Review Article

Linear psoriasis and Koebner phenomenon: A review

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Linear psoriasis (LP) is an unusual form of psoriasis with an unidentified prevalence and is characterized by psoriatic papules and plaques in a Blaschko linear distribution. Based on clinical features, this disorder is divided into two types: isolated LP and superimposed LP. Previous studies have suggested that LP presenting linear appearances is closely related to the Kobner phenomenon (KP) caused by external provocation, including trauma, skin incision, drugs, infections, and striae distensae. Pathogenesis of LP is mainly attributed to the concept of genetic mosaicism but not completely illustrated until now. In this review, we summarize its epidemiological characteristics, clinicopathological features, diagnosis/differential diagnosis, corresponding therapies, focus on the possible pathogenesis of LP, and explore the relationship between LP and KP.

Key words: Blaschko lines, inflammatory linear verrucous epidermal nevus, Koebner phenomenon, linear psoriasis, nevus, psoriasis

How to cite this article: Zheng L, Han X, Zheng H, Zhang Y. Linear psoriasis and Koebner phenomenon: A review. J Res Med Sci 2026;31:6.

INTRODUCTION

Psoriasis is a chronic, recurrent, inflammatory, and immune-mediated skin disease characterized by well-demarcated, scaly erythematous papules and plaques with a positive Auspitz sign. Under certain specific conditions, following a scratching or trauma to the healthy skin areas, the uninvolved skin of psoriatic patients easily develops new psoriatic lesions.[1-12] This phenomenon was named with the Koebner phenomenon (KP), first described in 1877 by the German dermatologist Heinrich Koebner (1838–1904), in the light of the secondary lesions of KP with the same clinicopathological features as the primary lesions.[1,13]

Clinically, the distribution patterns of the lesions of KP are diverse, such as strip/band-shaped, clustered, regular or irregular geometric figures, linear or other styles, closely associated with primary stimulation or trauma.[1] It has been usually considered that KP would occur under individual unstable or intense clinical conditions but not present during the remission of the disorder. Previous studies indicated that KP is not related to the disease's activity or severity;[2] however, the opposite viewpoint holds that KP could serve as a reference indicator for evaluating the disease stage.[14]

And KP presenting with psoriatic lesions along Blaschko lines is rare, termed with Blaschko linear psoriasis (LP) in 1951 by Leslie[15] So far, more and more accumulative data indicate that the presentation of new LP lesions may be caused by a number of trigger factors, including trauma, skin incision, medications, infections, and striae distensae, etc., suggesting the key role of KP [Figure 1].[1,16-20]

Pathogenesis of LP is generally explained by the concept of genetic mosaicism, added with the predisposing

Access this article online

Quick Response Code:

Website: https://journals.lww.com/jrms

DOI: 10.4103/jrms.jrms_364_25

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Submitted: 12-Apr-2025; **Revised:** 06-Nov-2025; **Accepted:** 03-Feb-2026; **Published:** 26-Feb-2026

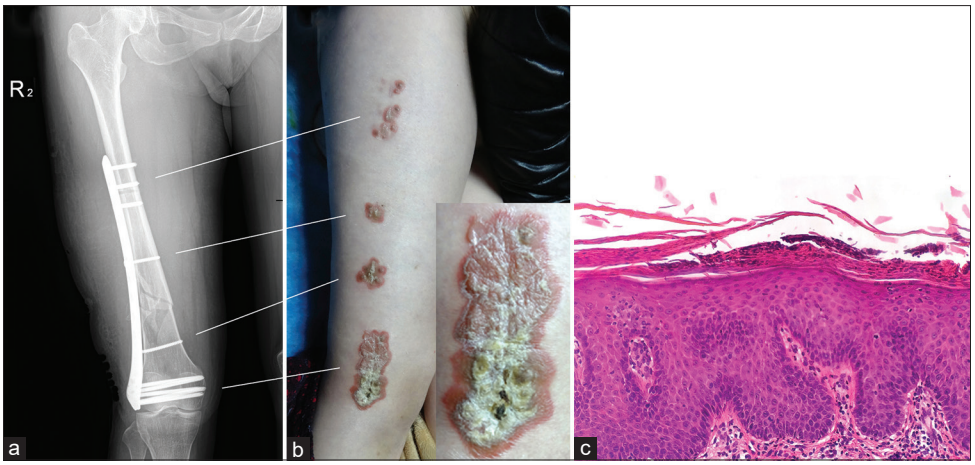

Figure 1: A 45-year-old female presented with isolated linear psoriasis for 2-week duration after open reduction and plate fixation in the right femoral shaft fracture, (a) Several well-demarcated, erythematous papules and plaques with silver scales grew along Blaschko line, (b) The anatomic sites of lesions were almost in accordance with the surgery incision, (c) Classical plaque lesions showed parakeratosis with Munro microabscess. There is marked dilatation and tortuosity of the capillaries within the dermal papillae

factors aforementioned, which still remains fully undefined. This article outlines its epidemiological characteristics, clinicopathological manifestations, diagnosis/differential diagnosis, and treatment measures, and places emphasis on the possible pathogenesis of LP.

