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CASE REPORT

Scrotal lichen simplex chronicus triggered by an insect bite: a case report with neuroimmune pathophysiological insights

Líquen simples crónico escrotal desencadeado por picada de inseto: relato de caso e perspetivas fisiopatológicas neuroimunológicas

Aimane Zaim[1], Hanane Baybay[1], Sofia El Haitami[1], Laila Chbani[2], and Fatima Zahra Mernissi[1]

[1]Department of Dermatology; [2]Department of Pathology. Hassan II University Hospital, Sidi Mohamed Ben Abdellah University, Fez, Morocco

Abstract

Lichen simplex chronicus (LSC) is a chronic pruritic disorder characterized by localized lichenification resulting from a persistent itch-scratch cycle. We report a 73-year-old male farmer with type 2 diabetes mellitus who presented with a 30-year history of severe scrotal pruritus. Symptoms began approximately 2 months after a documented insect bite in the scrotal region. Examination revealed a linear hypertrophic plaque with central ulceration on lichenified, hyperpigmented skin. Dermoscopy showed polymorphic vessels, ulceration, red globules, and structureless white areas. Histopathology confirmed LSC. Treatment with high-potency topical corticosteroids, local anesthetic cream, and emollients resulted in partial improvement. Insect bites may initiate persistent itch through acute inflammation and activation of cutaneous pruriceptors, potentially leading to neuroimmune dysregulation in predisposed individuals. This case highlights a possible neuroimmune link between an insect bite and chronic scrotal pruritus.

Keywords: Lichen simplex chronicus. Neurodermatitis. Scrotal pruritus. Insect bite. Neuroimmune dysregulation. Itch-scratch cycle.

Resumo

O líquen simples crónico é uma doença pruriginosa caracterizada por liquenificação localizada resultante de um ciclo persistente de prurido-escoriação. Relatamos um homem de 73 anos, agricultor, com diabetes mellitus tipo 2, com prurido escrotal intenso de 30 anos. Os sintomas começaram dois meses após uma picada de inseto na região escrotal. Ao exame, observava-se uma placa hipertrófica linear com ulceração central sobre pele liquenificada e hiperpigmentada. A dermatoscopia revelou vasos polimórficos, ulceração, glóbulos vermelhos e áreas brancas sem estrutura. O exame histopatológico confirmou o diagnóstico. O tratamento com corticosteroides tópicos de elevada potência, anestésico local e emolientes proporcionou melhoria parcial. As picadas de insetos podem desencadear prurido persistente através da inflamação e ativação dos pruricetores cutâneos, contribuindo para desregulação neuroimunitária. Este caso sugere uma ligação neuroimunitária entre picada de inseto e prurido escrotal crónico.

Palavras-chave: Líquen simples crónico. Neurodermatite. Prurido escrotal. Picada de inseto. Desregulação neuroimunitária. Ciclo prurido-escoriação.

***Correspondence:**
Aimane Zaim
E-mail: aimane.zaim@usmba.ac.ma

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Introduction

Lichen simplex chronicus (LSC), also known as circumscribed neurodermatitis, is a common yet underrecognized chronic, relapsing pruritic dermatosis characterized by localized lichenification resulting from irresistible and persistent scratching or rubbing. The condition is driven by a self-perpetuating itch-scratch cycle and is increasingly recognized as a neuroimmune and psychodermatological disorder, involving complex interactions between cutaneous nerves, immune mediators, and psychological factors.[1]

Although LSC may arise idiopathically, it is frequently secondary to chronic pruritic stimuli, including inflammatory dermatoses, infections, systemic diseases, or environmental triggers.[2] Any initial insult capable of inducing intense or persistent pruritus may initiate the itch-scratch cycle that ultimately leads to LSC. In this context, insect bites represent a plausible but underreported triggering factor, particularly in anatomically sensitive and highly innervated areas such as the scrotum.

We report a case of long-standing scrotal LSC in which the onset of symptoms was temporally related to a scrotal insect bite, and we discuss the potential pathophysiological mechanisms linking insect-induced cutaneous inflammation to chronic neuroimmune dysregulation.

