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Therapeutic outcomes across Janus kinase inhibitors in prurigo nodularis

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Abstract

Background Prurigo nodularis (PN) is a debilitating skin condition. When inadequate disease control is achieved or other systemic therapies are contraindicated, Janus kinase inhibitors (JAKis) may be considered, although real-world evidence remains limited.

Objectives To investigate clinical findings and treatment outcomes among patients diagnosed with PN and treated with JAKis in a real-world setting.

Methods Retrospective cohort study across 23 Italian tertiary referral hospitals. Patients with PN were eligible if aged ≥ 18 years and had received a JAKi with a minimum follow-up of 12 weeks. The primary outcome was defined as the proportion of patients achieving a reduction of ≥ 4 points from baseline in the Peak Pruritus Numerical Rating Scale score. Key secondary outcomes included the rates of patients reaching a significant reduction in Investigator Global Assessment of Prurigo Nodularis Stage (IGA PN-S) and Investigator Global Assessment of Prurigo Nodularis Activity (IGA PN-A) scores.

Results Of the total, 71 patients met the inclusion criteria. At week 16, the proportion of patients achieving the primary outcome was 94% for upadacitinib [*n* = 52/55; 95% confidence interval (CI) 87–100%], 83% for abrocitinib (*n* = 10/12; 95% CI 52–98%) and 100% for baricitinib (*n* = 4/4; 95% CI 40–100%), with results sustained at week 24. An IGA PN-A score of 0/1 was achieved in 90% of patients treated with upadacitinib and abrocitinib and in 50% of those on baricitinib by week 24. Improvements were observed across all other secondary outcomes assessed, with no safety concerns reported.

Conclusions This study suggests that JAKis can achieve clinically meaningful outcomes in PN irrespective of atopic background, supporting their use across diverse patient profiles. Further research is warranted to validate these observations and explore their long-term effects.

What is already known about this topic?

- Prurigo nodularis (PN) is a debilitating condition marked by raised, intensely itchy, often painful nodules on the skin.
- When inadequate disease control is achieved or other systemic therapies are contraindicated, Janus kinase inhibitors may be considered in its management.
- Evidence supporting their therapeutic potential remains limited.

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What does this study add?

- This retrospective cohort study shows that upadacitinib, abrocitinib, and baricitinib achieved clinically meaningful outcomes in treating PN without raising any safety concerns.
- Our findings indicate that Janus kinase inhibitors may be considered for treating PN, regardless of atopic background, supporting their use across diverse patient profiles in clinical practice.
- Further research is warranted to validate these observations and explore their long-term effects.

Prurigo nodularis (PN) is a debilitating condition marked by raised, intensely itchy, often painful nodules on the skin.[1–3] Conventional treatments include corticosteroids, neuromodulating agents and immunosuppressants such as methotrexate, azathioprine or mycophenolate.[4–7] In recent years, the therapeutic landscape of PN has been enhanced by the introduction of dupilumab, a fully human IgG4 monoclonal antibody targeting the interleukin (IL)-4 receptor α, and nemolizumab, an IL-31 receptor α antagonist.[8–13] The Janus kinase (JAK)–signal transducer and activator of transcription protein pathway has also been implicated in PN development, playing a pivotal role in the signalling of several key drivers of its pathogenesis.[14,15] When inadequate disease control is achieved or other systemic therapies are contraindicated, JAK inhibitors (JAKis) may be considered, although evidence supporting their therapeutic potential remains limited.[16–23]

This study sought to address this knowledge gap by investigating clinical findings and treatment outcomes among patients diagnosed with PN who were treated with JAKis in a real-world setting.

Patients and methods

We performed a multicentre, retrospective cohort study across 23 Italian tertiary referral hospitals (Table S1; see Supporting Information). Eligible participants were aged ≥ 18 years, diagnosed with PN by a dermatologist and had received upadacitinib, abrocitinib or baricitinib between 1 January 2018 and 1 July 2024. Patients were excluded if they had a follow-up period less than 12 weeks, if their PN was secondary to medications or comorbidities or if they had concomitant moderate to severe atopic dermatitis (AD) or other active dermatological conditions that could potentially interfere with PN assessment. JAKis were prescribed either for the presence of comorbidities with an approved indication for these therapies or used for PN where the disease proved refractory to multiple systemic treatments. Alternatively, they were recommended in cases initially misdiagnosed with AD prurigo-like, who were later re-evaluated and newly diagnosed as PN based on an integrated assessment of disease course, lesion morphology and distribution, and absence of classic eczematous features. Identification of eligible patients was achieved through a retrospective review of electronic health records and specific datasets from the participating sites.

Data collection

Data including demographics (Table 1), medical history, disease severity, treatment outcomes and adverse events (AEs) were collected (Table 2). The overall population was stratified into six subgroups based on treatment type and daily dosage: upadacitinib (15 mg and 30 mg), abrocitinib (100 mg and 200 mg) and baricitinib (2 mg and 4 mg). Disease severity and therapeutic response were assessed by means of the Peak Pruritus Numerical Rating Scale (PP-NRS), Investigator Global Assessment of Prurigo Nodularis Activity (IGA PN-A), Investigator Global Assessment of Prurigo Nodularis Stage (IGA PN-S), Skin Pain Numerical Rating Scale (SP-NRS) and Sleep Disturbance Numerical Rating Scale (SD-NRS).[3–5] IGA PN-A and IGA PN-S were obtained directly from the medical records or calculated retrospectively with archival photos taken when treatment was initiated. The PP-NRS, SP-NRS and SD-NRS scales range from 0 (no symptoms) to 10 (severe symptoms) and assess symptom intensity over the preceding 24 h. Patient-reported outcomes related to overall mental health were assessed using the Hospital Anxiety and Depression Scale (HADS) and Dermatology Life Quality Index (DLQI).[3–5] Data were collected at baseline (defined as the initiation of JAKi therapy) and at 4, 16, 24, 48, 72 and 104 weeks post-baseline. At all timepoints, assessments were conducted within a permissible ± 4-week window.

