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CASE REPORT

Coexistence of Multiple Cutaneous Leiomyomas and Psoriasis Vulgaris: A Case Report and Review of Pathogenetic Insights

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Abstract: The co-occurrence of multiple cutaneous leiomyomas (MCL) and psoriasis vulgaris (PV) is exceptionally rare, with no established pathophysiological link between these two conditions. We present a case of a 56-year-old woman with a 10-year history of widespread psoriatic plaques who presented with the concurrent development of multiple, firm, dark red papules and nodules. Histopathological examination confirmed the diagnosis of MCL, revealing interlacing bundles of spindle-shaped smooth muscle cells in the dermis, which were immunoreactive for smooth muscle actin and desmin. A separate biopsy from a psoriatic plaque showed characteristic features of PV. The patient was treated with a combination of thalidomide and tofacitinib, resulting in significant symptomatic and clinical improvement. This unique case highlights a potential, yet unexplored, interplay between smooth muscle proliferation and chronic inflammation, suggesting that shared immunogenetic mechanisms warrant further investigation. Clinicians should be aware of such unusual comorbidities when managing complex dermatological cases.

Keywords: cutaneous leiomyoma, multiple cutaneous leiomyomatosis, psoriasis vulgaris, coexistence, immunomodulatory therapy, pathogenesis

Introduction

Cutaneous leiomyomas are rare benign tumors that arise from smooth muscles, including arrector pili, genital, and vascular types. They typically present as firm, painful papules or nodules, often distributed in grouped patterns. Histopathologically, they are characterized by interlacing bundles of spindle-shaped smooth muscle cells.[1] While the pathogenesis of sporadic cutaneous leiomyomas remains incompletely understood, familial cases have been associated with mutations in the fumarate hydratase (FH) gene, linking them to hereditary leiomyomatosis and renal cell cancer (HLRCC) syndrome.[2]

Psoriasis vulgaris, in contrast, is a common immune-mediated chronic inflammatory skin disease. Its pathogenesis involves a complex interplay of genetic predisposition and dysregulated immune responses. In particular, the IL-23/Th17 axis leads to keratinocyte hyperproliferation and the formation of characteristic erythematous, scaly plaques.[3]

The co-occurrence of these two distinct pathophysiological entities-a proliferative smooth muscle tumor and a chronic inflammatory dermatosis-is exceptionally rare. Their pathophysiological mechanisms appear distinct, with no established link reported in the existing literature. The lack of any documented association makes the simultaneous presentation of both conditions in a single patient particularly noteworthy. To the best of our knowledge, this is the first reported case of MCL coexisting with PV.

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This case report describes a patient presenting with both multiple cutaneous leiomyomas and psoriasis vulgaris. We document this unusual clinical overlap, describe the diagnostic features, and discuss the therapeutic challenges in managing both conditions concurrently. This report aims to raise clinical awareness among dermatologists and encourage further research into possible links between these two disorders.

Case Report

A 56-year-old woman presented to our dermatology clinic with a 10-year history of generalized erythematous papules and intense pruritus. The lesions initially appeared on her neck and, over the past three years, had progressively spread across her trunk and extremities. She had previously been treated with topical corticosteroids and calcipotriol ointment for psoriasis with suboptimal control. Notably, for approximately 1 year, she had noticed the appearance of multiple new, firm, and occasionally tender papules and nodules amidst her psoriatic plaques. Her personal medical history was significant for uterine leiomyoma status post hysterectomy and type 2 diabetes mellitus. Notably, her daughter also had a history of uterine leiomyoma.

Physical examination revealed multiple, variably sized, erythematous patches with silvery-white scales on the trunk and extremities, consistent with psoriasis vulgaris. Interspersed among these were scattered, firm, dark red papules and nodules, ranging in size from millet to soybean, with some showing superficial ulceration (Figure 1A–C). The scalp exhibited patchy erythema with scaling (Figure 1D).