Case report

A 73-year-old male farmer living in a rural environment, with a history of type 2 diabetes mellitus treated with oral antidiabetic drugs, presented with a 30-year history of intense scrotal pruritus. The itching was permanent, severe, and exacerbated at night, significantly impairing sleep and quality of life. According to the patient's history, pruritus first appeared approximately 2 months after a documented insect bite to the scrotal region. No other triggering factors were identified. The condition resisted multiple courses of topical corticosteroids and antifungal treatments over the years. Clinical examination revealed a linear hypertrophic plaque measuring approximately 5 cm in length, with central ulceration and an infiltrated base. The lesion was surrounded by a mildly lichenified hyperpigmented scrotum with a few lichenoid papules and accentuation of normal skin markings (Fig. 1). Dermoscopy demonstrated diffuse polymorphic vessels, including dotted, glomerular, and irregular linear vessels, as well as ulcerations, red globules, and structureless white areas (Fig. 2). A skin biopsy

Figure 1. Clinical image showing a linear hypertrophic plaque with central ulceration, surrounded by mildly lichenified hyperpigmented scrotal skin with a few lichenoid papules and accentuation of normal skin markings.

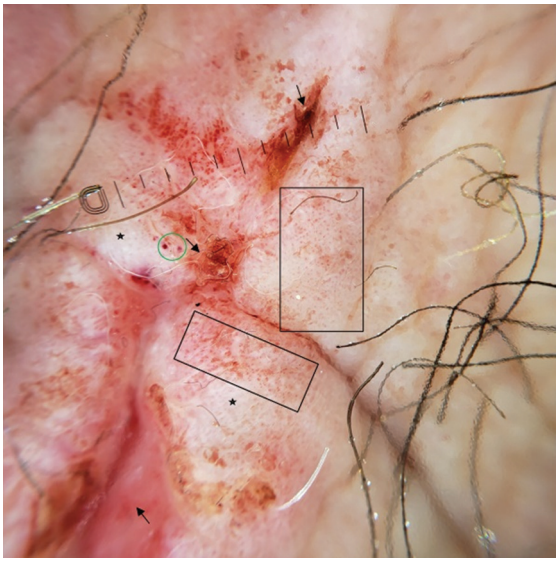

Figure 2. Dermoscopic image (DermLite™ DL5, contact polarized mode, ×10) showing diffuse polymorphic vascularization (black squares), including dotted, glomerular, and irregular linear vessels, along with ulcerations (black arrows), red globules (green circle), and structureless white areas (black stars).