Primary and secondary outcomes

The primary outcome was defined as the proportion of patients achieving a reduction of ≥ 4 points from baseline in PP-NRS score at any timepoint of analysis. Key secondary outcomes included the proportion of patients reaching an IGA PN-S or IGA PN-A score of 0 (clear) or 1 (almost clear) accompanied by a ≥ 2-point reduction from baseline at any timepoint of analysis. Additional secondary outcomes encompassed the analysis of trends over time in SP-NRS, SD-NRS, DLQI and HADS.

Statistical analysis

For primary and key secondary analyses, the number and percentages of patients achieving the outcome of interest were estimated, together with 95% confidence intervals (CI) based on the exact binomial distribution model for proportion. All percentages presented in this study were calculated based on the number of patients with available data at each timepoint. For patients treated with upadacitinib, the primary and key secondary outcomes were analysed using a univariable Fine–Gray model, with the drug dosage as an independent variable. PP-NRS was also summarized as a continuous variable. Other secondary analyses included continuous outcome scores, such as DLQI, PP-NRS, SD-NRS, SP-NRS and HADS. For continuous variables, absolute values, changes from baseline and percentage changes from baseline were summarized. Continuous data were analysed

Table 1 Patient demographics and clinical characteristics

	Upadacitinib 15 mg[d] (*N*=25)	Upadacitinib 30 mg[e] (*N*=30)	Abrocitinib 100 mg (*N*=9)	Abrocitinib 200 mg (*N*=3)	Baricitinib 2 mg (*N*=1)	Baricitinib 4 mg (*N*=3)	Overall (*N*=71)
Age (years)[a]							
Median (IQR)	46.0 (34.0, 57.0)	54.7 (45.8, 58.8)	55.9 (22.0, 57.9)	43.0 (42.0, 54.5)	53.0 (53.0, 53.0)	43.0 (38.0, 46.5)	51.1 (39.5, 58.3)
Mean (SD)	47.1 (13.7)	50.0 (12.1)	45.1 (18.0)	50.0 (13.9)	53.0 (NA)	42.0 (8.5)	48.1 (13.2)
Female sex	13 (52)	16 (53)	6 (67)	1 (33)	0 (0)	1 (33)	37 (52)
Body mass index							
Median (IQR)	25.0 (22.7, 26.6)	25.5 (22.5, 27.5)	23.1 (22.4, 28.0)	24.4 (24.1, 26.1)	26.6 (26.6, 26.6)	24.5 (23.2, 24.7)	25.0 (22.5, 27.0)
Mean (SD)	24.9 (2.8)	25.4 (3.4)	25.0 (3.9)	25.3 (2.1)	26.6 (NA)	23.8 (NA)	25.1 (3.1)
Ethnic groups							
White	24 (96)	29 (97)	9 (100)	3 (100)	1 (100)	3 (100)	69 (97)
Black	0 (0)	1 (3.3)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.4)
Asian	1 (4.0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.4)
Smoking habits							
Never	14 (56)	20 (67)	3 (33)	1 (33)	0 (0)	2 (67)	40 (56)
Former	6 (24)	7 (23)	6 (67)	1 (33)	0 (0)	1 (33)	21 (30)
Current	5 (20)	3 (10)	0 (0)	1 (33)	1 (100)	0 (0)	10 (14)
Family history							
PN	0 (0)	1 (3.3)	0 (0)	0 (0)	0 (0)	0 (0)	1 (1.4)
AD	9 (36)	11 (37)	2 (22	1 (33)	0 (0)	1 (33)	24 (34)
Atopy	11 (44)	11 (37)	3 (33)	2 (67)	0 (0)	3 (100)	30 (42)
PN duration (years)[a]							
Median (IQR)	3.0 (2.0, 6.0)	3.9 (3.0, 5.8)	4.9 (2.0, 5.9)	10.0 (6.5, 18.5)	2.0 (2.0, 2.0)	(0.5, 1.5)	(2.0, 6.0)
Mean (SD)	5.5 (6.5)	4.8 (3.1)	8.6 (15.3)	13.3 (12.3)	2.0 (NA)	1.0 (1.0)	5.7 (7.4)
Hospitalized due to PN[b]	1 (4.0)	1 (3.3)	0 (0)	1 (33)	1 (100)	0 (0)	4 (5.6)
Allergic rhinitis	11 (44)	7 (23)	1 (11)	2 (67)	0 (0)	1 (33)	22 (31)
Asthma	3 (12)	6 (20)	1 (11)	1 (33)	0 (0)	0 (0)	11 (15)
Allergic conjunctivitis	6 (24)	6 (21)	2 (22)	1 (33)	0 (0)	0 (0)	15 (21)
Food allergies	(0)	2 (6.9)	0 (0)	0 (0)	0 (0)	0 (0)	2 (2.9)
Serum IgE levels (IU mL^{-1})[a]							
Median (IQR)	125.5 (66.0, 437.0)	210.0 (89.0, 767.0)	134.0 (124.0, 154.0)	1517.5 (786.3, 2248.8)	25.7 (25.7, 25.7)	191.5 (172.3, 210.8)	153.0 (87.0, 497.0)
Mean (SD)	565.7 (1244.6)	724.5 (1195.4)	186.8 (157.1)	1517.5 (2068.3)	25.7 (NA)	191.5 (54.4)	595.4 (1119.4)
Previous PN medications[b]							
Topical steroids	21 (84)	30 (100)	9 (100)	3 (100)	1 (100)	3 (100)	67 (94)
Topical calcineurin inhibitor	8 (32)	10 (33)	2 (22)	1 (33)	0 (0)	0 (0)	21 (30)
Topical capsaicin	2 (8.0)	2 (6.7)	0 (0)	1 (33)	0 (0)	0 (0)	5 (7.0)
Phototherapy	3 (12)	3 (10)	1 (11)	1 (33)	0 (0)	0 (0)	8 (11)
Systemic steroids	17 (68)	14 (47)	4 (44)	3 (100)	0 (0)	3 (100)	41 (58)
Antihistamines	20 (80)	29 (97)	9 (100)	3 (100)	1 (100)	2 (67)	64 (90)
Antidepressants	1 (4.0)	2 (6.7)	1 (11)	2 (67)	0 (0)	0 (0)	6 (8.5)
Gabapentin	0 (0)	0 (0)	0 (0)	1 (33)	0 (0)	0 (0)	1 (1.4)
Pregabalin	0 (0)	2 (6.7)	0 (0)	1 (33)	0 (0)	0 (0)	3 (4.2)
Ciclosporin	18 (72)	25 (83)	9 (100)	2 (67)	0 (0)	3 (100)	57 (80)
Methotrexate	3 (12)	14 (47)	5 (56)	0 (0)	0 (0)	0 (0)	22 (31)
Dupilumab[c]	11 (44)	11 (37)	2 (22)	3 (100)	1 (100)	0 (0)	28 (39)
Tralokinumab[c]	0 (0)	5 (17)	0 (0)	0 (0)	0 (0)	1 (33)	6 (8.5)

