, Palakkal S, Shaj K: Becker's nevus and lichen planus: a rare coexistence. J Skin Sex Transm Dis. 2022, 4:250-3. 10.25259/JSSTD_11_2022