Abstract N°: ID-257

Topic: Pruritus

Efficacy of Tofacitinib in Three Children With Prurigo Nodularis and Its Mechanistic Rationale

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Introduction

Prurigo nodularis (PN) is a chronic dermatologic condition manifesting as multiple papulonodular lesions occurring on the background of intense pruritus, often restricted to middle-aged and older persons (5th decade), and rarely in children. It is an extremely challenging condition with no FDA approved therapies and is often associated with higher failure and relapse rates. Considering the multiple mechanism involed in the pathogenesis, a combinations of several medications are used to control the disease activity rather than monotherapy. Tofacitinib is a non-selective Janus kinase (JAK) inhibitor and has been used in adult PN with successful results, however there is a paucity of data on its efficacy in paediatric PN. This study aims to assess the efficacy of oral tofacitinib in children with refractory prurigo nodularis and explain the mechanistic rationale.

Materials and Methods

Three children of age 9 , 6 and 12 years (2 females and 1 male respectively) presented to the dermatology outpatient department with generalised excoriated papulonodular lesions over both upper and lower limbs (extensors > flexors) and over the trunk. Duration of the disease were 6 months , 5 month and 1 year respectively. The Baseline Pruritus grading severity score (PGSS) was 8 , 9 and 11 respectively out of 19. Treatment history included the use of topical and oral steroids, and antihistamines without satisfactory results for the first patient, no prior treatments for the second patient, topical steroid and methotrexate use for the third patient. We started tofacitinib in a dose of 5 mg twice daily, after ruling out secondary causes for PN. Other supportive treatment included emollients and antihistamines. The disease activity was monitored using PGSS score and the dose was reduced to 5mg once the PGSS score reduced by 75%. Baseline investigations, including complete hemogram, liver and kidney function test, Mantoux test, chest X-ray, fasting lipid profile, and viral markers were done prior to the initiation of tofacitinib.

Results

All 3 children responded well to the treatment. The average duration of treatment was 7 months. Average Baseline PGSS score was 9.3 and post treatment score was 2. One child had a history of atopic dermatitis. There was a dramatic decrease in pruritus (>50% reduction) for all 3 patients with in 1 week after starting the treatment. After achieving a 75% decrease in PGSS score the dose was reduced to 5mg and was stopped after a month for one patient and after 2 months for other 2 patients. The response was maintained on topical emollients and antihistamines. No relapse were noted for a period of 6 months.