Definition of disease

LP is a rare and underdiagnosed subtype of psoriasis. It is characterized by the linear distribution of psoriatic skin lesions along lines of Blaschko. Mainly depending on the differences in clinical manifestations, it generally can be divided into isolated LP (Type I) and superimposed LP (Type II).[21,22] Isolated LP is the only manifestation of psoriasis without other lesions of psoriasis vulgaris (PV).[23-25] It can be superimposed in less severe disseminated classical lesions of PV and revealed when classical lesions subside with antipsoriatic treatment, which is named by superimposed LP.[20,26-30] The term ''superimposed LP'' was introduced in 2007 to highlight the distinction between polygenic and monogenic susceptibility backgrounds,[30] which can help to elucidate the molecular basis of psoriasis.

In previous reports, LP was once documented with "isolated linear psoriasis" or "superimposed linear psoriasis" or "Blaschkoid psoriasis"[31] or "naevoid Blaschkoid psoriasis"[32-34] or "linear naevoid psoriasis."[35,36] Sometimes, it is confused with "naevoid psoriasis"[37] or "unilateral psoriasis"[38,39] in individual literature. Atherton *et al*. first described that naevoid psoriasis is found with erythematous, scaly plaques along the Blaschko lines, nearly similar to LP.[37]

However, doubts have been proposed earlier as regards the existence of naevoid psoriasis as a distinct disease.[37] Bolognia *et al*. hold the opinion that LP is present in a linear style, while naevoid psoriasis shows a band-like configuration which is composed of several or more Blaschko lines approximately arranged in parallel or merged together.[40] Skin lesions may eventually be confined to specific zones of any half of the body, namely, unilateral psoriasis. This is consistent with the later reports by Ginarte *et al*. and Rawal.[38,39]

In our opinion, "linear" or "unilateral" is mainly used to describe the arrangement or distribution of skin lesions, while "naevoid" tends to depict the morphology of the rash. Whether it is "linear naevoid" or "naevoid Blaschkoid," actually these medical terminologies are not contradictory due to the cognitive difference in naming the same disease before and after. Hence, both diseases with similar clinicopathological features actually should be referred to as LP, which is reported in the context of this article.

Epidemiological characteristics

Psoriasis is a common chronic inflammatory skin disorder that affects only 2%–4% of the general population.[41] Hence, there has been no unified method to evaluate the prevalence of KP among psoriasis due to the lack of accurate data. Previous studies from multiple large sample investigations showed that the incidence of KP in psoriasis varies widely from 11% to 75%.[2,42] However, it does not seem that these data were convincing due to discrepancies in research methods, such as cross-sectional study and experimental-inducing method *in vivo*.

Recently, Chen *et al*. summarized that about 33% of LP patients report exogenous triggering or exacerbating factors, including antipsychotic drug (lithium),[18] biological agents (pembrolizumab[19] and Secukinumab[43,44]), climate changes, striae, upper respiratory tract infection, etc. [Figure 2].[16,17,20,45] LP is an extremely rare disease, as one lesion form of the various KP. It is very difficult to

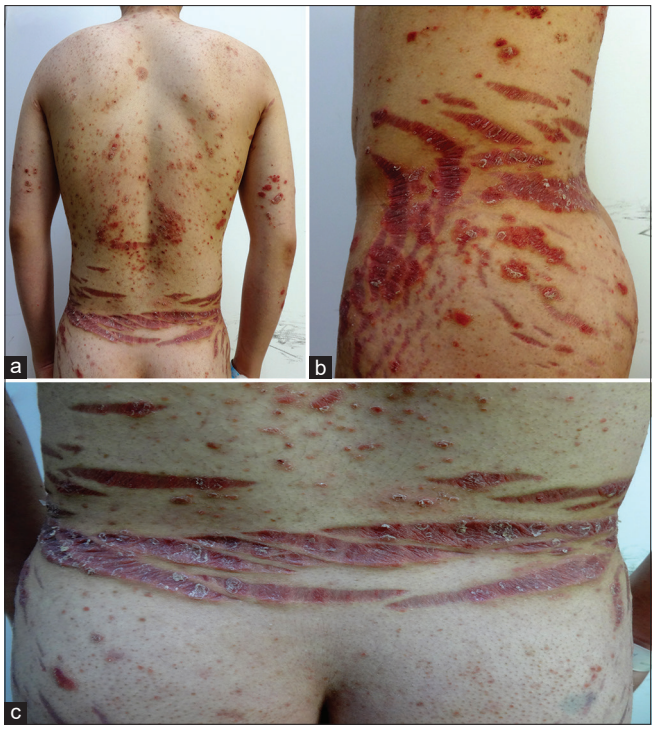

Figure 2: A 19-year-old male developed psoriatic lesions precisely over the *striae distensae* after weight loss, (a-c) Multiple linear shape, well-demarcated erythematous, scaly papules and plaques, located on the abdomen, lumbar area, and thighs, measuring up to 3 cm in width and 20 cm in length, along *striae distensae distribution*

estimate the prevalence of LP. A small sample of research revealed that male patients with LP seem to have a higher proportion (83.3%), the average age of onset is 8.3 years, and only 16.6% of the cases reporting a positive family history of psoriasis.[25] However, statistical data may be overestimated because of too few clinical cases. Another study indicated that in the general population, two-thirds of LP cases start in childhood compared with only one-third of classical psoriasis cases.[20] Moreover, the mean age at diagnosis of LP is 20 years, and LP tends to appear earlier in type I than in type II.[20]