All data are presented as *n* (%) unless otherwise indicated. AD, atopic dermatitis; AE, adverse event; BMI, body mass index; F, female; IgE, immunoglobulin E; IQR, interquartile range; M, male; *N* number, NRS, numerical rating scale; PN, prurigo nodularis; SD, standard deviation. [a]At JAK introduction. [b]Prior to introduction of JAK inhibitors. [c]Among patients treated with upadacitinib, three had a treatment history that included both dupilumab and tralokinumab. [d]Two patients initially treated with upadacitinib 15 mg daily increased their dosage to 30 mg daily during the follow-up period. One patient increased the dosage after 2 weeks and was included in the upadacitinib 30 mg group for data analysis, while the other increased the dosage after 10 months and was also grouped with the upadacitinib 30 mg cohort. [e]Two patients initially treated with upadacitinib 30 mg daily reduced their dosage to 15 mg daily. One reduction was due to the development of JAK-related acne and the other followed successful disease control. Both patients were categorized in the upadacitinib 30 mg group for data analysis.

by median and interquartile range and categorical data with counts and percentages; the denominator for all percentages was the number of participants in the specific population, unless otherwise specified. All analyses were stratified by timepoint and treatment group. A P-value < 0.05 was considered statistically significant. All analyses were performed with R software, version 4.1.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Study population

At the cut-off date for this analysis, we retrospectively screened the records of 77 patients who received JAKis, regardless of age and sex, and identified 71 who met the inclusion criteria. The cohort included 69 White patients and