Laboratory investigations demonstrated elevated glycated hemoglobin (7.5%). Immunological assessment revealed decreased complement levels (C3: 0.7 g/L, C4: 0.14 g/L). Computed tomography showed mild pulmonary fibrosis, minimal pelvic fluid, and mildly enlarged axillary and inguinal lymph nodes. To definitively establish the diagnosis and rule out Hereditary Leiomyomatosis and Renal Cell Cancer (HLRCC) syndrome, genetic testing for the FHgene was recommended to the patient. After a thorough explanation of the test's significance, the patient declined to proceed due to personal financial constraints.

A histopathological examination of a nodule from the back revealed parallel arrays of spindle-shaped smooth muscle cells with eosinophilic cytoplasm and blunt-ended nuclei in the dermis, without significant atypia or mitosis (Figure 2A and B). Immunohistochemistry was positive for SMA(+) and Desmin(+), with Ki-67 proliferation index approximately 5% (Figure 2C–E). In contrast, a biopsy from a psoriatic plaque at the hairline demonstrated parakeratosis, diminished granular layer, spongiotic changes with elongated rete ridges, and mild perivascular lymphocytic infiltration in the dermis (Figure 3).

Diagnosis and Treatment

Final Diagnosis: 1. Multiple cutaneous leiomyomatosis 2. Psoriasis vulgaris. Based on the clinical and histopathological findings, the patient was diagnosed with multiple cutaneous leiomyomatosis and psoriasis vulgaris. Given the extensive skin involvement, the patient's refusal of surgical excision for numerous lesions, and the challenge of managing two distinct conditions, an off-label combination therapy was attempted. She was started on a combination therapy of thalidomide (50 mg twice daily) and tofacitinib (5 mg once daily), along with supportive treatments. After 12 weeks of treatment, significant improvement in both pruritus and skin lesions was observed. The Psoriasis Area and Severity Index (PASI) score decreased from 15.2 at baseline to 4.5.

Discussion

This case presents a rare co-occurrence of MCL and psoriasis vulgaris. While both conditions are well-documented individually, their simultaneous presentation is exceptionally uncommon, with no established pathophysiological connection. The association is more likely coincidental rather than indicative of a shared pathogenesis; however, this unique clinical presentation warrants a comprehensive discussion.

Consideration of Hereditary Leiomyomatosis and Renal Cell Cancer (HLRCC)

The patient's personal and familial history of uterine leiomyoma raises the crucial consideration of HLRCC syndrome, associated with germline FHgene mutations.[2] Multiple cutaneous leiomyomas can be a sentinel sign of this condition,