Moreover, Chen *et al*. analyzed 74 LP patients and found similar results that there is also a predominance for male sex (62%), especially for type II patients.[45] The overall age of onset is relatively young, which is markedly different from classical psoriasis. Moreover, no statistical difference was found between type I and II.[45] There may be three possible reasons accounting for this: (a) Most of data derive from single case or series-case reports and lack of cross-sectional study; (b) LP disease is rare or underestimated, and it is easy to escape diagnosis or be misdiagnosed; (c) LP tends to spontaneously resolve, and sometimes clinical data collection can be very difficult, which results in report bias.

Skin lesions are mainly distributed unilaterally, and there is no difference between the two sides of the body.[20] The involved areas mostly include the trunk and limbs distributed along Blaschko lines. Approximately 70% of LP patients show no scalp or joint involvement. More than 75% of cases of LP involve one or two linear lesions, and sometimes the nails on the limbs can also be affected.[20,45]

Koebner phenomenon tends to be suddenly present in some specific stage of psoriasis. It usually appears within about 1–2 weeks but can also occur, ranging from 3 days to 2 years.[46] This wide variation reflects the degree of sensitivity for the development of the Koebner response, which may be a distinctive characteristic of the patients.[2] However, the incubation period from skin injury to the onset of KP has not been determined.

Possible pathogenesis

Researchers have attempted to probe the pathogenesis of inducing the development of new psoriatic lesions as Koebernization. It is generally accepted that KP linking with LP is triggered by external factors based on genetic mosaicism background. Besides, multiple past studies demonstrated that it is not only related to disruption to the epidermis but also with deep tissue injury, which involves multiple molecules and signal pathways. There are several aspects participating in the pathogenesis of KP, such as mechanical scratch/stretch inducing inflammatory response, keratinocyte proliferation and differentiation, angiogenesis, and genetic susceptibility or mosaicism.[14]

First, several important molecules from epidermis injury, initiating inflammation and immunological reactions, are tightly associated with KP. *In vitro*, mechanical scratching on the keratinocytes induces release of cysteine-cysteine motif chemokine ligand 20 (CCL20) and C-X-C motif chemokine ligand 8 via the EGFR-ERK/JNK pathway, which results in accumulation of Th17 cells and neutrophils in the lesions.[47] Furthermore, the increasing level of interleukin-17 (IL-17) strongly promotes keratinocyte proliferation and inhibits differentiation, which may enhance Koebnerization in psoriasis patients. Similarly, mechanical stretch also aggravates psoriasis by stimulating keratinocyte proliferation and inflammatory factors production like IL-1a, IL-6, IL-23, and CCL20 *in vitro*.[48] This trauma induces extracellular adenosine triphosphate (ATP) release from keratinocytes, resulting in subsequent production of Th17-polarizing cytokines, such as pro-IL-1 β and IL-6, and triggers KP, which initiates and perpetuates psoriatic lesion from healthy skin.[49]

Additionally, the release of double-stranded RNAs from the necrotic keratinocytes by skin injury, which causes IL-36 γ production through TLR3 signaling. This process can be synergistically enhanced by IL-17A derived from the mast cells, which subsequently attracts neutrophils into the

epidermis.[50] Meanwhile, antimicrobial peptides like LL37 and DNA derived from stressed keratinocytes consist of LL37-DNA complexes, activating dermal pDCs through TLR7/9 signaling to produce IFNα that promote myeloid dendritic cells (DC) maturation. Activated mDCs provoke the adaptive immune system by expressing tumor necrosis factor (TNF-α), IL-12, and IL-23, which further activate Th17, Th1, and Th22 autoimmune cells and release cytokines, leading to the development of psoriatic KP.[51]

Keratinocyte-derived nerve growth factor (NGF) also plays a key role in inducing keratinocyte proliferation and neuroinflammation through NGF receptor, p75 neurotrophin receptor, and TrkA (tyrosine kinase A) *in vivo*, further causing immunologic abnormalities.[52] Moreover, an increased formation of NGF and vascular endothelial growth factor (VEGF) also promotes angiogenesis and Koebernization.[52,53]

The number of ulex europaeus agglutinin-I (UEA-I) binding sites on keratinocytes is frequently utilized as a marker of terminal differentiation. Compared with KP-negative and healthy individuals, Koebner-positive lesions indicate noticeably increased binding sites of UEA-I on keratinocytes. This change in the behavior of keratinocytes increases the likelihood of developing KP.[54] Repeated injury leading to persistent skin barrier disruption increases epidermal metabolic demands and further reduces oxygen levels in local tissues. The reduction of oxygen content in the epidermis activates hypoxia-inducible factor-1α, a crucial transcription factor, which upregulates VEGF production under hypoxia stress.[55,56]

