

Diagnosis

Lichen simplex chronicus.

Discussion

Lichen simplex chronicus (LSC), a localized form of lichenification, is a chronic, superficial, pruritic inflammation of the skin occurring in well-circumscribed plaques. LSC is caused by habitual repetitive scratching or rubbing of the skin in regions accessible to scratching. Pruritus, which is more pronounced during periods of inactivity, seems to provoke rubbing resulting in the clinical lesions. LSC is also considered as a localized variant of atopic/neurodermatitis. No racial predilection has been noted. The condition has a predilection for females.¹

LSC typically presents as single or multiple, slightly erythematous, scaly, well-demarcated, hyperpigmented, lichenified, rough plaques, on any location that the patient can reach including the nape of the neck, extensor forearms and elbows, vulva or scrotum, upper medial thighs, knees, lower legs, and ankles.² Repeated scratching, which plays a key role in lesion formation, is depicted variably by white scratch marks, erosions, and ulceration. In its most common presentation, a typical LSC plaque may have 3 zones. A 2 to 3cm wide peripheral zone that is barely thickened may have isolated papules. The middle zone has lenticular and hemispheric prurigo papules that may be excoriated. The central zone has the greatest thickening and pigmentary alteration. LSC may become secondarily infected after excoriation.

Pruritus of LSC is usually mild to moderate, but paroxysms may occur that are relieved by rubbing and scratching. Pruritus is usually described as much worse during periods of inactivity, usually at bedtime and during the night. Touch and emotional stress also may provoke pruritus, which is relieved by moderate-to-severe rubbing and scratching. Patients may have a past medical history of a chronic skin condition or acute trauma. Patients with atopic dermatitis may have LSC in areas of former atopic outbreaks. Sites of irritant or allergic contact dermatitis, insect bites, or other past minor skin trauma sometimes demonstrate pruritus and, subsequently, LSC. Lichen simplex chronicus (LSC) of the anogenital area is an eczematous disease characterized by unremitting itching and scratching.³

A relationship likely exists between central and peripheral neural tissue and inflammatory cell products in the perception of itch and ensuing changes in LSC. The possible interplay among primary lesions, psychic factors, and the intensity of pruritus may also influence the extent and severity of LSC.

The exact etiology of LSC remains unknown but atopic dermatitis results in a higher probability of developing LSC.⁴ Other triggering factors for the development of LSC include insect bites, traumatic or postherpetic scars, acne keloidalis nuchae, xerosis, venous insufficiency, and asteatotic eczema.^{5,6} Psychological factors, as mentioned earlier, appear to play a role in the development or exacerbation of LSC. Anxiety has been reported to be more prevalent in patients with LSC. Neurodermatitis is a term formerly used

interchangeably with LSC, suggesting a role of anxiety or obsession as part of the pathological process of developing lesions.⁷ LSC may need to be differentiated from many conditions with similar clinical presentation. These include lichen amyloidosis, atopic dermatitis, allergic and irritant contact dermatitis, cutaneous T cell lymphoma, lichen planus, discoid eczema, plaque psoriasis, seborrheic dermatitis, and stasis dermatitis.⁸ Potassium hydroxide examination and fungal cultures are usually required to exclude tinea cruris or candidiasis particularly in patients with genital LSC. Patch testing may help exclude allergic contact dermatitis as an underlying primary dermatosis. Other closely mimicking conditions are excluded by histopathological examination of biopsy specimens.

Histologic examination demonstrates hyperkeratosis, acanthosis, spongiosis, and patches of parakeratosis in the epidermis. Epidermal thickening of all layers is noted, with elongation of rete ridges and pseudoepitheliomatous hyperplasia. Papillary dermal fibrosis with vertical streaking of collagen bundles is characteristic of LSC.

LSC frequently persists as an itch-scratch cycle, even when environmental triggers are removed and the underlying disease is treated. For this reason, successful therapy requires attention not only to triggering factors, but also to repair of the damaged barrier layer, reduction in inflammation, and breakup of the itch-scratch cycle.³

Topical steroids are the current treatment of choice because they decrease inflammation

and itch while concurrently softening the hyperkeratosis. On larger and more active lesions, a midpotency steroid may be used to treat acute inflammation. Occasionally, occlusion is used to increase potency and enhance delivery of the agent. Occlusion also provides a physical barrier to scratching. Midpotency topical steroids are not recommended for thin skin (e.g. vulva, scrotum, axilla, face). High-potency topical corticosteroids may be used on thicker-skinned areas. Oral anxiolytics and sedatives may be considered in certain patients. According to individual need, treatment can be scheduled throughout the day, at bedtime, or both. Oral antihistamines such as diphenhydramine and hydroxyzine are also effective. Oral doxepin and clonazepam may be considered in appropriate cases.⁹ For infected lesions, a topical or oral antibiotic can be considered. Other topical medications reported to decrease pruritus include doxepin cream, capsaicin cream and topical aspirin.^{9,10,11} In the future, both topical and systemic immunomodulators, such as topical tacrolimus, may be used in directing the changes in cellular activity that induce itching and inflammation.

References

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