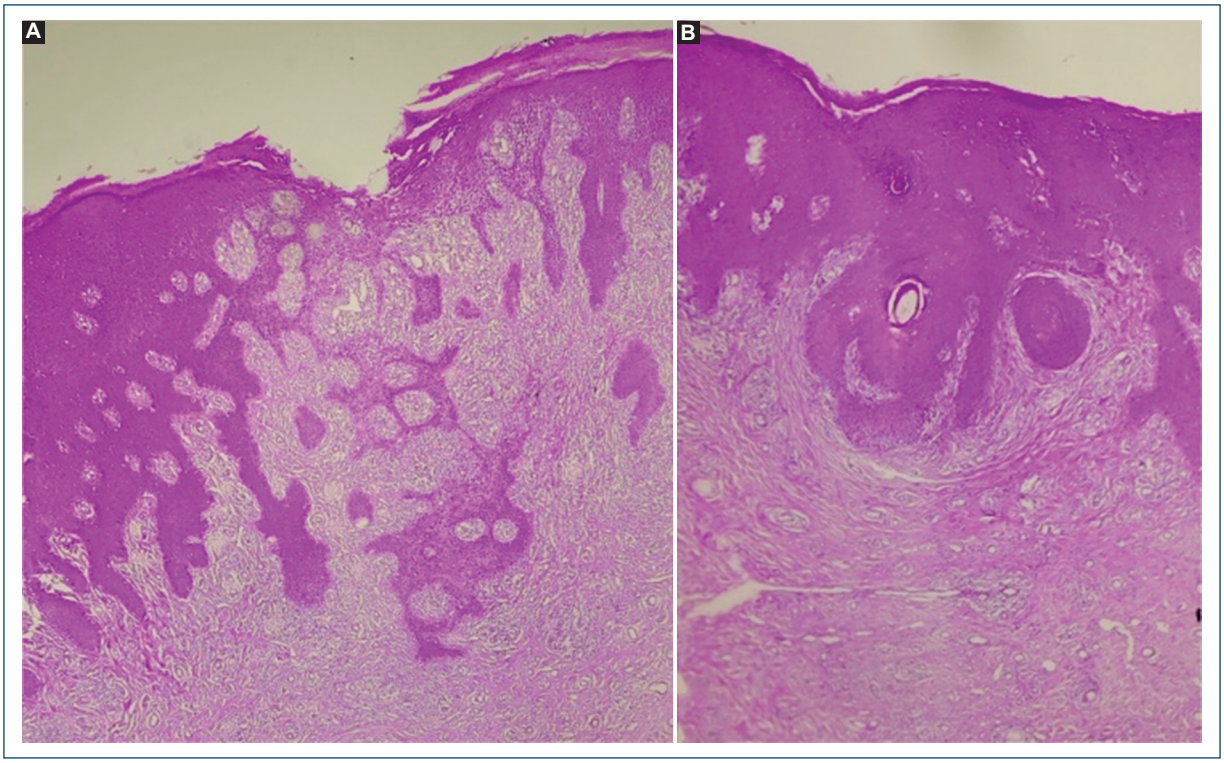

Figure 3. Histopathological examination showing marked acanthosis, papillomatosis, hypergranulosis, and compact orthokeratotic hyperkeratosis, with congested dermal capillaries and a mild interstitial and perivascular lymphocytic infiltrate in the papillary dermis. **A:** ×40 and **B:** ×100 magnification.

and histopathological examination revealed a markedly acanthotic and papillomatous epidermis with hypergranulosis and orthokeratotic hyperkeratosis. The papillary dermis showed an interstitial and perivascular inflammatory infiltrate composed predominantly of lymphocytes and eosinophils. Numerous congested dermal capillaries were observed, without red blood cell extravasation or hemosiderin deposition (Fig. 3). These findings were consistent with the diagnosis of LSC. The patient was treated with a compounded topical preparation combining a super-high-potency corticosteroid, a local anesthetic cream containing lidocaine 2.5% and prilocaine 2.5%, and a reparative emollient balm based on paraffinum liquidum, panthenol, glycerin, and bisabolol. A moderate improvement with a marked reduction in pruritus was observed. The patient was subsequently lost to follow-up.

Discussion

LSC is one of the most common localized chronic itch disorders.[3] It may occur as a primary condition, often associated with psychological stressors or environmental factors such as sweating, chemical irritants, or yeast colonization.[1] Alternatively, LSC may be secondary to other chronic pruritic dermatoses (e.g., atopic dermatitis, eczema, and psoriasis), systemic diseases (e.g., diabetes mellitus and cholestatic liver diseases), or neuropathic conditions.[2] LSC is more prevalent in women and typically affects individuals between 30 and 50 years of age.[4]

LSC is now recognized as a neuroimmune cutaneous disorder driven by a self-perpetuating itch-scratch cycle. An initial pruritic stimulus leads to repeated scratching, resulting in skin barrier disruption and epithelial damage, which in turn triggers the release of cytokines, proteases, and antimicrobial peptides that activate immune cells.[5] Itch in LSC is mediated by activation of protease-activated receptor 2 and by type 2 inflammatory cytokines, particularly interleukin (IL)-4, IL-13, and IL-31, the latter playing a central role in chronic pruritus.[2] These mediators further promote chronic inflammation and stimulate itch-sensory neurons and ion channels, leading to both peripheral and central neural sensitization.[3] Consequently, pruritus in LSC is predominantly non-histaminergic and is sustained by

complex interactions between keratinocytes, immune cells, and cutaneous sensory nerve fibers.

In this context, an insect bite represents a biologically plausible triggering event in our patient. Insect bites induce an acute localized inflammatory response characterized by the release of histamine, proteases, prostaglandins, and proinflammatory cytokines, which can activate cutaneous pruriceptors and initiate scratching behavior. In susceptible individuals, particularly elderly patients or those with comorbidities such as diabetes mellitus, this acute inflammatory stimulus may lead to sustained peripheral neural sensitization, lowering the itch threshold and predisposing to chronic pruritus. Persistent neuroimmune activation following the initial insult may therefore contribute to the initiation and maintenance of LSC, even after resolution of the primary inflammatory lesion. Although a direct causal relationship cannot be definitively established, the temporal association between the scrotal insect bite and the onset of chronic pruritus in our patient supports this hypothesis.