Furthermore, the elevated levels of S100A7 (psoriasin) and S100A15 (koebnerisin),[57] increasing proportion of CD4+/CD8+ T cells in the epidermis, and the abnormal presence of α2 β1 integrin in the suprabasal epidermal layers,[58] downregulation of mechanosensitive polycystin 1 protein,[59] atypical chemokine receptor 2,[60] and ionotropic NMDARs on the keratinocytes also contribute to inducing the formation of KB following skin injury.[61]

Secondly, regarding the role of injury in initiating Koebernization, superficial injury alone seems to be inadequate to promote the development of KP, and the dermis injury may be more prominent.[62] For instance, a previous report showed that suction bullae do not cause KP because suction separates the epidermis from the dermis with minimal injury and little immune reaction.[2]

There has been a pivotal role of mast cells and their mediators, mainly comprising three key molecules/five pathways in inducing the formation of KB.(a) The mast cell in dermis-releasing tryptase directly acts as a mitogen to increase the proliferation of keratinocytes and dermal fibroblasts.[63] Furthermore, tryptase may activate the protease-activated receptor (PAR-2) receptors located on the mast cells to positively feedback regulate itself secretion.[64] PAR-2 activation may further release IL-8 and promote the inflammatory process.[65] (b) Mast cell-derived IL-17 activates two signaling cascades, including the activation of p38 mitogen-activated protein kinase and NF-kB STAT3, to induce the development of KP.[50] (c) Moreover, keratinocytes may release IL-33, which activates the mast cells to release IL-6 and potentiate the inflammation.[66] Considering these mechanisms aforementioned, it strongly suggested that the pharmacological modulation of mast cells and corresponding molecules/pathways may serve therapy targets to inhibit the induction and/or progression of KP following skin injury or agents.[67]

Admittedly, injury of the epidermis accompanied by inflammation of the dermis may induce the formation of common KP, but to LP, an unusual and linear manifestation, the role of genetic background is indispensable.

Finally, the genetic mechanism of mosaicism and susceptibility may account for the pathogenesis of LP.[21,30] The concept of genetic mosaicism means that an individual with a heterozygous gene predisposes to psoriasis. During early embryogenesis, crossing over of the gene would occur in a somatic cell, leading to a loss of heterozygosity (LOH). LOH results in daughter cells mutated with hemizygousity or homozygousity. These daughter cells harboring somatic mutation(s) migrate following Blaschko's lines and are more susceptible to psoriasis.[42,43] This concept would offer an explanation as to why the linear lesions preceded the nonsegmental lesions and showed resistance to topical therapy.[21,28]

In addition, there may be an association between its distributions along Blaschko lines, human leukocyte antigen (*HLA*) class I alleles, and somatic recombination.[21,68] Studies in early-onset psoriasis patients indicated that *HLA-B*13* and *HLA-C*06* are the most frequent alleles.[69] By contrast, the types of alleles in LP patients from Brazilian children in recent findings are not exactly the same. These alleles involve *HLA-B*13*, *HLA-C*06*, *HLA-B*37*, *HLA-B*39*, and *HLA-Cw*12*, which are considered to be closely related to psoriasis under the onset age of 18 years.[68,70] The most reasonable explanation is that genetic mosaicism phenomena due to chromosomal abnormalities are believed to be responsible for these distinctions.

LCE3B (late cornified envelope, *LCE*) and *LCE3C*, as two members of the *LCE* group genes, locating on chromosome 1 in the epidermal differentiation complex, are responsible for skin injury owing to their participation in epidermal

differentiation and maintaining cutaneous barrier function. While deletion of the *LCE3B* and *LCE3C* (*LCE3C_LCE3B-del*) genes weakens this function. This would make the skin more vulnerable to invasion by microorganisms or other environmental molecules, which could trigger congenital or adaptive immune responses that could further lead to clinical diseases. One study demonstrated that *LCE3C_LCE3B-del*, a well-known psoriasis risk factor, has 23% population-attributable risk.[71] And then one hypothesis holds that the KP may be caused by damaging the skin barrier and/or insufficient barrier repair. However, Bergboer *et al*. found that there is no correlation between *LCE3C_LCE3B-del* and the KP in psoriasis patients by Logistic regression analysis. In addition, the strongest known psoriasis risk factor, *HLA-C*06*, is not related to the KP. These results suggest that the KP in psoriasis is unlikely to be dependent on the *LCE3B*/*C* genotype.[72] Therefore, it also cannot rule out the role of other members of the *LCE* genome.