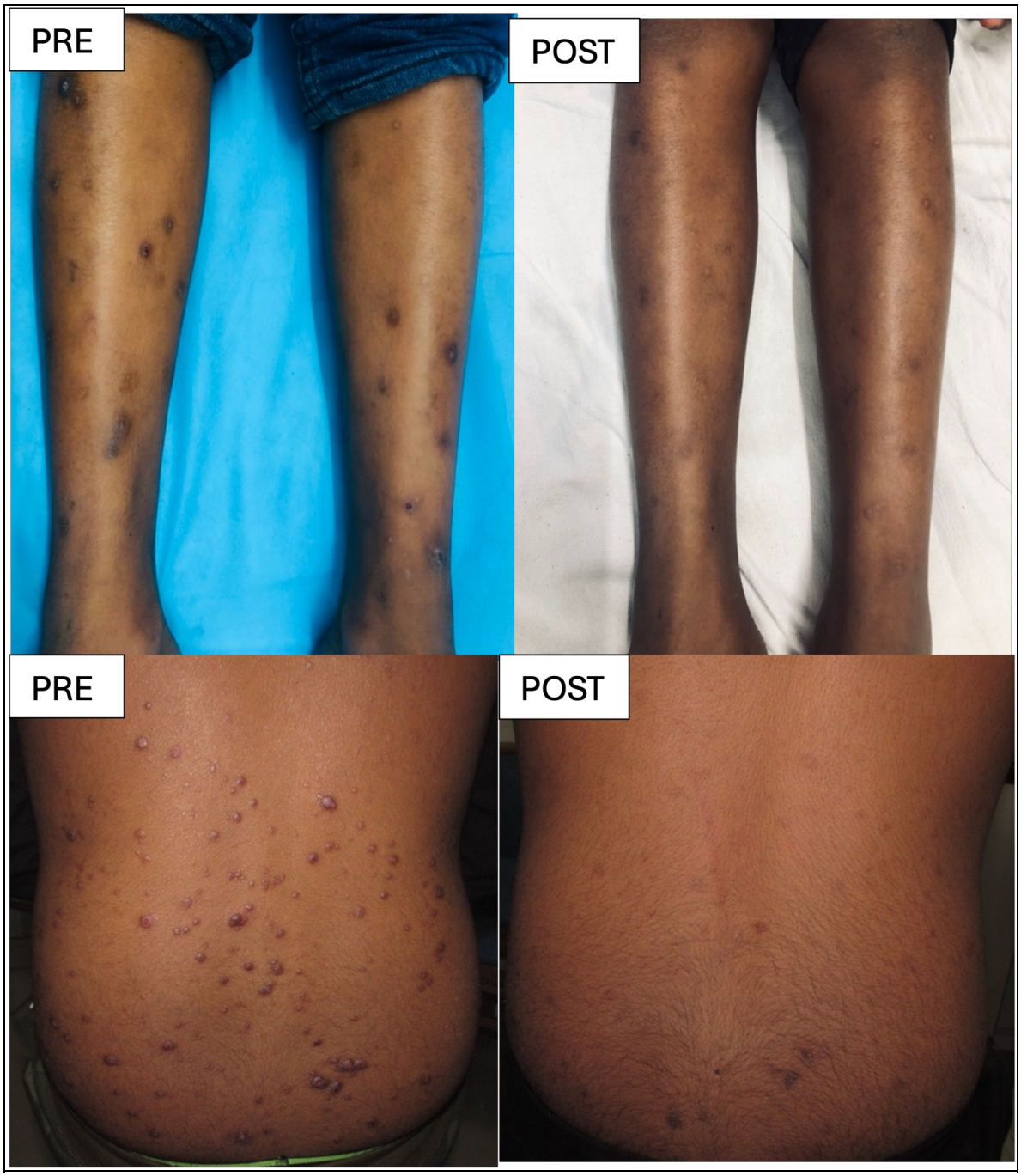

Image showing improvement with tofacitinib in a 12-year-old child with refractory prurigo nodularis

Conclusions

There is an increased expression of interleukin (IL) 4, IL 13, IL 31, IL22 & IL 17 in PN, which mediate their action via the JAK-STAT pathway. Tofacitinib, a JAK 1,3 inhibitor, would inhibit Th2 and Th17 expression and specifically inhibit the signalling of IL4 (JAK1, JAK3, STAT6) and IL31(JAK1, STAT3, STAT5), making it a cost-effective treatment option in refractory prurigo nodularis when compared to biologicals.

Abstract N°: ID-703

Topic: Pruritus

Impact of Dupilumab on Sleep Quality in Patients with Prurigo Nodularis: A Narrative Literature Review

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Introduction

Prurigo nodularis (PN) is a chronic dermatosis characterized by intensely pruritic nodules, with a significant impact on quality of life and sleep, being strongly associated with insomnia and non-restorative sleep[1–3]. Persistent pruritus leads to excoriations and perpetuates the itch–scratch cycle, sustained by immune and neural dysfunctions mediated by interleukins (IL)-4, IL-13, and IL-31[1,3,4]. Therapeutic strategies capable of reducing pruritus, improving skin lesions, and restoring sleep are therefore essential[5]. Dupilumab, a monoclonal antibody that selectively blocks IL-4 and IL-13 signaling, has demonstrated efficacy in relieving pruritus and improving skin manifestations, showing promise in sleep restoration[6].

Materials and Methods

A narrative review was conducted using the PubMed (Medline) and SciELO databases, including studies published in English over the past five years. Five studies were selected for analysis. Outcomes were assessed using validated instruments, including the Sleep Numeric Rating Scale (Sleep-NRS), Dermatology Life Quality Index (DLQI), Pruritus Numeric Rating Scale (p-NRS), Non-Restorative Sleep Scale (NRSS), and Skin Pain Numeric Rating Scale (NRS).