Table 2 Baseline features at the time of introduction of Janus kinase inhibitors

	Upadacitinib 15 mg (*N*=25)[b]	Upadacitinib 30 mg (*N*=30)[c]	Abrocitinib 100 mg (*N*=9)[c]	Abrocitinib 200 mg (*N*=3)	Baricitinib 2 mg (*N*=1)	Baricitinib 4 mg (*N*=3)	Overall (*N*=71)
Concomitant PN-specific therapy[a]							
Topical steroids	11 (44.0)	7 (23.2)	3 (33.3)	1 (0.0)	1 (100)	1 (33.3)	24 (33.8)
Topical calcineurin inhibitor	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (100)	0 (0.0)	1 (1.4)
Systemic steroids	1 (4.0)	2 (6.7)	0 (0.0)	1 (33.3)	0 (0.0)	0 (0.0)	4 (5.6)
Antihistamines	0 (0.0)	3 (10.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (4.2)
Antidepressants	1 (4.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (4.2)
Distribution of PN							
Bilateral	18 (72)	21 (70)	5 (56)	3 (100)	1 (100)	3 (100)	51 (72)
Unilateral	7 (28)	9 (30)	4 (44)	0 (0)	0 (0)	0 (0)	20 (28)
Involved sites							
Head	3 (12.0)	4 (13.3)	1 (11.1)	1 (33.3)	0 (0.0)	1 (33.3)	10 (14.1)
Neck	7 (28.0)	5 (16.7)	1 (11.1)	0 (0.0)	0 (0.0)	0 (0.0)	13 (18.3)
Trunk	16 (64.0)	25 (83.3)	6 (66.7)	1 (33.3)	0 (0.0)	1 (33.3)	49 (69.0)
Upper limbs	17 (68.0)	27 (90.0)	8 (88.9)	2 (66.7)	0 (0.0)	2 (66.7)	56 (78.9)
Lower limbs	18 (72.0)	24 (80.0)	9 (100)	3 (100)	1 (100)	2 (66.7)	57 (80.3)
Type of cutaneous lesions							
Hypo/hyperpigmented macules	13 (52.0)	9 (30.0)	4 (44.4)	2 (66.7)	1 (100)	2 (66.7)	31 (43.7)
Nodules	22 (88.0)	13 (43.3)	2 (22.2)	3 (100)	0 (0.0)	3 (100)	43 (60.6)
Papules	15 (60.0)	14 (46.7)	4 (44.4)	2 (66.7)	1 (100)	2 (66.7)	38 (53.5)
Plaques	2 (8.0)	15 (50.0)	7 (77.8)	1 (33.3)	0 (0.0)	0 (0.0)	25 (35.2)
Ulcers	3 (12.0)	2 (6.7)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	5 (7.0)
PN lesions							
0	2 (8.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (2.8)
1–19	4 (16.0)	1 (3.3)	2 (22.2)	0 (0.0)	0 (0.0)	2 (66.7)	9 (12.7)
20–100	16 (64.0)	28 (93.3)	7 (77.8)	3 (100.0)	1 (100.0)	1 (33.3)	56 (78.9)
> 100	3 (12.0)	1 (3.3)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	4 (5.6)
Lesions with excoriations or crusts							
0	0 (0.0)	6 (20.7)	5 (55.6)	0 (0.0)	0 (0.0)	0 (0.0)	11 (15.7)
1–25	5 (20.0)	5 (17.2)	1 (11.1)	0 (0.0)	0 (0.0)	1 (33.3)	12 (17.1)
26–50	10 (40.0)	8 (27.6)	1 (11.1)	2 (66.7)	0 (0.0)	1 (33.3)	22 (31.4)
51–75	9 (36.0)	7 (24.1)	1 (11.1)	0 (0.0)	1 (100.0)	1 (33.3)	19 (27.1)
76–100	1 (4.0)	3 (10.3)	1 (11.1)	1 (33.3)	0 (0.0)	0 (0.0)	6 (8.6)
Healed lesions							
0	13 (52.0)	22 (73.3)	8 (88.9)	2 (66.7)	1 (100.0)	2 (66.7)	48 (67.6)
1–25	10 (40.0)	8 (26.7)	1 (11.1)	1 (33.3)	0 (0.0)	1 (33.3)	21 (29.6)
26–50	2 (8.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (2.8)
51–75	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
76–100	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
PP-NRS score baseline							
N	24	30	9	3	1	3	70
Mean (SD)	8.9 (1.08)	8.3 (1.67)	8.4 (0.73)	9.0 (1.73)	10.0 (0.00)	9.3 (1.15)	8.6 (1.36)
Median (IQR)	9.0 (8.0, 10.0)	8.0 (8.0, 10.0)	8.0 (8.0, 9.0)	10.0 (7.0, 10.0)	10.0 (10.0, 10.0)	10.0 (8.0, 10.0)	8.0 (8.0, 10.0)
IGA PN-A baseline							
N	25	30	9	3	1	3	71
Mean (SD)	3.2 (0.69)	3.1 (0.57)	2.8 (0.83)	3.0 (0.00)	3.0 (0.00)	2.7 (0.58)	3.1 (0.64)
Median (IQR)	3.0 (3.0, 4.0)	3.0 (3.0, 3.0)	3.0 (3.0, 3.0)	3.0 (3.0, 3.0)	3.0 (3.0, 3.0)	3.0 (2.0, 3.0)	3.0 (3.0, 3.0)
IGA PN-S baseline							
N	25	30	9	3	1	3	71
Mean (SD)	3.0 (0.71)	3.0 (0.41)	2.9 (0.60)	3.3 (0.58)	3.0 (0.00)	2.3 (0.58)	3.0 (0.57)
Median (IQR)	3.0 (3.0, 3.0)	3.0 (3.0, 3.0)	3.0 (3.0, 3.0)	3.0 (3.0, 4.0)	3.0 (3.0, 3.0)	2.0 (2.0, 3.0)	3.0 (3.0, 3.0)
Skin pain NRS baseline							
N	22	30	9	3	1	2	67
Mean (SD)	5.0 (3.48)	5.8 (2.23)	6.1 (0.78)	6.7 (4.16)	7.0 (0.00)	8.0 (1.41)	5.7 (2.66)
Median (IQR)	6.0 (2.0, 8.0)	7.0 (5.0, 7.0)	6.0 (6.0, 7.0)	8.0 (2.0, 10.0)	7.0 (7.0, 7.0)	8.0 (7.0, 9.0)	7.0 (5.0, 7.0)
Sleep NRS baseline							
N	24	30	9	3	1	3	70
Mean (SD)	6.6 (2.67)	7.1 (2.48)	7.4 (1.81)	5.3 (5.03)	9.0 (0.00)	7.3 (3.79)	6.9 (2.59)
Median (IQR)	7.5 (5.0, 8.0)	8.0 (5.0, 8.0)	8.0 (8.0, 8.0)	6.0 (0.0, 10.0)	9.0 (9.0, 9.0)	9.0 (3.0, 10.0)	8.0 (5.0, 8.0)
DLQI score baseline							
N	21	30	9	3	1	2	66
Mean (SD)	21.7 (7.97)	23.2 (6.32)	23.2 (6.38)	24.3 (4.93)	18.0 (0.00)	30.0 (0.00)	22.9 (6.76)
Median (IQR)	22.0 (18.0, 28.0)	24.0 (20.0, 26.0)	24.0 (24.0, 25.0)	22.0 (21.0, 30.0)	18.0 (18.0, 18.0)	30.0 (30.0, 30.0)	24.0 (20.0, 27.0)
HADS D score baseline							

(*Continued*)

Table 2 (*Continued*)

	Upadacitinib 15 mg (*N*=25)[b]	Upadacitinib 30 mg (*N*=30)[c]	Abrocitinib 100 mg (*N*=9)[c]	Abrocitinib 200 mg (*N*=3)	Baricitinib 2 mg (*N*=1)	Baricitinib 4 mg (*N*=3)	Overall (*N*=71)
N	14	14	3	2	1	–	34
Mean (SD)	8.7 (6.71)	12.1 (9.18)	13.0 (4.36)	15.5 (0.71)	4.0 (0.00)	–	10.8 (7.61)
Median (IQR)	9.0 (2.0, 15.0)	12.5 (4.0, 19.0)	15.0 (8.0, 16.0)	15.5 (15.0, 16.0)	4.0 (4.0, 4.0)	-	12.5 (4.0, 16.0)
HADS A score baseline							
N	14	14	3	2	1	-	34
Mean (SD)	8.7 (6.98)	12.0 (9.23)	11.3 (6.43)	16.5 (6.36)	4.0 (0.00)	–	10.6 (7.89)
Median (IQR)	8.5 (3.0, 15.0)	10.5 (5.0, 20.0)	14.0 (4.0, 16.0)	16.5 (12.0, 21.0)	4.0 (4.0, 4.0)	–	10.5 (3.0, 16.0)

