ORIGINAL REPORT

Efficacy and Safety of Tofacitinib in Palmoplantar Pustulosis: A Retrospective Study

Shuang LYU[1,2#], Xiaotong XUE[1,2#], Na WANG[1,2], Hong LIU[1,2*] and Furen ZHANG[1,2]
[1]*Dermatology Hospital of Shandong First Medical University, Jinan, Shandong Province, and* [2]*Shandong Provincial Institute of Dermatology and Venereology, Shandong Academy of Medical Sciences, Jinan, Shandong Province, China*
[#]*Shuang Lyu and Xiaotong Xue contributed equally to this manuscript.*

Palmoplantar pustulosis (PPP) remains a therapeutic challenge with limited options, and recurrence is a major issue for traditional systemic therapies and biologics. Data on long-term efficacy, safety, and relapse of Janus kinase inhibitors in PPP are limited. Drug efficacy, safety, and recurrence was retrospectively evaluated in 29 PPP patients treated with tofacitinib from January 2022 to June 2024. Disease severity and efficacy were assessed using the Palmoplantar Pustular Psoriasis Area and Severity Index (PPPASI) and Palmoplantar Pustular Psoriasis Physician's Global Assessment (PPP PGA) at baseline, week 4, and week 12, with a minimum 6-month follow-up. PPP-related haematological inflammatory indicators, including plateletcrit (PCT), pan-immune-inflammation value (PIV), neutrophil/lymphocyte ratio (NLR), and monocyte/lymphocyte ratio (MLR), were also evaluated. By week 12, mean PPPASI score significantly decreased from 18.62 to 6.17 ($p < 0.001$), with 72.41% achieving PPPASI-50 and 62.1% achieving PPP PGA ≤ 1. Mild adverse events (gastric discomfort) occurred in 6.9% of patients. During a mean 12.2-month follow-up, 27.6% relapsed, while 34.5% maintained clearance without medication. Disease severity-related haematologic indicators, PCT, and PIV improved significantly. Tofacitinib demonstrates significant efficacy and a favourable safety profile in PPP, warranting consideration as a therapeutic option, though larger prospective studies are needed to confirm long-term outcomes.

Key words: palmoplantar pustulosis; Janus kinase inhibitors; retrospective study; efficacy; safety; inflammatory markers.

Submitted Jun 5, 2025. Accepted after revision Oct 28, 2025

Published Nov 12, 2025. DOI: 10.2340/actadv.v105.44083

Acta Derm Venereol 2025; 105: adv44083.

Corr: Hong Liu, MD, PhD, Dermatology Hospital of Shandong First Medical University; Shandong Provincial Institute of Dermatology and Venereology, Shandong Academy of Medical Sciences. Jingshi Lu, 27397, Jinan, Shandong Province, China. E-mail: hongyue2519@hotmail.com

SIGNIFICANCE

Palmoplantar pustulosis is a chronic skin disease causing painful palm/sole pustules, often resistant to treatment and prone to relapse. This study shows that tofacitinib – an oral Janus kinase inhibitor – significantly improves symptoms with low side effects: 72.41 % of patients achieved a 50% lesion reduction by week 12. Averaging 12 months of follow-up, one-third maintained clearance without medication. The findings provide real-world evidence to support Janus kinase inhibitors as a safe, accessible oral option for palmoplantar pustulosis, enhancing treatment choices and guiding future research to improve patient outcomes.

Palmoplantar pustulosis (PPP), a chronic and recurrent cutaneous disorder manifesting on the palms and/or soles, is characterized by the occurrence of sterile pustules against an erythematous and scaly backdrop with an unknown aetiology (1). The prevalence of PPP ranges from 0.01% to 0.05%, and a prevalence of 0.12% has been reported in the Japanese population (2, 3).The mean age of onset is 40–58 years (1). Middle-aged women are considered to be at relatively higher risk (3, 4). Moreover, risk factors such as smoking, infections, and metal sensitivities have been associated with PPP (2, 5, 6).

The treatment of PPP poses a challenge for clinicians. The treatments for PPP encompass systemic therapies such as acitretin, cyclosporine, and methotrexate, alongside topical treatments and phototherapy (1, 7–11). Recently, biologic agents targeting interleukin (IL-1, IL-8, IL-17, IL-23, and IL-36) and tumour necrosis factor-α (TNF-α) have emerged as new options (12–20). Despite these therapies, the management of PPP remains challenging, due to interindividual variability in treatment response, marked differences in efficacy among agents, and the persistent problem of recurrence – especially in refractory cases lacking consistent effective therapies.