Employing transcriptomic or molecular analysis to compare DNA samples from LP and non-LP skin areas, LP and PV, respectively, Onoufriadis et al. discovered that the transcriptomic changes of LP are highly similar to those of PV.[73] Moreover, 8 of the 664 differentially expressed genes DEGs (*CARD14*, *CARD6*, *FUT2*, *GJB2*, *LCE3D*, *PRSS53*, *RPS6KA4*, and *STAT3*) in the LP datasets are also remarkable candidate PV genes implicated by genome-wide association studies and were overexpressed in LP lesional skin.[74] Another top 14 DEGs (*S100A9, SERPINB3, LCE3D, S100A8, RHCG, GJB2, SERPINB4, HEPHL1, VNN3, FABP5, UGT1A7, SPRR2E, LCN2, and GBP6*) organizing dendrogram of log-fold change (FCs) (lesional vs. nonlesional skin) in the LP dataset also shows the distinctions between LP and PV. Search tool for the retrieval of interacting genes/proteins functional enrichment analysis on the selected clusters highlighted pathways mainly involved in the formation of cornified envelopes and inflammatory responses, such as the IL-4, IL-13, and IL-36 signaling pathways. Nevertheless,due to the limitation of rare cases, more studies will need to explore differences between LP and PV in order to help elucidate the molecular basis of psoriasis.[73]

Collectively, for the final manifestation of LP, the presence of predisposing genetic and environmental factors, infection, or trauma is required [Figure 3]. This explains why LP less occurs at birth but develops later in life.[14,21,22,31] Although we can acquire some key information or speculative conclusions about the pathogenesis of LP from the underlying pathogenesis of KP linked to psoriasis, anyway, this lacks more convincing or direct evidence to verify these opinions. Also, there has not been a better animal model, like genetic mosaicism of gain-of-function mutations of causal gene, or knock-out of susceptibility genes, focusing on the research of function of these loci, which may be a promising research prospects. Again, gene microarray analysis of LP will bring significant interest and challenges to provide more evidence. The exact mechanisms for LP may need to be investigated in depth in the future.

Clinical manifestation

The clinical manifestation of LP is a continuous or intermittent linear arrangement of scaly papules and plaques similar to PV, with clear boundaries. And pustular LP is even rarer.[75]

According to the age of onset disease, in our opinion, it can be divided into congenital LP and acquired LP, although there has always been controversy about the actual existence of congenital LP as a unique entitative disease.[76] Lehman and Rahil observed that LP is reported in one-third of congenital psoriasis cases, in contrast to being rare in general childhood psoriasis.[77] Moreover, all three patients with congenital LP are female, supporting the hypothesis that this disorder may originate from functional X chromosome mosaicism.[77,78] In addition, HLA CW6/A2 has been found to be associated with congenital LP.[79]

In 2018, Say and his colleagues[20] reported the first large series of 30 LP patients from 14 French medical centers and described 6 main points: (a) patients with type II more than those with type I, (b) two-thirds of LP cases onset in childhood and one-third patients with familial history of psoriasis, (c) the clinical type of LP comprising two components: Plaque and pustule, (d) very rare head involvement, (e) systemic therapies such as methotrexate and TNF-a possibly revealing the true nature of LP, and (f) less effective treatment for LP than for classical psoriasis, especially for biotherapy.

However, this study may have some limitations. One is why patients with KP are excluded from studies exists selection bias, although most scholars believed that the presentation of new psoriatic lesions related to LP suggest the role of KP. The other is that some cases without complete histological and immune-histochemical examination inevitably remains diagnostic bias, since some cases might have been ILVEN.

By contrast, a larger sample report by Chen *et al*. summarized LP's 10 characteristics. These features include: (a) earlier onset than PV, (b) More common in male individuals, notably in type II, (c) mostly cases distributing unilaterally, with no differences for left or right site, (d) asymptomatic or slight itching, similar to classical psoriasis, (e) about 10% of patients with a positive family history, (f) 80% involvement of the nails/scalp in type II, (g) one-third of patients with exogenous triggering or exacerbating factors, (h) approximately 40% concomitant PV, especially in