All data are presented as *n* (%) unless otherwise indicated. A, anxiety; D, depression; HADS, Hospital Anxiety and Depression Scale; IGA PN-A, Investigator Global Assessment of Prurigo Nodularis Activity; IGA PN-S, Investigator Global Assessment of Prurigo Nodularis Stage; IQR, interquartile range; NRS, numerical rating scale; PN, prurigo nodularis; PP-NRS, Peak Pruritus Numeric Rating Scale; SD, standard deviation. [a]Throughout the study period. [b]Two patients initially treated with upadacitinib 15 mg daily increased their dosage to 30 mg daily during the follow-up period. One patient increased the dosage after 2 weeks and was included in the upadacitinib 30 mg group for data analysis, while the other increased the dosage after 10 months and was also grouped with the upadacitinib 30 mg cohort. [c]Two patients initially treated with upadacitinib 30 mg daily reduced their dosage to 15 mg daily. One reduction was due to the development of JAK-related acne and the other followed successful disease control. Both patients were categorized in the upadacitinib 30 mg group for data analysis.

37/71 (52%) were female, with a mean age of 48.1 (SD 13.2) years. Upadacitinib, abrocitinib and baricitinib were recommended in 55 patients (77%; 25 treated at 15 mg and 30 at 30 mg), 12 patients (17%; 9 treated at 100 mg and 3 treated at 200 mg) and 4 patients (6%; 1 treated at 2 mg and 3 at 4 mg), respectively. Nearly half of the PN population had prior exposure to biologic therapies, with a higher prevalence of patients previously treated with dupilumab (*n*=28; 39%) compared with those treated with tralokinumab (*n*=6; 8%). Throughout the study period, at least 1 concomitant therapy for PN was administered in 24 of 55 patients treated with upadacitinib (44%), 5 of 12 with abrocitinib (42%) and 3 of 4 with baricitinib (75%).

Effectiveness outcomes

At week 4, the proportion of patients achieving the primary outcome, defined as a reduction of at least 4 points in the PP-NRS, was 61% in the upadacitinib group (*n*=32/52; 95% CI 4775%), 33% in the abrocitinib group (*n*=4/12; 95% CI 9.9–65%), and 50% in the baricitinib group (*n*=2/4; 95% CI 6.8–93%). By week 16, the response rates increased to 94% for upadacitinib (*n*=52/55; 95% CI 87–100%), 83% for abrocitinib (*n*=10/12; 95% CI 52–98%) and 100% for baricitinib (*n*=4/4; 95% CI 40–100%). At week 24, the primary outcome was maintained in 87% of patients receiving upadacitinib (*n*=42/48; 95% CI 77–96%), while 100% of patients in both the abrocitinib (*n*=10/10; 95% CI 69–100%) and baricitinib (*n*=2/2; 95% CI 16–100%) groups achieved this level of improvement.

Regarding the IGA scores at week 24, an IGA PN-S score of 0/1 was observed in 81% of patients treated with upadacitinib (*n*=39/48; 95% CI 67–91%), and in 100% of patients treated with abrocitinib (*n*=10/10; 95% CI 69–100%) and baricitinib (*n*=2/2; 95% CI 16–100%). Similarly, an IGA PN-A score of 0/1 was achieved by 90% of patients in the

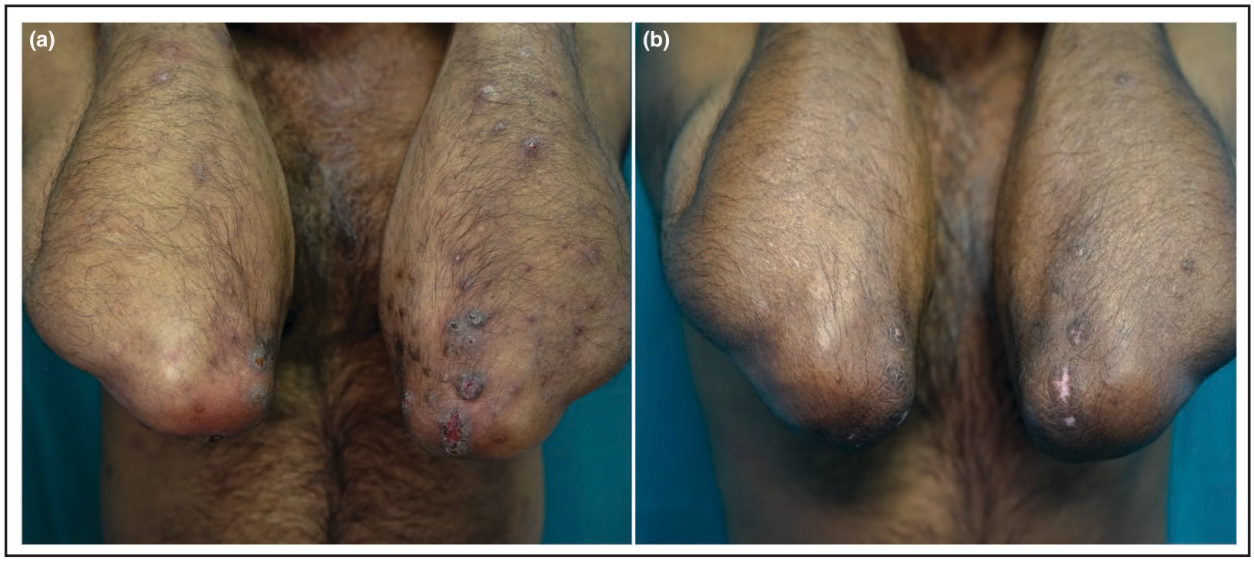

Figure 1 Response of a patient with prurigo nodularis to upadacitinib treatment over time. (a) Patient at week 0. (b) After 8 weeks of treatment.