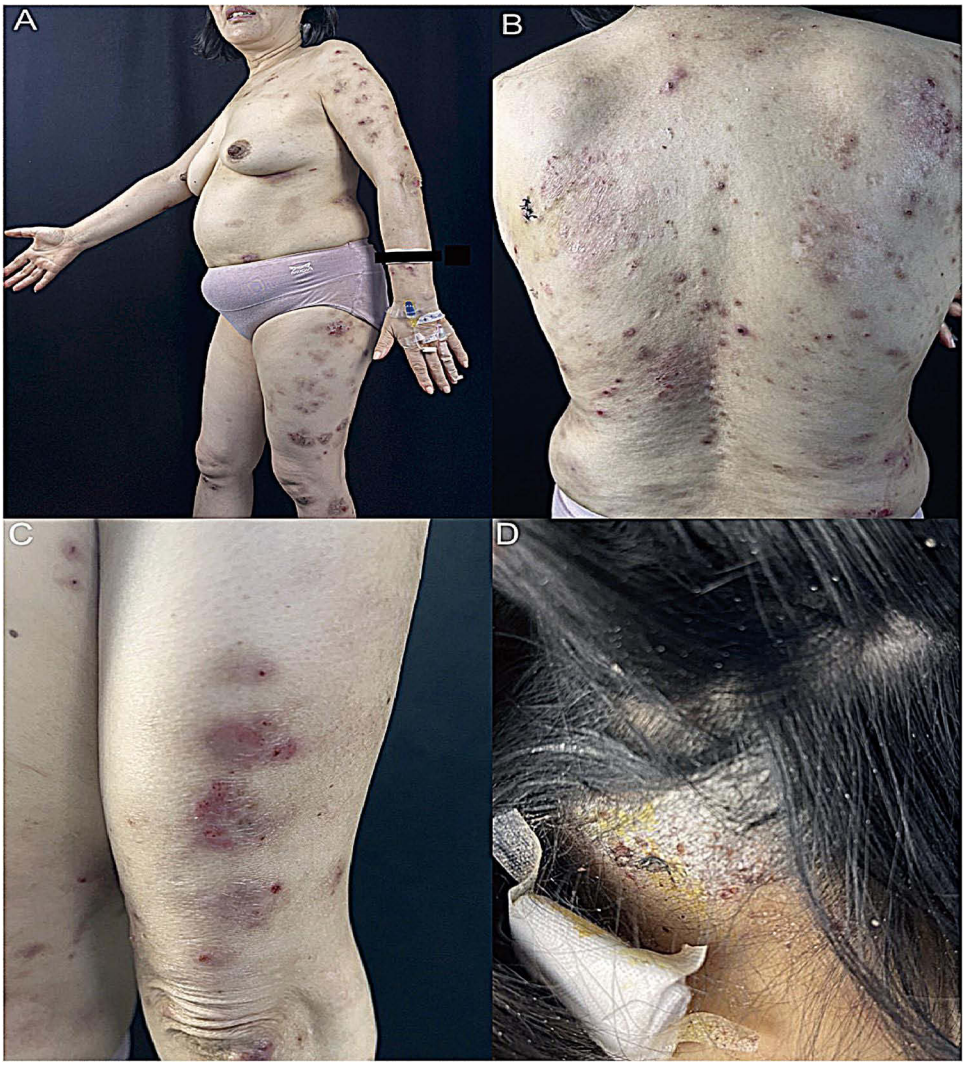

Figure 1 Clinical presentation of the patient. (**A**) Limbs, (**B**) Back, and (**C**) Thighs showing erythematous, scaly plaques of psoriasis vulgaris (diffuse, well-demarcated patches with silvery scale) interspersed with multiple, firm, dark red papules and nodules of cutaneous leiomyomas (smaller, discrete, dome-shaped lesions). (**D**) Scalp demonstrating patchy erythema with scaling.

which carries a significant risk for renal cell carcinoma.[4] In the management of any patient with multiple cutaneous leiomyomas, excluding HLRCC is mandatory. This should include detailed renal imaging (eg, contrast-enhanced CT or MRI) and, ideally, genetic counseling and testing for FHmutations. While our patient's imaging did not show renal masses, the presence of mildly enlarged lymph nodes and pelvic fluid, though non-specific, underscores the need for continued surveillance. A key limitation of this report is the absence of genetic testing to definitively rule out HLRCC.

Potential Shared Pathogenetic Insights

Although the coexistence may be coincidental, exploring potential overlapping pathways is intriguing. Both conditions involve dysregulated proliferation, albeit of different cell types (smooth muscle cells vs. keratinocytes). Several factors could theoretically serve as a nexus:

Growth Factors and Cytokines

Insulin-like Growth Factor (IGF) and Vascular Endothelial Growth Factor (VEGF) are implicated in both smooth muscle tumor growth and psoriatic angiogenesis and inflammation. Similarly, Transforming Growth Factor-β (TGF-β) plays