Results

Five studies were included, comprising a total of 270 patients treated for periods ranging from 16 to 84 weeks. Chiricozzi et al. (2020)[4] reported a reduction in pruritus NRS scores from 8.9 to 2.7 and sleep disturbance scores from 8.2 to 1.7 after 16 weeks ($p < 0.001$), with sustained improvement up to 36 weeks. Gao et al. (2023)[6], in a cohort of 24 patients, observed improvements in pruritus (7.50 ± 2.21 to 1.41 ± 0.91) and sleep disturbance (5.33 ± 3.29 to 0.18 ± 0.59) after 16 weeks ($p < 0.001$), alongside improvements in DLQI scores. Paganini et al. (2024)[7] identified progressive improvement up to 84 weeks, with near-zero scores for both pruritus and sleep disturbance. Gael et al. (2025)[8] reported an increase in NRSS scores from 3.89 to 8.67 after 16 weeks, indicating sleep restoration. Yosipovitch et al. (2023)[9] (PRIME study) demonstrated a mean improvement of 2.7 points in Sleep-NRS scores at 24 weeks. Overall, the findings confirm that chronic pruritus significantly impairs sleep and quality of life, while dupilumab exerts a multidimensional effect by reducing pruritus, improving sleep, and alleviating physical and psychological burden in the management of PN[4,6,10].

Conclusions

Dupilumab is an effective and safe therapeutic option for prurigo nodularis, significantly reducing pruritus, restoring sleep, and providing an overall improvement in patients' quality of life.

Abstract N°: ID-895

Topic: Pruritus

Neurocutaneous Mechanisms Underlying Gabapentinoid Therapy for Neuropathic Pruritus

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Introduction
Neuropathic pruritus is defined as itch arising secondary to neuronal injury and represents a distinct subtype of chronic itch. Neuropathic pruritus may arise from a wide range of underlying etiologies, including root compression, multiple sclerosis, small fiber neuropathy, cerebral or spinal infarctions, and space-occupying lesions of the central nervous system. Neuronal injury in these cases causes dysregulation and hypersensitivity of the somatosensory nervous system, leading to increased localized itch sensations. Conventional antipruritic therapies are frequently ineffective due to the nonhistaminergic nature of neuropathic itch. Gabapentinoid therapy, specifically gabapentin and pregabalin, is increasingly utilized to modulate abnormal neural signaling and improve pruritic symptoms in this context. The objective of this review is to assess the current literature regarding the efficacy of gabapentinoid therapy in neuropathic pruritus and to summarize the implications for clinical practice.

Materials and Methods
A comprehensive literature search was conducted using PubMed and Google Scholar. Articles published between 2000 and 2024 were reviewed. Search terms included *neuropathic pruritus, chronic pruritus, gabapentin therapy, mechanism of gabapentinoid therapy, postherpetic pruritus, brachioradial pruritus, notalgia paresthetica, sensory processing, and central sensitization.* Studies were included if they addressed etiologies, clinical manifestations, treatment strategies for neuropathic pruritus, or the mechanisms and safety considerations of gabapentinoid therapy. Thirteen sources, including review articles, clinical trials, case series, and observational studies, were included in the final analysis.

Results
Neuropathic pruritus is associated with altered neuronal excitability, inflammatory mediators, and central sensitization, often in the absence of epidermal nerve fiber loss. Clinical conditions such as postherpetic pruritus, notalgia paresthetica, and brachioradial pruritus exemplify these mechanisms. Gabapentin has demonstrated efficacy in these settings, with clinical studies showing meaningful reductions in itch severity and improved treatment adherence. Gabapentinoids exert their effects by binding the α2δ subunit of voltage-gated calcium channels, reducing abnormal afferent signaling, dorsal horn excitability, and central sensitization.

Conclusions
Gabapentinoid therapy is increasingly utilized in the management of neuropathic pruritus, reflecting growing recognition of the limitations of histaminergic treatments. Modulation of central and peripheral neural pathways supports the central sensitization model of chronic itch, providing a rational pharmacological target. Improved recognition of neuropathic pruritus allows for positive patient outcomes. Further studies are needed to optimize treatment protocols, evaluate long-term safety, and standardize clinical use in clinical practice.