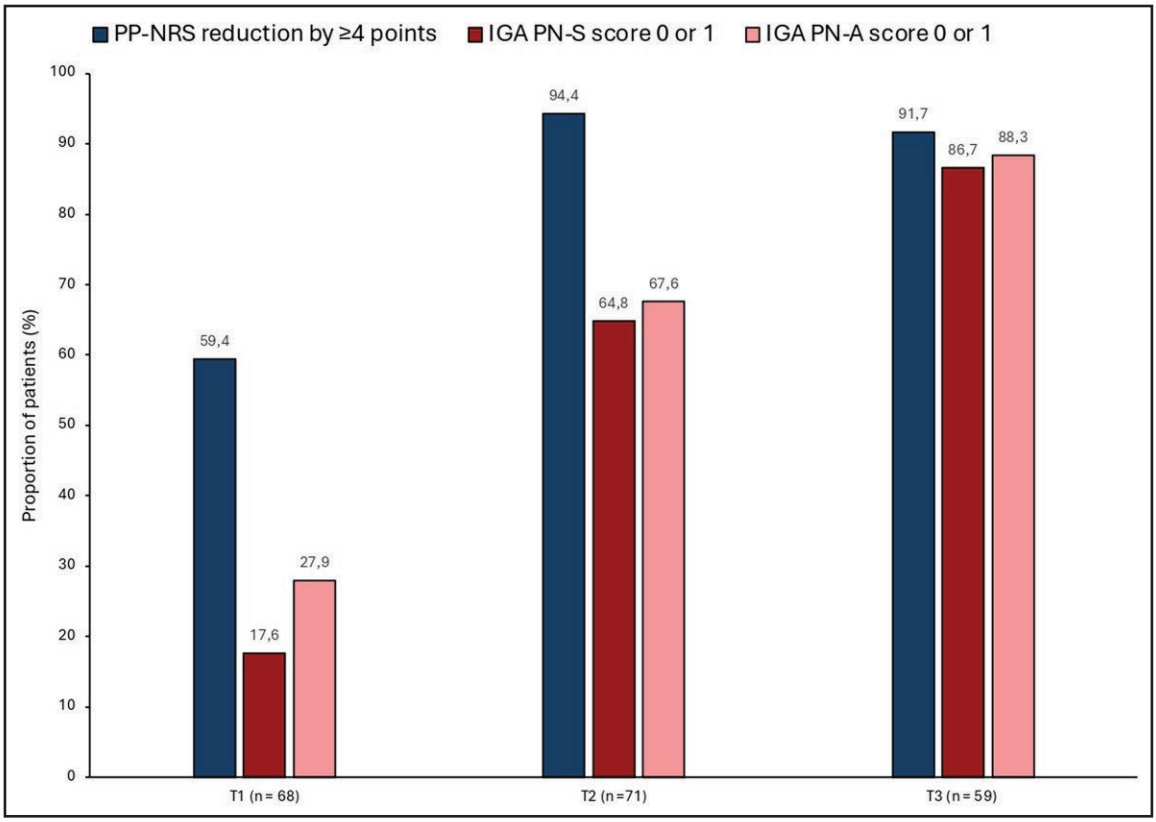

Figure 2 The proportion of patients receiving Janus kinase inhibitors who achieved a ≥ 4-point reduction in Peak Pruritus Numerical Rating Scale (PP-NRS), Investigator Global Assessment for Prurigo Nodularis Stage (IGS PN-S) score of 0 or 1 and Investigator Global Assessment for Prurigo Nodularis Activity (IGA PN-A) score of 0 or 1 at T1, T2 and T3. T1, 4 weeks; T2 16 ± 4 weeks; T3, 24 ± 4 weeks.

upadacitinib group (*n* = 43/48; 95% CI 77–97%), 90% in the abrocitinib group (*n* = 9/10; 95% CI 43–95%) and 50% in the baricitinib group (*n* = 1/2; 95% CI 7–93%; Figure 2).

Mean (SD) PP-NRS scores at baseline were 8.6 (1.4) for upadacitinib, 8.6 (1.0) for abrocitinib and 9.5 (1.0) for baricitinib. At week 24, these scores decreased to 1.0 (1.9) for upadacitinib (an 88% reduction from baseline), 0.6 (1.0) for abrocitinib (a 93% reduction), and 1.5 (2.1) for baricitinib (an 84% reduction).

A further analysis focusing on patients treated with upadacitinib compared two dosing regimens using a Fine–Gray model with the 15 mg dosage as the reference. For the 30-mg dose, the hazard ratio (HR) for achieving a PP-NRS reduction was 0.93 (*N* = 54, events = 52; 95% CI 0.85–1.03; *P* = 0.157). The HR for improvement in IGA PN-S was 1.18 (*N* = 55, events = 46; 95% CI 0.81–1.72; *P* = 0.398) and for IGA PN-A improvement it was 1.09 (*N* = 55, events = 49; 95% CI 0.78–1.51; *P* = 0.612).

