Editorial

Lichen planus: The newer treatment trends

Lichen planus(LP) is a pruritic inflammatory disorder affecting skin, mucous membranes, skin appendages, and other organ systems.[1] It has internal associations, including autoimmune conditions, glucose intolerance, dyslipidemia, and cardiovascular disorders.[2] In skin classically LP is presented as small, sharply demarcated, flat and polygonal erythematous-livid papules, that may progress to form plaques which is often described by "six-P"; planar, purple, polygonal, pruritic, papule and plaque.[3] In classic oral lichen planus (OLP), the mucosa is involved bilaterally and symmetrically with six clinical patterns: reticular (most common), erosive/ulcerative, plaque-like, papular, bullous, and atrophic.[4] The classic cutaneous LP is typically a self-limiting disease with a good prognosis. Most patients with cutaneous lesions spontaneously clear within 12–24 months.[5]

Lichen planus is thought to be a T-cell–mediated autoimmune disease of unknown aetiology. It is believed to be influenced by genetic and environmental factors, where certain exogenous antigens alters epidermal self-antigens, leading to the activation of cytotoxic T(Tc) cells. The altered self-antigens cross-react with normal self-antigens on basal keratinocytes, resulting in T-cell targeting and apoptosis.[6] A complex inflammatory network comprising Tc, Th1, Il-23/Th-17 axis, dendritic cells, keratinocytes, NK cells, macrophages, mast cells, and Tregs are involved in its initiation and maintenance.3Treating LP is practically challenging, tricky and variable for different subtypes. The apex treatment objective is the clearing of the cutaneous lesions and accompanying symptoms including painful oral erosions which significantly hampers food intake.[7]

A gold standard treatment for LP is lacking. However, potent topical, intralesional or systemic corticosteroids, systemic retinoids or cyclosporine are proven first-line treatments.7Phototherapy using ultraviolet B light, psoralen plus ultraviolet A light, the combination of UV/PUVA with retinoids, photodynamic therapy, oral sulphasalazine and topical calcineurin inhibitors are approved as second-line modalities. Hydroxychloroquine, azathioprine, methotrexate, mycophenolate mofetil, or biologics targeting IL-12/23 are listed as third-line modalities.[7] Though therapeutic options for LP have remained largely stagnant, with advances in drug development and understanding of the pathophysiology of, novel therapies targeting cytokines (biologics) and small molecules blocking intracellular signalling are included in the list. In refractory erosive/ulcerative OLPanti-IL-17, anti-IL-12/IL-23, and anti-IL-23 monoclonal antibodies represent an effective and safe alternative therapy.[8] Janus kinase–signal transducer and activator of transcription (JAK-STAT)-dependent cytokines like IFN-γ can play a significant role in the molecular pathogenesis of LP.[9] JAKs are one of the therapeutic targets to control overreactive immune responses. Tofacitinib is the most reported JAK inhibitor, and it is effective in treating oral, nail, hypertrophic, and scalp LP.[10-12] The success of oral JAK1 inhibitor, upadacitinib in the treatment of refractory, biopsy-proven lichen planus has been recently published.[13]Considering the wide spectrum anti-inflammatory role of the PDE4 inhibitor, apremilast has been evaluated and found effective in LP refractory to topical steroids.[14] Mycophenolate mofetil can also be considered as another off-label option for recalcitrant cases not responding to first and second-line treatments.[15]

With the innovations of newer molecules, it appears that diseases like LP are going to be within the reach of physicians control and patients suffering will be significantly reduced down.

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