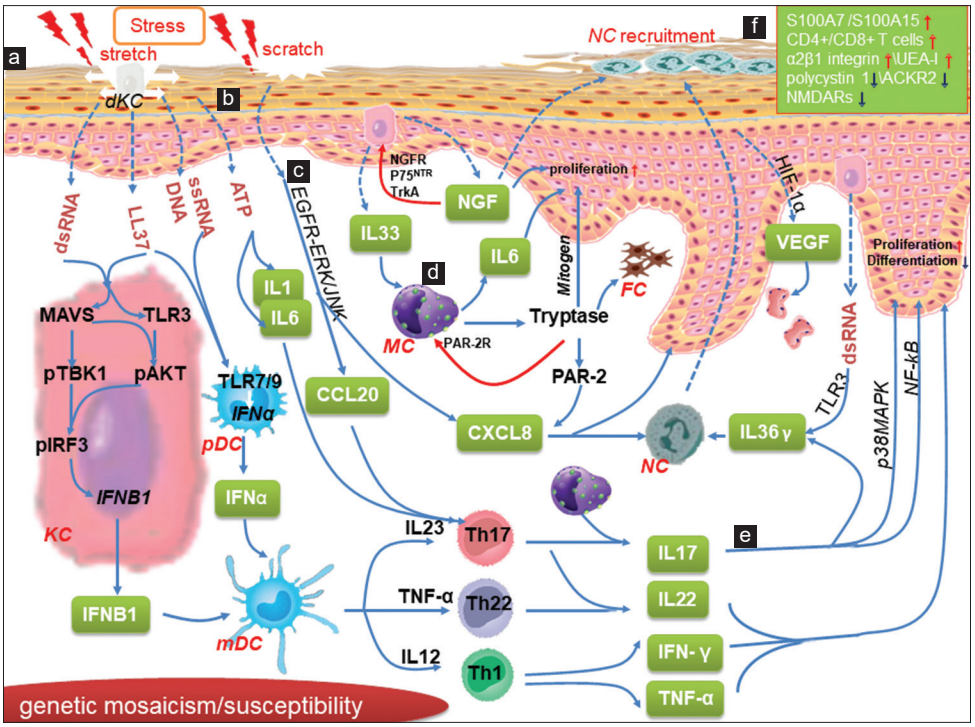

Figure 3: Schematic pathogenesis of KP related to linear psoriasis. Under genetic mosaicism/susceptibility background, environmental stress factors drives skin inflammation through multiple signaling pathways or molecules. These changes ultimately exacerbate psoriatic phenotypes, thus inducing KP related to LP, (a) Skin injury (stretch/scratch) causes keratinocyte (KC) damage, which induces LL37 production and releases self-nucleic acids (double-stranded RNA [dsRNA], single-stranded RNA, and DNA). One is that LL37 enables dsRNA recognition by mitochondrial antiviral signaling protein and toll-like receptors3 in KC, further activates pAKT-pTBK1-pIRF3 signaling cascade and subsequently initiates transcription of *IFNB1* gene. The other is that LL37-DNA complexes was recognized by TLR7 or 9 in pDCs, promoting *IFNA* gene transcription. Both IFNb and IFNa, facilitate mDCs maturation and further release tumor necrosis factor-alpha, interlukin-12 (IL-12), and IL-23, activate Th1 and Th17 autoimmune cells and produce cytokines, leading to the development of psoriatic KP, (b) Extracellular adenosine triphosphate releasing from stressed keratinocytes, leads to subsequent production of IL-1 and IL-6, further activates Th17 cell and triggers KP, (c) Mechanical scratching on the keratinocytes induces release of cysteine-cysteine motif chemokine ligand 20 and C-X-C motif chemokine ligand 8 via the EGFR-ERK/JNK pathway, which results in accumulation of Th17 cells and neutrophils in the lesions. An increased formation of vascular endothelial growth factor induced by hypoxia-inducible factor-1α also promotes angiogenesis and Koebernization, (d) Mast cells-derived mediators including IL-6, tryptase and proteinase-activated receptor-2, further release IL-8 and induce the formation of new psoriatic lesions, (e) Key cytokine IL-17 activates two signaling cascades, including the activation of p38MAPK and NF-kB STAT3 to induce the development of KP, (f) In addition, S100A7/S100A15 ↑, CD4+/CD8+ T cells↑,α2β1 integrin ↑, UEA-I ↑, NGF↑, polycystin 1↓, ACKR2 ↓, and NMDARs↓, etc., regulate the formation of LP lesion

type II(100%),(i) histopathologically conformed to features of classical psoriasis, (j) relatively favorable response to antipsoriatic treatment, although poor for superimposed areas in type II.[45]

So far, at the molecular level, there have been no experiments in psoriasis to attempt to clarify the concept of superimposed segmental manifestation. Most likely, such studies will be performed in the near future in patients with superimposed LP. The results may provide new insight into the molecular basis of psoriasis.[27]

The occurrence of psoriasis in a unilateral or linear pattern can give rise to a significant diagnostic dilemma. So LP is easily confused with other diseases such as inflammatory linear verrucous epidermal nevus (ILVEN), lichen striatus (LS), linear lichen planus, and invasion of epidermal nevus by psoriasis based on clinical and histological resemblance.[32,35,80]

About 20% of patients show comorbidity of systemic diseases, including psoriatic arthritis,[20] Down syndrome,[25] HIV infection,[81] systemic lupus erythematosus,[82] bipolar disorder,[18] and posttraumatic stress disorder.[83] Moreover, accompanied local disorders like porokeratotic eccrine ostial and dermal duct nevus, and LS are also documented.[84,85]

Inspection methods: Dermoscopy and reflectance confocal microscope

Dermoscopy and reflectance confocal microscopy (RCM) provide noninvasive, high-resolution imaging techniques that contribute to acquiring a correct diagnosis with dynamic, repeated analyses of multiple lesions in the same individual.

On dermoscopy, LP lesions exhibit evenly distributed dotted and globular blood vessels, or linear/curved blood vessels (hairpin-like/twisted ring), over a pinkish background with diffuse or scattered silvery-white scales.[86] RCM shows particles with medium and high refraction in the stratum corneum, which is larger than those with regular distribution, indicating parakeratosis. Under the corner layer, a decrease in the number of cellular layers of the honeycomb structure

is observed. Moreover, enlarged dermal nipples are present at the dermal-epidermal junction, and dilated capillaries are seen in the middle, showing a psoriasis-like pattern of hyperplasia. The superficial dermis contains numerous enlarged low-refractive canalicular structures.[86,87]

Taken together, all the manifestations under scopy are in accordance with PV, not specific for LP lesions. Hence, merely relying on the results of Dermatoscopy or RCM is not sufficient to diagnose LP, and it is quite necessary to closely combine clinical manifestations with other laboratory tests.