Measures of quality of life and mental health also showed substantial therapeutic benefit. Baseline mean (SD) DLQI scores were 22.6 (7.0) for upadacitinib, 23.5 (5.8) for abrocitinib and 26 (6.9) for baricitinib. Baseline scores for the HADS were: for HADS-A, 10.4 (8.2) for upadacitinib, 13.4 (6.2) for abrocitinib and 4 for baricitinib; for HADS-D, 10.4 (8.0) for upadacitinib, 14.0 (3.3) for abrocitinib and 4 for baricitinib. Baseline SP-NRS scores were 5.5 (2.8), 6.3 (1.9) and 7.7 (1.1), respectively. By week 24, the DLQI scores improved by 86% in the upadacitinib group, 88% in the abrocitinib group and 96% in the baricitinib group. Improvements in HADS-A scores were 65%, 71% and 25%, respectively, while HADS-D scores improved by 69%, 67% and 25%. Finally, SP-NRS scores decreased by 93% in the upadacitinib group and reached 100% improvement in both the abrocitinib and baricitinib groups.

Additional analyses, including those stratified by dose and by the impact of JAKis on the type and number of cutaneous lesions (Figure 1a, b), are provided in Tables S2 and S3 (see Supporting Information). Long-term assessments for the upadacitinib subgroup (up to 24 months) are summarized in Tables S4 and S5 (see Supporting Information), while data stratified by potential confounders (such as concomitant atopic background or mild active AD) are available in Table S6 (see Supporting Information).

Safety

JAKis demonstrated a favourable safety profile with no serious or life-threatening AEs occurring throughout the study. AEs were reported in 9 of 71 patients (13%): 3 on upadacitinib 15 mg, 5 on upadacitinib 30 mg and 1 on abrocitinib 200 mg. Common AEs were dyslipidaemia (*n* = 3; 4%) and headache (*n* = 3; 4%). Less frequent AEs included JAKi-associated acne (*n* = 1; 1%), oral candidiasis (*n* = 1; 1%), neutropenia (*n* = 1; 1%) and herpes simplex (*n* = 1; 1%). None of the patients had to stop taking JAKis because of AEs and temporary treatment suspensions were reported in a small subset of patients (*n* = 5; 7%).

Discussion

In this multicentre retrospective cohort study, we evaluated the therapeutic outcomes and safety profile of upadacitinib, abrocitinib and baricitinib in a real-world setting. The effectiveness of JAKi in PN is probably attributable to their capacity to modulate multiple immune pathways involved in the disease pathogenesis. All three agents evaluated in this study share JAK1 inhibition as a key mechanism, a critical mediator of type 2 inflammatory cytokines such as IL-4, IL-13 and IL-31, cytokines whose signalling is closely linked to itch intensity and is known to be dysregulated in PN.[4,14] Inhibition of JAK1 not only disrupts the downstream signalling of these cytokines but may also attenuate pruritus through additional mechanisms. For example, the engagement of IL-4Rα on sensory neurons leads to subsequent JAK1 activation, thereby facilitating itch signal propagation.[24] Moreover, JAK blockade may reduce pruritus by modulating transient receptor potential vanilloid receptor 1 (TRPVR1) signalling within dorsal root ganglia.[25] Robust evidence from murine models further substantiates the role of JAK1 inhibition in suppressing itch.[24]

Our findings demonstrated that patients treated with these agents experienced a notable reduction in disease burden across all primary and secondary outcomes assessed. The rapid reduction in the PP-NRS scores and the high rates of patients achieving clinically significant outcomes in both the IGA PN-S and IGA PN-A highlight the therapeutic effect of JAKis. Improvements in core signs and symptoms of PN were evident as early as 4 weeks, with benefits continuing to increase through week 24. Of note, slightly more than half of the patients in the upadacitinib and abrocitinib subgroups achieved these outcomes without concomitant therapy for PN. In a subset of patients treated with upadacitinib, follow-up data extending beyond 24 weeks revealed that the therapeutic benefits from baseline were maintained for up to 2 years, underscoring the potential for long-term disease control.

Although direct comparisons should be interpreted with caution, as there were differences in study design and an absence of restrictions on concomitant therapies, our results align with those of a single open-label trial in which abrocitinib 100 mg achieved an 80% ≥ 4 PP-NRS reduction at week 12 in a small cohort of patients.[26] Interestingly, the outcomes observed in this analysis appear to exceed the 59% ≥ 4 worst itch-NRS reduction reported for dupilumab (administered in conjunction with topical agents) in pooled LIBERTY-PN PRIME trial (NCT04183335) data, as well as the 56% reduction achieved with nemolizumab at week 16 in the OLYMPIA-1 study (NCT06091254; administered without concomitant topical steroids or calcineurin inhibitors).[9,12] Real-world studies have further characterized the effectiveness of dupilumab in PN, with a large Asian cohort reporting that approximately 84.9% of patients achieved a ≥ 4-point reduction in PP-NRS by week 12.[27] Similar findings were observed in predominantly White populations, with itch from baseline ranging from 70% to 88% at week 16.[28,29] Furthermore, the effectiveness of JAKi was also notable in biologic-experienced patients who had previously received dupilumab or tralokinumab, a group for whom data on switching therapies are particularly scarce. This observation is critical, as it suggests that JAKis may provide an effective alternative for patients who have not achieved optimal outcomes with other biologic agents. The overall low incidence of AEs (characterized primarily by mild and transient issues such as dyslipidaemia and headache) reinforces the favourable safety profile of JAKis in PN, a finding consistent with their established use in other dermatological conditions.[30,31]

Exploratory analyses using the Fine–Gray model in patients treated with upadacitinib did not reveal a statistically significant association between dosage and primary outcomes. Descriptive trends from stratified subanalyses of abrocitinib and baricitinib further suggest that a dose-dependent effect may not always be evident. These findings may be partly attributed to the relatively small sample sizes and potential confounding factors inherent to a retrospective design.