The primary symptom of LSC is intense, chronic pruritus, often described as paroxysmal, characterized by sudden urges to scratch followed by refractory relief periods lasting from 1 to 5 h, and with a tendency to worsen at nighttime.[2]

LSC typically localizes to highly innervated and easily accessible areas, including the neck, ankles, scalp, vulva, scrotum, and extensor forearms.[2] Genital regions are associated with a high degree of "scratch pleasurability," which contributes to the persistence and severity of lesions at these sites.[3] Clinically, LSC presents as a well-demarcated, lichenified, thickened plaque, often hyperpigmented and surrounded by lichenoid papules, with accentuation of normal skin markings.[4] Lesions may show variable discoloration, with erythema ranging from pink to dark brown. Over time, plaques may evolve into hypopigmented areas with a darker peripheral border.[3]

A characteristic three-zone architecture may be observed: a central area of flat lichenification, a middle ring of lichenoid papules, and a peripheral zone of mild thickening and hyperpigmentation.[2] Scratching during refractory periods, particularly in the context of psychological stress, may result in clear skin sores, as observed in our patient.[2] Persistent scratching not only exacerbates LSC severity but may also predispose to secondary infection and, in rare cases, malignant transformation, most commonly squamous cell carcinoma.[3]

Anogenital LSC (AGLSC) is slightly more common in males, with the scrotum being the most frequently involved site.[6] Various pruritic conditions, including viral and fungal infections, infestations with scabies or lice, psoriasis, neoplasia, allergic contact dermatitis, and lichen sclerosus, may promote the development of AGLSC.[6] Additional contributing factors include heat, sweating, friction, occlusive clothing, topical irritants, lubricants, and preservatives. Farmers have been reported to be more commonly affected, consistent with our patient's occupational background.[6] In the anogenital region, the thickened stratum corneum appears white, wrinkled, and smoother due to increased moisture absorption. Rajalakshmi et al. described three clinical patterns of AGLSC: lichenified plaques surrounded by multiple papules at the margins, multiple discrete papules overlying the lichenified plaque, or lichenified plaques with excoriations.[6] AGLSC lesions may also present with scaling, crusting, fissuring, maceration, or nodules. Although pruritus in LSC is often perceived as rewarding or pleasurable,[2] involvement of the genital area is frequently associated with significant physical, sexual, and psychological distress, with patients commonly reporting embarrassment, impaired intimacy, and reduced quality of life.

Dermoscopy of LSC has been infrequently reported in the literature. In a series of 43 cases of genital LSC, dermoscopic features included increased skin rugosity mimicking sulci and gyri (93%), white scales (86%), erythematous background (65%), brown dots, and erosions.[7] In our case, additional dermoscopic findings were noted, including polymorphic vessels, ulcerations, red globules, and structureless white areas, contributing to the expanding dermoscopic spectrum of scrotal LSC.

The diagnosis of LSC is primarily clinical. Skin biopsy is reserved for atypical or refractory cases and when malignancy is suspected. Histopathology shows marked epidermal hyperplasia with hyperkeratosis, hypergranulosis, acanthosis with elongated and thick rete ridges, and mild spongiosis. A stratum lucidum may be seen.[1] A dermal perivascular and interstitial inflammatory infiltrate of lymphocytes, histiocytes, eosinophils, and eventually macrophages may be seen, as well as vertically oriented, thickened collagen bundles.[3]

Differential diagnoses include hypertrophic lichen planus, lichen amyloidosis, psoriasis and psoriasiform rashes, contact dermatitis, squamous cell carcinoma, and mycosis fungoides.[1,3] In AGLSC, lichen sclerosus, lichen planus, and genital neoplasia must also be considered.[1]

Management of LSC requires a multimodal approach aimed at restoring the skin barrier, controlling inflammation, and breaking the itch-scratch cycle.[3] High-potency topical corticosteroids remain the first-line treatment, whereas topical calcineurin inhibitors are particularly useful in sensitive areas such as the genital region.[1]

Adjunctive measures include emollients, occlusive dressings, avoidance of triggers, loose cotton clothing, and behavioral interventions. In refractory cases, systemic therapies such as gabapentin, antidepressants, methotrexate, or cyclosporine may be considered. Emerging treatments, including biologics targeting Th2 cytokines such as IL-4, IL-13, and IL-31, Janus kinase inhibitors, narrowband ultraviolet B phototherapy, and procedural approaches, are generally reserved for severe or treatment-resistant disease.[1,3]

Conclusion

This case highlights the potential role of an insect bite as an initiating event in the development of scrotal LSC, likely mediated through sustained neuroimmune activation and peripheral neural sensitization. Early identification of triggering factors and implementation of a comprehensive, individualized therapeutic strategy are essential to interrupt the itch-scratch cycle and improve long-term outcomes.

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Conflicts of interest

None.

Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from all patients, and secured approval from the Ethics Committee. SAGER guidelines have been followed as applicable to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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