Histopathology

Although noninvasive examination methods such as dermoscopy and RCM help to establish a more accurate diagnosis, the role of histopathology is nonsubstitutable for evaluating LP. Biopsy examination of the plaque-type lesions reveals an acanthotic epidermis is composed of pale keratinocytes with the absence of the granular layer. Parakeratosis and Munro microabscesses have been observed in the stratum corneum. The dermis shows elongated papillae with a thin suprapapillary plate covering the tips.[86,87]

Diagnosis and differential diagnosis

LP, inflammatory linear verrucous epidermal nevus (ILVEN), LS, blaschkitis, and other linear dermatoses often have similar clinical manifestations, which sometimes makes clinical diagnosis very difficult.[87] Especially, it has striking alike to ILVEN. Exploration of the relation between LP and ILVEN has been attempted through clinical, histological, genetic, immunohistochemical, and therapeutic studies.

Clinically, LP demonstrates late onset but rapid progression of asymptomatic or slightly pruritic lesions with a possible involvement of the scalp and nails, and a favorable response to antipsoriatic treatment.[31] In contrast, Altman and Mehregan established six clinical diagnosis criteria for ILVEN: (a) early age of onset, (b) more frequent in women with a ratio of 4:1, (c) frequent involving the left leg, (d) itching, (e) classical psoriasiform appearance, and (e) resistance to antipsoriasis therapy.[88]

And histologically, LP presents with papillomatosis, acanthosis, and parakeratosis with an absent or minimal granular layer, generally similar to PV. By comparison, ILVEN demonstrates abruptly alternating areas of hypergranulosis with orthokeratosis, and parakeratosis with agranulosis. An inflammatory infiltration is present in the upper dermis.

Immunohistochemical staining can be done in such doubtful cases. Ito and colleagues showed that in ILVEN the involucrin expression is increased in orthokeratotic regions but is deficient in parakeratotic regions; by contrast, in parakeratotic areas of psoriasis, most suprabasal keratinocytes express involucrin.[89,90] Previously, Ki67 expression is increased in typical lesions of LP but lower in ILVEN, making it an alternative potential marker to distinguish LP from ILVEN.[91,92] There is little expression of keratin 10 (K10) in psoriasis, whereas these levels remain normal in ILVEN. Other authors have suggested that the behavior of other markers (elastin, K10, K16) may be useful to differentiate between unilateral psoriasis and ILVEN.[89,91]

Although immunohistochemical expression of K10, K16, Ki-67, and involucrin may be useful for differentiating ILVEN from LP, these results have been reported in only a few ILVEN cases.[38,89-92] But subsequent research indicated that the immunostaining pattern of Ki-67, K16, involucrin, and filaggrin may be insufficient to discriminate inflammatory linear verrucous epidermal nevus from PV.[93] This distinction between PV and LP may be attributed to different genetic backgrounds.

Therefore, it is not always easy to differentiate between LP and ILVEN, even with a pathological and immunohistochemical evaluation. In fact, there are not absolute diagnostic criteria. Munro microabscesses, which are typically seen in LP, may also be observed in ILVEN. Many authors have suggested that LP is an ILVEN or the result of an isomorphic effect on a preexisting epidermal nevus.[94] However, could it be the opposite? That means ILVEN might also be a form of LP. So, more data is needed to support this opinion.[94]

Further differential diagnoses are LS and blaschkitis. Although sometimes it is very difficult or even impossible to distinguish the disorders on purely clinical grounds, in most cases a definite diagnosis of either disease can be made easily.

Therapies of linear psoriasis

Currently, there are no official guidelines for how to treat LP. It seems reasonable to follow the treatment recommendations for classical psoriasis. In order to facilitate clinical treatment decisions, the International Psoriasis Council 2020 Delphi Consensus recommends reclassifying the severity of psoriasis by continuing to use binary classification based on the three levels of mild, moderate, and severe psoriasis.[95]

Hence, psoriasis patients should be categorized as candidates for topical therapy or systemic therapy. The systemic treatment meets at least one of the following criteria: (a) BSA >10%, (b) disease involving special areas: Face, palms, soles, genitalia, scalp, or nails, and (c) failure of topical therapy. The main systemic therapies for psoriasis include biological agents and traditional treatment measures such as older drugs and phototherapy.[95]

Moreover, previous studies have shown that more than half of patients with LP achieve favorable outcomes, particularly for those who accept topical drugs, and systemic treatment/phototherapy.[26,31,83,96,97]

Topical corticosteroids and vitamin D derivatives are the most prevalent therapies for localized BP. In more complex and resistant situations, systemic antipsoriatic drugs, phototherapy, and biologics should be considered.[31] Chen *et al*. demonstrated that over 50% of LP patients utilize topical treatment.[45] However, sometimes, the treatments are more challenging, with variable reported clinical responses to traditional treatments of LP. Ghoneim *et al*. speculated that it may be caused by the LOH in lesion cells, leading to distinct variations in the proliferation/differentiation of keratinocytes, further affecting treatment outcomes.[83] For instance, Seitz *et al*. found that one patient with the classic psoriatic lesions on their limb joints responds well to treatment with topical corticosteroids and dithranol, whereas the linear lesions only showed slight and temporary improvement.[28]

