Abstract N°: ID-1397

Topic: Hair and nail disorders

Nail Lichen Planus: From Basics to Breakthroughs

Carina-Andreea Bazon*[1], Andreea-Caterina Rusu[1], Petronela Balaceanu[1], Maria-Georgiana Pîslariu[1], Anca Zbranca[1, 2]

[1]"Saint Spiridon" County Clinical Emergency Hospital, Iasi, Romania
[2]"Grigore T. Popa" University of Medicine and Pharmacy, Iași, Romania

Introduction

Nail lichen planus (NLP) is an inflammatory disorder characterized by lichenoid involvement of the nail matrix and/or nail bed, with a significant risk of permanent nail destruction. Matrix involvement occurs in the vast majority of patients, and delayed or insufficient treatment may lead to irreversible scarring and anonychia.

Recent therapeutic approaches emphasize early, often systemic, intervention to prevent progression and permanent nail loss.

Materials and Methods

We conducted a narrative synthesis of the clinical features and therapeutic approaches to nail lichen planus based on expert consensus recommendations and recent therapeutic developments. Disease severity was stratified into mild, moderate, and severe forms according to clinical features such as longitudinal ridging, nail plate thinning, onycholysis, fissuring, pterygium formation, and anonychia. Treatment strategies were analyzed according to the number of affected nails, presence of matrix and/or nail bed involvement, and signs of scarring.

Results

NLP predominantly affects the nail matrix, resulting in longitudinal ridging and grooves, thinning of the nail plate, fissuring, erythema of the lunula and onicolysis. Progressive cases may develop dorsal pterygium, severe atrophy, or complete nail loss. The distinction between active inflammatory disease and cicatricial, irreversible stages is essential, as only the active phase responds to therapy.

Traditional first-line therapy includes intralesional corticosteroids, particularly triamcinolone acetonide, administered every 4–6 weeks for several sessions.

In cases involving multiple nails or showing rapid progression, systemic therapy with corticosteroids, retinoids, or cyclosporine is recommended.

Severe or refractory disease may require additional immunosuppressants such as azathioprine or mycophenolate mofetil.

Recent years have introduced new therapeutic perspectives. Activation of the JAK-STAT pathway has been implicated in lichen planus pathogenesis, and emerging data suggest that JAK inhibitors, including tofacitinib, baricitinib, and abrocitinib, may represent effective options in refractory cases. There is also evidence of topical formulations of tofacitinib and ruxolitinib showing promising results.

Low-dose naltrexone has also shown positive outcomes, with clinical improvement or stabilization in a small patient series, and a favorable safety profile.

A key paradigm shift in current management is the recommendation for early systemic treatment in active matrix disease, even when only a few nails are affected, in order to prevent permanent scarring.

Conclusions

Nail lichen planus is a potentially destructive condition that requires early recognition and prompt treatment. Disease severity and the presence of matrix involvement or scarring determine the therapeutic approach. Intralesional corticosteroids remain the standard first-line therapy, while systemic agents are indicated in moderate to severe or progressive disease. Emerging therapies, particularly JAK inhibitors and low-dose naltrexone, represent promising options for refractory cases. Early, aggressive treatment is essential to prevent irreversible nail damage.