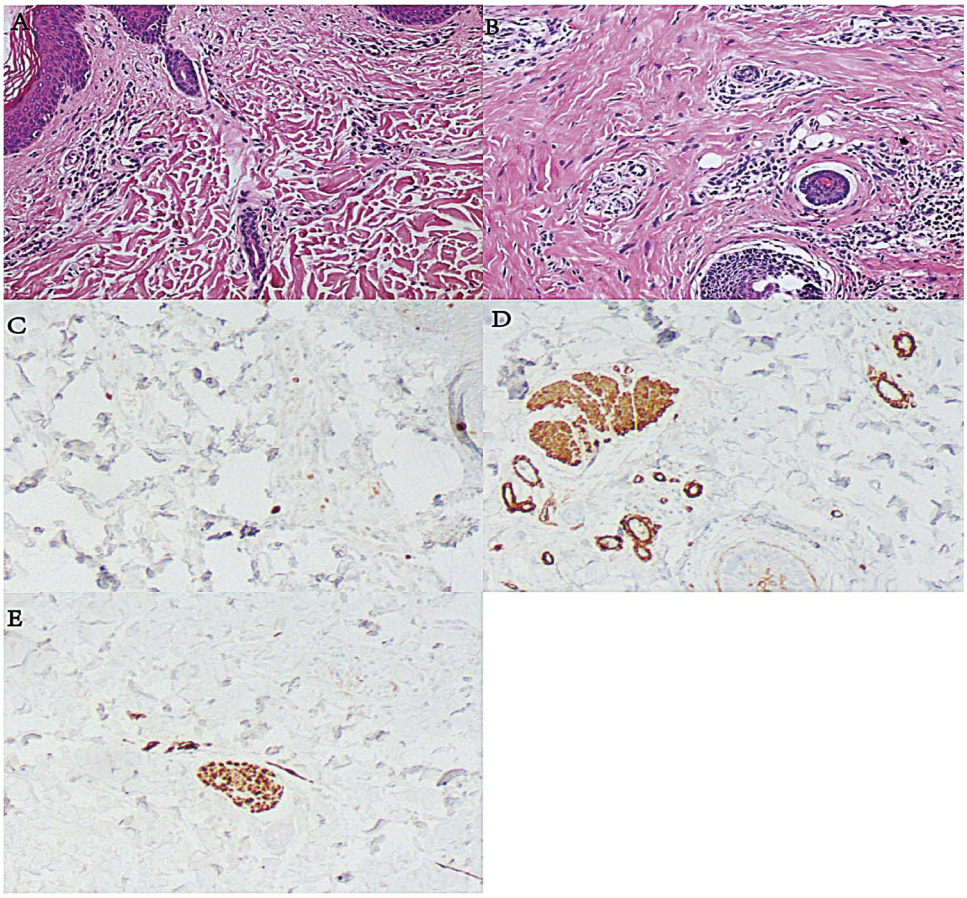

Figure 2 Histopathological and immunohistochemical features of a cutaneous leiomyoma. (**A** and **B**) Hematoxylin and eosin (H&E) staining at different magnifications reveals interlacing bundles of spindle-shaped smooth muscle cells with eosinophilic cytoplasm and blunt-ended nuclei in the dermis. (**C**) Immunohistochemical staining shows strong positivity for smooth muscle actin (SMA). (**D**) Immunohistochemical staining shows strong positivity for Desmin. (**E**) The tumor cells exhibit a Ki-67 proliferation index of approximately 5%.