And the proportion of patients who attempt systemic treatment, light therapy, or biologics is approximately 20%. Based on varying in treatment responses, type II patients can be categorized into at least three conditions: (a) LP resistant and classical plaque sensitive to treatment, (b) both LP and classical plaque sensitive, and (c) treatment sensitive for LP.[96]

The disparities in treatment effect may mainly be due to discrepancies in racial susceptibility backgrounds, or divergent targets of biological agents. Chen *et al*. found that type I patients seem more satisfied with antipsoriatic treatment than type II patients.[45] This suggests that there are still differences between type I and II in the pathogenesis. Chen *et al*.[45] found all four patients respond favorably to ixekizumab in their investigation. Say and colleagues indicated that ustekinumab treatment results in a response in two out of three LP patients.[20] Interestingly, one patient with severe psoriasis was successfully treated with adalimumab. However, paradoxical Blaschko LP lesions persisted and were resistant to etanercept after adalimumab withdrawal. Eventually, ustekinumab therapy resulted in complete resolution for the remaining lesions.[98] Similar research about LP treated by infliximab also supports this result.[96] Furthermore, owing to the nature of malignant tumors, it is not recommended to utilize cyclosporine and JAK inhibitors in the treatment of PD-1 inhibitor-related LP, as they have the potential to induce or exacerbate tumor progression.[20,45,99]

CONCLUSION

The occurrence of LP suggests that the KP may be responsible for these lesions. A number of agents/triggers have been reported to induce the development of LP through activation of KP in healthy skin areas. The different mechanisms that may contribute to promoting the formation of KP include multiple molecules and signal

Table 1: Key findings relevant to linear psoriasis and Kobner phenomenon

Year	Authors	Key findings related with LP and KP
2016	Diani *et al*.[13]	Koebner published the article about KP following trauma to healthy skin areas of patients with psoriasis in 1877
1951	Leslie[15]	Leslie and Sobel named Blaschko LP
1982	Miller[46]	The KP tends to be suddenly present in some specific stage of psoriasis, usually appears within about 1–2 weeks, but may range from 3 days to 2 years
1989	Atherton *et al*.[37]	A 6-years-old boy presented with naevoid psoriasis
1990	al-Fouzan *et al*.[79]	*HLA CW6/A2* gene is associated with congenital LP case
1991	de Jong *et al*.[89]	Differential diagnosis of ILVEN versus LP was performed by a clinical, histological and immunohistochemical study
1991	Happle[21]	Somatic recombination may explain etiology of LP
2002	Weiss *et al*.[1]	LP formation is not only related to disruption to the epidermis but also with deep tissue injury
2006	Happle[22]	LP is classified with isolated LP (type I) and superimposed LP (type II)
2007	Kalayciyan *et al*.[42]	Experimental induced KP in patients with psoriasis
2007	Magalhães *et al*.[68]	Genetic research of LP in Brazilian children revealed different HLA haplotypes
2009	Seitz *et al*.[28]	Superimposed LP showed differential therapeutic response of linear and nonlinear lesions
2009	Verma[16]	Striae were shown to be responsible for causing the KP with LP
2012	Sengupta *et al*.[80]	Naevoid psoriasis and ILVEN are distinct entities which on rare occasions may co-exist in the same subject
2012	Bergboer *et al*.[72]	KP in psoriasis is not associated with deletion of late cornified envelope genes *LCE3B* and *LCE3C*
2013	Ferreira *et al*.[90]	Involucrin plays an important role in differentiating diagnosis between LP and ILVEN
2018	Say *et al*.[20]	A review described 6 main clinical and therapeutic aspects of LP
2019	Ji and Liu[67]	KP leading to the formation of new psoriatic lesions involved mast cells and their mediators, and mainly comprised three key molecules/five pathways
2022	Onoufriadis *et al*.[73]	Transcriptomic analysis of LP revealed shared and distinct features with psoriasis vulgaris
2022	Wen *et al*.[86]	Dermoscopy combined with reflectance confocal microscopy is a better, noninvasive diagnostic method for LP
2023	Zhang *et al*.[14]	Mechanical scratch/stretch inducing inflammatory response, keratinocyte proliferation and differentiation, angiogenesis, and genetic susceptibility or mosaicism, participate in the pathogenesis of KP
2024	Chen *et al*.[45]	Recent review summarized 10 clinical characteristics of LP and related therapeutic progress

LP=Linear psoriasis; KP=Kobner phenomenon; HLA=Human leukocyte antigen; ILVEN=Inflammatory linear verrucous nevus; *LCE*=Late cornified envelope

pathways based on genetic background. Currently, there are no official guidelines for the treatment of LP. It seems logical to follow the treatment guidelines for classical psoriasis. All Key findings relevant to LP and Kobner phenomenon were summarized in Table 1. LP is very rare, and most literatures consist of case or series reports with insufficient evidence-based medicine. Further research, such as the establishment of LP experimental models or gene microarray analysis, is needed to clarify the pathogenic genes of LP.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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