Notably, a subset of patients in our cohort presented with mild AD as a comorbidity. Consistent with the PRIME, PRIME 2 (NCT04202679) and NCT03816891 trials, these cases were classified as PN rather than an AD prurigo-like phenotype.[12,32] This distinction was reasonably based on the predominance of nodular lesions and the limited extent of eczematous lesions, which were unlikely to render PN secondary. Additionally, some patients had an atopic background in the absence of continuing mild AD at baseline. To address these potential confounders, we stratified the analysis by atopic background and active AD status, demonstrating therapeutically relevant clinical benefits of JAKis across all subgroups, regardless of atopic background.[33]

This study is subject to several limitations inherent to its retrospective design, including the potential for selection bias. The absence of a standardized washout period and the lack of restrictions on concomitant therapies may have influenced treatment responses, albeit that such variability reflects real-world prescribing patterns. The relatively limited sample size, particularly in the baricitinib group, precluded a comparative inferential analysis across JAKis. Nonetheless, our study examined one of the largest cohorts reported to date. Finally, our sample consists almost entirely of White patients, potentially limiting the generalizability of our data.

This cohort study suggests that JAKis offer rapid and sustained clinical improvements in PN. Our findings indicate that these agents achieve clinically meaningful outcomes irrespective of atopic background, supporting their use across diverse patient profiles. Further research is warranted to validate these observations and explore their long-term effects.

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Conflicts of interest

C.P. has acted as investigator, speaker, consultant and/or advisory board member for AbbVie, Almirall, Amgen, Eli Lilly, Galderma, La Roche-Posay, LEO Pharma, Novartis, Pfizer, Pierre Fabre and Sanofi. K.P. has received consulting fees for advisory board meetings from AbbVie, Almirall, Galderma, Janssen, LEO Pharma, Lilly, Novartis, Pierre Fabre, Philogen, Sanofi and Sunpharma. C.F. has served on advisory boards and has received honoraria

for lectures from Amgen, Almirall, AbbVie, Boehringer Ingelheim, Incyte, LEO Pharma, Lilly, Pfizer, Novartis and Sanofi. N.G. has served as an advisory board member and received honoraria for lectures for AbbVie, Sanofi, Almirall, Leo-Pharma, Pfizer and Novartis. A. Balato has served as a consultant and has received fees from AbbVie, Almirall, Amgen, Boehringer Ingelheim, BMS, LEO Pharma, Janssen, Eli Lilly, Novartis, Sanofi and UCB. A.N. has served as a consultant and has received fees from AbbVie, Almirall, Amgen, Boehringer Ingelheim, BMS, LEO Pharma, Janssen, Eli Lilly, Novartis, Sanofi, UCB and Pfizer. G.G. has received personal fees from AbbVie, Almirall, Amgen, Boehringer-Ingelheim, Bristol-Myers Squibb, Eli Lilly, LEO Pharma, Merck Serono, Novartis, Pfizer, Pierre Fabre, Samsung Bioepis and Sanofi. M.E. has served as a speaker/consultant for AbbVie, Amgen, Almirall, Eli Lilly, Janssen, Leo Pharma, Novartis, Pfizer, Sanofi and UCB. L.G. has been a consultant and speaker and has participated in advisory boards for AbbVie, Almirall, Eli Lilly, Pfizer, Janssen, Sanofi and UCB Pharma. K.H. reports personal fees from AbbVie, Almirall, Amgen, BMS, LEO Pharma, Novartis, Sanofi and UCB. L.S. reports personal fees from AbbVie, Almirall, Amgen, BMS, Eli Lilly, Incyte, LEO Pharma, Novartis, Pfizer and Sanofi. F.R. has received consulting fees for advisory boards from AbbVie, Almirall, Incyte, Sanofi, Janssen, Bristol Meyers and Boehringer. M.C.F. has served on advisory boards, received honoraria for lectures and/or research grants from AMGEN, Almirall, AbbVie, Boehringer-Ingelheim, BMS, Galderma, Kyowa Kyrin, Incyte, LEO Pharma, Pierre Fabre, UCB, Lilly, Pfizer, Janssen, MSD, Novartis, Sanofi, Regeneron and Sun Pharma. M.N. has received consulting fees from AbbVie, Lilly, LEO Pharma, Almirall and Sanofi; payment/honoraria from AbbVie, Lilly, LEO Pharma, Almirall and Sanofi; has participated in data safety monitoring/advisory boards for Lilly, Sanofi, LEO Pharma, AbbVie and Almirall; and has received support for attending meetings/travel from Lilly. E.N. has served as a consultant and has received fees from AbbVie, Almirall, LEO Pharma, Eli Lilly, Novartis, Sanofi, Gsk, Chiesi, AstraZeneca, Pfizer and Firma. S.C. has served as a consultant and has received fees from AbbVie, Almirall, Boehringer Ingelheim, LEO Pharma, Janssen, Eli Lilly, Novartis, Sanofi, UCB and Pfizer. C.G. has served as a consultant and/or has received fees from Sanofi and AbbVie. The other authors report no conflicts of interest.

Data availability

The data that support the findings of this study will be shared on reasonable request to the corresponding author.

Ethics statement

This study was conducted in accordance with Good Clinical Practice guidelines, the provisions of the Declaration of Helsinki and all applicable regulations. This study was approved by the Ethics Committee of the Department of Medicine at the University of Turin (with ethics committee approval number: 0006349; Infiammatorie-dermo26). The institutional review board approved the study protocol and STROBE reporting guidelines for cohort studies were followed.

Patient consent

Written informed consent was obtained from the patient for the publication of the clinical photograph and any potentially identifiable data included in this article.

Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's website.

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