

Classical Cutaneous Lichen Planus Exhibiting Locus Minoris Resistentiae

To the Editor,

A young male sought consultation for multiple itchy lesions on his right leg that had developed over the last 15 days. The patient had sustained a major road traffic accident 2 years prior with an open fracture in the right leg. The fractures were subsequently treated surgically with orthopedic implants containing stainless steel and skin grafting.

Examination revealed multiple well-defined, flat-topped, and violaceous papules and plaques surmounted with Wickham's striae on his right lower leg and foot, suggestive of classical lichen planus [Figure 1a and b]. The part was swollen and disfigured with significant lymphedema that had been exacerbated in the past 6 months. The largest plaque was linear and corresponded to the suture line (with equal distribution on either side of the suture line) on the right lower leg. Predominantly, the lesions were localized to the locoregional site affected by trauma/surgery, while the rest of the body parts were spared. The donor site on left thigh or other cutaneous/mucosal sites were not involved.

A skin biopsy was obtained from the dorsum of the foot. In addition, he was patch tested from the orthopedic implant series [Table 1], which did not reveal any positive results on the fourth day. His hepatitis B and C serology were negative. The histopathology from skin biopsy revealed orthokeratosis, hypergranulosis, basal cell vacuolization, apoptotic bodies, and a band-like dense lymphomononuclear

infiltrate at the dermoepidermal junction, suggestive of classical lichen planus [Figure 2a and b]. He was advised topical clobetasol propionate 0.05% and experienced significant improvement. On the next visit, he presented with scattered papules of classical lichen planus on both legs and trunk. He was prescribed prednisolone 30 mg/day, which was tapered within a month. All the lesions resolved, except those on the right leg and foot, which were persistent despite the significant improvement.

The index case highlights an important manifestation of "locus minoris resistentiae (LMR)," a Latin proverb that means "offering less resistance." The same has been called as Ruocco's immunocompromised district (ICD)^[1,2] and rightly explains the preferential localization of multiple dermatoses to the sites that have experienced some prior pathological insult in the form of trauma, burns, vaccination, herpes zoster, ultra-violet/ionizing radiation, lymphedema, and certain genetic/developmental defects. These dermatoses can be infectious, inflammatory, or neoplastic.

The development of lichen planus localizing on healed erythema multiforme, saphenous venectomy,^[3] graft-donor

Table 1: The orthopedic implant series used to rule out implant dermatitis in the patient

Allergen (with conc. % w/w and vehicle)

Nickel sulfate hexahydrate 5% pet
Potassium dichromate 0.5% pet
Cobalt chloride hexahydrate 1% pet
Titanium dioxide 10% pet
Vanadium 5% pet
Methyl methacrylate 2% pet
N,N-Dimethyl-4-toluidine 5% pet
Hydroquinone 1% pet
Benzoyl peroxide 1% pet
Gentamicin sulfate 20% pet



Figure 1: (a) Multiple, well-defined, flat-topped, violaceous papules and plaques surmounted with Wickham's striae are present on the right lower leg and foot. The part is swollen and disfigured with significant lymphedema. The largest plaque is linear and corresponds to the suture line on right lower leg. The other leg is conspicuously lesion free. (b) Close-up of the lesions on right lower limb

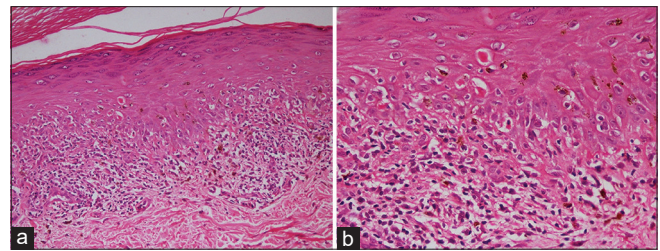


Figure 2: (a) Histopathology shows orthokeratosis, hypergranulosis, basal cell vacuolization, apoptotic bodies and a band-like dense lymphomononuclear infiltrate at the dermoepidermal junction (Hematoxylin and Eosin, 200x). (b) Higher magnification revealing multiple apoptotic keratinocytes, dermal colloid bodies, extensive basal cell degeneration, pigment incontinence, and lymphohistiocytic infiltrate obliterating the dermoepidermal junction (Hematoxylin and Eosin, 400x)

site,^[4] and radiation site^[5-7] has been previously described. Of all, the radiation field seems to be the most important precipitating factor for the development of lichen planus.^[2,3]

Orthopedic and surgical trauma constitutes an important contributor of LMR or ICD. Major fractures, associated trauma, and healing can effectively jeopardize microcirculation in the affected areas. It commonly manifests as phlebotymphedema and significantly alters the local immune response through dysregulated distribution and the clearance of lymphocytes and other immune effector cells. This can result either in an enhanced susceptibility or an apparent resistance to the development of certain dermatoses in that site. Eczematous lesions developing after total knee replacement were described as an entity named SKINTED^[8] (surgery of the knee, injury to the infra-patellar branch of the saphenous nerve, and traumatic eczematous dermatitis) and recent literature has attributed autonomic innervation dermatitis resulting from surgical trauma as another important cause for SKINTED, and formation of ICD at the sites of prior to accidental or planned, orthopedic or surgical trauma.^[9]

Brodell syndrome/recurrent lymphangitic cellulitis syndrome,^[10] Stewart Treves syndrome (development of angiosarcoma on lymphedema), and development of pemphigus, bullous pemphigoid, and various malignancies have been described over a lymphedematous limb following trauma. We could not find a prior report describing the development of lichen planus on an orthopedic trauma site or over a grafted skin.

The term Wolf's isotopic response has historically been used to define the "occurrence of a new dermatosis on the site of a previously healed unrelated dermatosis". Since there was no history or evidence of a prior primary dermatosis before lichen planus occurred, using this term shall not be ideal. ICD, however, can explain the occurrence of LP on a grafted skin or otherwise damaged skin (like radiotherapy, or herpes zoster); therefore, it is a more inclusive term. On a similar note, Koebner's phenomenon describes the development of new lesions of a pre-existing dermatosis at the site of trauma. Since, in this patient, no lesions of lichen planus existed before trauma, using the term Koebner's phenomenon does not seem to be ideal, though the mechanism of new-onset LP in Koebner's phenomenon and ICD appears to be essentially similar.

When a patient with prior orthopedic trauma and implants develops a dermatosis over that site, contact dermatitis to implant should be ruled out, including the noneczematous variants. The index patient had negative patch tests and the histopathology was suggestive of classical lichen

planus rather than a lichenoid reaction to the implants. Dissemination to generalized lichen planus further substantiated the diagnosis. The T-cells once activated can travel to the rest of the skin and cause generalized version of the dermatosis that originated in the ICD.

To conclude, the onset and persistence of lichen planus at the site of orthopedic trauma and skin grafting represents an interesting event. The additional factors that govern the development of a particular dermatosis in an ICD should be studied further.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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