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Lichen Striatus

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A 22-month-old girl presented to the pediatric clinic with a 2-month history of a pruritic rash that h chest and had spread down her left arm (**Figures 1 and 2**). She had no known sick contacts and or contact with new detergents, lotions, soaps or other chemical substances. The mother applied cream as needed for itching.

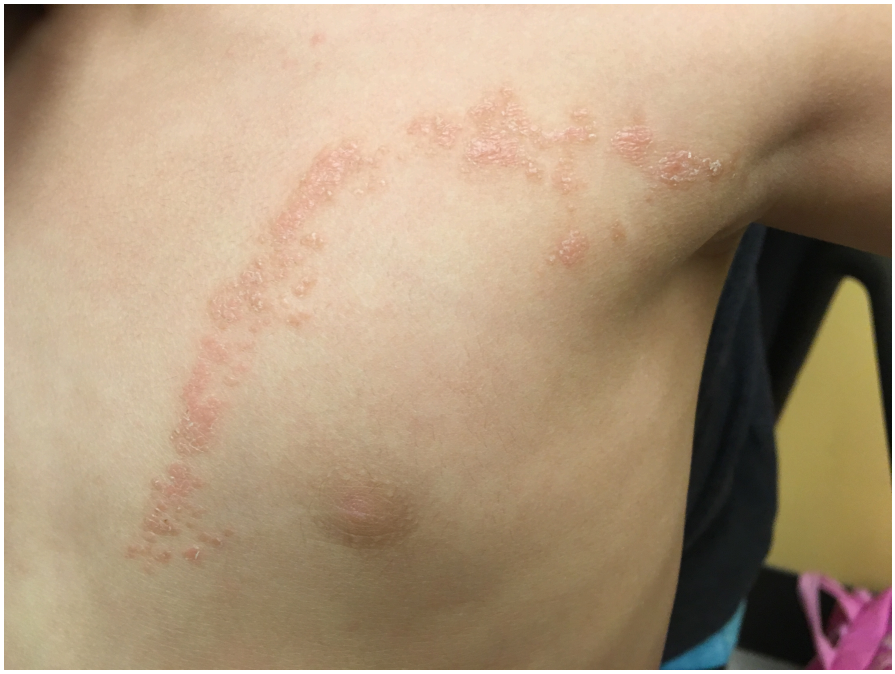

Figure 1. *The initial presentation of the patient's rash was confined to the left anterior chest wall i pattern.*