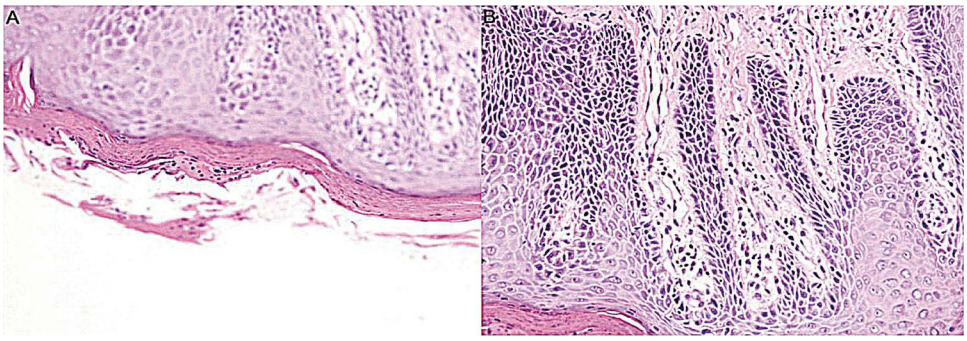

Figure 3 Histopathological examination of a psoriatic plaque (H&E staining). (**A**) Low-magnification view showing the overall architecture of the psoriatic plaque, including epidermal hyperplasia and the presence of a Munro's microabscess. (**B**) High-magnification view highlighting characteristic features, including confluent parakeratosis, loss of the granular layer, marked elongation of the rete ridges, and spongiosis with an associated perivascular lymphocytic infiltrate in the superficial dermis.

complex roles in fibrosis, immune regulation, and has been studied in both leiomyoma pathogenesis and the dermal changes of psoriasis.[5]

Vitamin D Metabolism

Vitamin D deficiency and polymorphisms in the vitamin D receptor have been associated with both an increased risk of uterine leiomyomas and the severity of psoriasis, suggesting a potential immunomodulatory and anti-proliferative link.[6,7]

Immunogenetic Background

The immunologic finding of reduced complement levels in our patient may point to underlying immune dysregulation. It is plausible that a common, yet unidentified, genetic predisposition could influence both immune homeostasis and mesenchymal cell proliferation control.[8]

Management and Unresolved Questions

The management of this patient was challenging. The observed improvement with the combination of thalidomide (with anti-angiogenic and immunomodulatory properties)[9,10] and tofacitinib (a JAK inhibitor)[11,12] must be interpreted with caution. This was an empirical, off-label regimen chosen based on the drugs' separate mechanisms targeting inflammation and proliferation. It does not constitute a standard recommendation, and its efficacy and safety in this specific context remain unknown. Surgical excision remains the definitive treatment for symptomatic leiomyomas, and standard systemic therapies (eg, methotrexate, biologics) are first-line for moderate-to-severe psoriasis. A multidisciplinary approach involving dermatology, urology/oncology, and genetics is essential for optimal long-term care, especially if HLRCC is a concern.

Evaluation of Incidental Findings and Study Limitations

The incidental lymphadenopathy and pelvic fluid observed on computed tomography (CT) likely represent reactive or inflammatory changes, possibly associated with the patient's chronic inflammatory dermatosis (psoriasis) or other undetermined etiologies. Regular clinical and radiological follow-up is warranted to monitor the evolution of these findings.

Several limitations should be acknowledged in this case report. First, definitive exclusion of Hereditary Leiomyomatosis and Renal Cell Cancer (HLRCC) syndrome—a key differential diagnosis in a patient with multiple cutaneous leiomyomas—was not possible, as the patient declined FHgene testing due to financial constraints. Furthermore, the diagnostic evaluation did not include a broader laboratory assessment, such as autoimmune serology (eg, antinuclear antibodies), evaluation for components of metabolic syndrome, or measurement of serum vitamin D levels. Finally, the conclusions are drawn from a single case with limited follow-up duration, which restricts the generalizability of the observations and the strength of any inferred pathogenetic associations.

Conclusion

This report underscores the importance of considering uncommon disease associations in dermatological practice. It demonstrates the potential utility of combined immunomodulatory therapy for complex cutaneous conditions. Further observation and reporting of similar cases may advance our understanding of potential pathophysiological connections between proliferative smooth muscle disorders and inflammatory skin diseases.

Ethics Approval

Ethical approval was exempted for this case report by the Institutional Review Board of Hospital of Chengdu University of Traditional Chinese Medicine. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Consent for Publication

Informed consent was obtained for the publication of the case. This article adheres to the applicable CAse REport (CARE) guidelines.

Informed Consent for Publication

The patient had signed informed consent and provided informed consent for the publication of the case details and any accompanying images.

Acknowledgments

Man Yu and Xingying Chen contributed equally to this work and are co-first authors.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors report no conflicts of interest in this work.

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