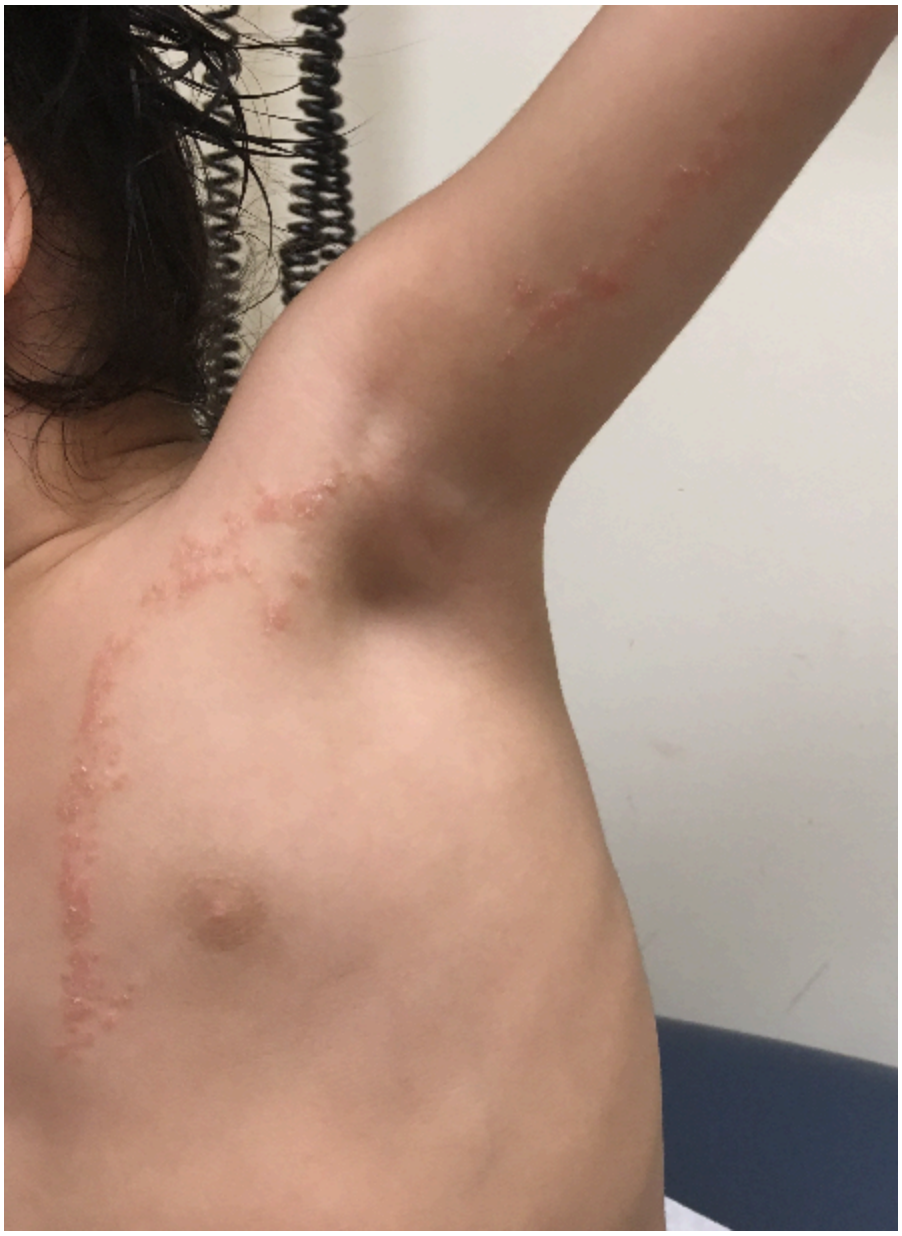

Figure 2. *Two months after the initial presentation, the rash had elongated down the patient's left of her elbow, continuing the U-shaped linear pattern on the chest wall.*

Physical examination revealed linear, skin-colored, flat-topped papules that coalesced into an inv pattern along the upper left trunk and extended down the left arm in a linear pattern. There was n involvement. She was referred to a dermatologist and received a diagnosis of lichen striatus.

Discussion. Lichen striatus is a linear grouping of erythematous papules that occasionally have a component.[1,2] Lesions range in color from pink, red, and tan to skin-colored and may be hypopig skinned individuals.[2,3] Although the lesions are usually asymptomatic, pruritis occurs in 5% to 46 is more common in patients with atopy.[4] The linear bands can be continuous or interrupted, 2 mm and may involve the entire limb.[2,5] The differential diagnosis includes genetic, infectious, and infla (**Table**).

Table. Differential Diagnosis of Linear Dermatological Lesions

Disease	Clinical Findings	Prognosis	Histopathology
Lichen striatus	Unilateral asymptomatic grouping of inflammatory papules coalescing into linear arrangement following lines of Blaschko.[1] Nail involvement confined to medial or lateral edges.[6]	Regresses spontaneously within weeks to years.[4,5] Postinflammatory hypopigmentation may persist for years.[1,4,6]	Dense perivascular lichenoid lymphohistiocytic band surrounding necrotic keratinocytes and variable epidermal changes.[5,6]
Inflammatory linear verrucous epidermal nevus	Unilateral pruritic, erythematous, and hyperkeratotic papules that coalesce into plaques in a linear array.[7]	Does not regress spontaneously, undergoes periods of exacerbation followed by improvement.[4]	Chronic dermal inflammatory infiltrate, psoriasiform epidermal hyperplasia, alternating bands of orthokeratosis and parakeratosis.[7]
Linear lichen planus	Pruritic, polygonal, violaceous, flat-topped papules and plaques.[6] Nail involvement involves multiple nails and the entire nail plate.[8] Oral mucosal lesions often present.[6]	Most cutaneous lesions spontaneously resolve within a few years.[6] Often leaves a postinflammatory hyperpigmentation that takes years to fade.[6]	Wedge-shaped hypergranulosis.[6] Cytoid bodies at the dermoepidermal junction. Direct immunofluorescence positive for multiple IgM immunoglobins.[8]
Linear psoriasis	Well-demarcated, erythematous papules or plaques with overlying micaceous scales.[9]	Patients may have other sequelae of psoriasis that determine the clinical course.[9]	Epidermal hyperplasia, parakeratosis, neutrophils in the stratum corneum, thinned granular cell layer, tortuous dilated dermal papillary capillaries.[10]

Incontinentia pigmenti	Presents in neonatal period with linear papules and vesicles. Progresses to verrucous streaks in weeks to months, then hypopigmentation.[11]	Permanent hyperpigmented whorls along lines of Blaschko. X-linked dominant, lethal in males.[11] Often has other concurrent developmental abnormalities.[12]	Eosinophilic spongiosis and intraepidermal vesicles and apoptotic keratinocytes in the epidermis. Marked melanin incontinence with numerous melanophages in the hyperpigmented stage.[12]
Linear Darier disease	Presents in teenage years with skin or yellow-brown keratotic papules on face, chest, and back.[13]	Generally a chronic condition with frequent exacerbations from several external factors.[13]	Acantholytic dyskeratosis.[13]

The 3 subsets of lichen striatus—typical lichen striatus, lichen striatus albus, and nail lichen striatu by their morphologic variation.[14] Typical lichen striatus is the most common type and occurs in 80 There is no hypopigmentation or nail involvement.[14] Lichen striatus albus is more common in darl patients.[2,15] Lesions in nail lichen striatus occur in isolation or in conjunction with skin lesions.[2] Th may present with onychodystrophy restricted to a single nail and may lead to nail splitting, longitu and/or thinning.[6,16]

The lesions of lichen striatus are usually unilateral and most commonly occur on the limbs, follow head, and neck. They form along the lines of Blaschko, a system of lines representing the pathwa cell migration and proliferation during embryologic development of the skin.[2,17] The lines are V-sh posterior midline overlying the spine, S-shaped or whorled on the lateral and anterior abdomen, ir arcs on the chest, and perpendicular lines on the extremities.[17,18] These unique shapes distinguis Blaschko from neurologic dermatomes.[18]

The distinct distribution of lichen striatus lesions suggests cutaneous mosaicism. Genomic mosai of postzygotic somatic mutations that produce abnormal keratinocyte clones that remain silent un environmental event.[17] The distribution of lines of Blaschko strongly supports this hypothesis, bec patterns are thought to be a result of the folding and stretching of an embryo during the first 2 mo organogenesis.[19] Some suggest that it may be a functional mosaicism due to lyonization of the X Regardless, it is classified as an acquired mosaic condition because it is not congenital and occu break in normal immunologic function. An autoimmune response activates previously silent and ir keratinocyte clones.[3] Triggers may include viral infection, certain vaccinations, medications, and p increased incidence in patients with a history of atopy also strengthens the theory that an autoimr occurs in genetically susceptible individuals.[2,4,5]

The diagnosis is usually based on clinical findings. However, if a biopsy is done, the histologic exa a dense lichenoid lymphohistiocytic band that is perivascular and surrounds the eccrine sweat gla This inflammatory infiltrate is composed of $CD3^+$ and $CD8^+$ cells surrounding necrotic keratinocyt Langerhans cells.[6] Focal spongiosis of the epidermis, dyskeratotic changes that include epiderma and focal parakeratosis, and interface dermatitis are found in the overlying epidermis.[3,6] These fir support an autoimmune process.[6,20]

The onset of lichen striatus lesions can be sudden and progress over several weeks. The lesions spontaneously over 3 months to a year, but some resolve in as soon as 4 weeks while other case several years to resolve, especially cases with nail involvement.[4,5] After resolution, postinflammat hypopigmentation occurs in 25% to 50% of patients, more commonly in dark-skinned patients.[1,4,6] Postinflammatory hyperpigmentation occurs in 3% to 33% of patients.[4] Both can persist for month resolution of the papular lesions.[2,5]

Given the condition's self-limited course, therapy is not usually recommended. Patients can be co condition is benign, and that there are no long-term sequalae.[2] Topical corticosteroids are useful i but they do not hasten resolution.[2,6] Topical calcineurin inhibitors, such as tacrolimus and pimecro be helpful for symptomatic patients.[6] One report demonstrated that oral cyclosporine therapy led t regression of the rash in 4 weeks.[3]

Outcome of the case. We discussed the diagnosis and the benign nature of the disease with the She was told that the rash could take 3 to 12 months to resolve, and that it did not require treatme instructed to apply topical hydrocortisone to relieve the girl's pruritus.

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