Janus kinase inhibitors (JAKi) are small molecule drugs that inhibit the JAK-STAT signalling pathway, demonstrating efficacy in various inflammatory skin disorders, including psoriasis and atopic dermatitis (21). To date, evidence supporting JAKi use in PPP mainly comes from case reports and small case series (22–28). However, these studies often have inconsistent follow-up durations and limited detailed descriptions of relapse situations.

This study aims to retrospectively evaluate the efficacy, safety, and recurrence outcomes of the JAKi tofacitinib in a cohort of 29 PPP patients, providing insights for clinical management and guiding future research.

METHODS

Patients

A retrospective study was conducted on 29 PPP patients treated with tofacitinib at the Hospital for Skin Diseases, Shandong First Medical University from January 2022 to June 2024. The study protocol was subject to review and received approval from the ethical committee of the Dermatology Hospital of Shandong First Medical University (approval number: 20240718KYKTKS002).

The inclusion criteria were: (*i*) diagnosis of PPP confirmed by 2 or more dermatology specialists or pathological diagnosis, and the disease is in an active state; (*ii*) insufficient response to at least 1 systemic treatment; (*iii*) treatment duration of more than 12 weeks; (*iv*) follow-up for at least 6 months.

The exclusion criteria were: (*i*) drug-induced PPP (e.g., by TNF inhibitors); (*ii*) active infections or infectious diseases (e.g., mycobacterium tuberculosis, hepatitis B, AIDS, herpes zoster); (*iii*) severe organ dysfunctions (cardiac, hepatic, renal, etc.); (*iv*) haematological disorders or tumours.

The demographic data (including age, gender, smoking status, alcohol abuse, family history, body mass index (BMI), and suspicious substance exposure) and disease characteristics (such as lesion site, disease duration, disease severity scores), and haematological inflammatory markers were recorded at baseline (week 0), week 4, and week 12. Treatment included tofacitinib ($n=29$) with optional topical corticosteroids.

Efficacy and follow-up

The Palmoplantar Pustular Psoriasis Area and Severity Index (PPPASI) and Palmoplantar Pustular Psoriasis Physician's Global Assessment (PPP PGA) were used to assess disease severity and treatment efficacy. The primary efficacy endpoint was the change in the total score of the PPPASI from baseline to week 12. The secondary efficacy endpoints assessed at week 12 included PPPASI-50/75/90 responses (the proportion of patients with ≥50%, ≥75%, and ≥90% reduction in PPPASI from baseline), the change in PPP PGA score from baseline, and the proportion of PPP PGA score of 0 or 1 (these patients were classified as responders). We also evaluated PPP-related haematological inflammatory indicators, including plateletcrit (PCT), pan-immune-inflammation value (PIV), neutrophil/lymphocyte ratio (NLR), and monocyte/lymphocyte ratio (MLR) (29, 30). Additionally, the correlation between PPPASI-50 and patient demographics was examined.

Patients were followed up for more than 6 months after 12 weeks of treatment, and the outcomes were defined as follows: relapse (patients lost PPPASI-50 or had PGA ≥3); clearance (no recurrence after drug withdrawal); lower dosage maintenance (maintaining clearance with a lower dose); standard-dose maintenance (maintaining clearance with a normal dose). All subjective and objective adverse events were recorded. Coagulation and D-dimer testing were performed in patients aged >60 years.

Statistics

Statistical analyses were performed in SPSS 24 (IBM Corp, Armonk, NY, USA). For haematological indicators with missing values (<10%), multiple interpolation was used for compensation. Repeated measures ANOVA was utilized to analyse the data at 4 and 12 weeks of treatment vs baseline. If the data did not meet normal distribution, Friedman's test was applied. When significant differences among the 3 groups emerged, two-by-two comparisons were made via two-way analysis of variance (by rank) for related samples after Bonferroni correction. To evaluate the association between PPPASI-50 and risk factors related to PPP disease, the related-samples Wilcoxon signed-rank test was applied to compare the 2 factors.

RESULTS

Demographics and disposition of the patients

Table I summarizes the clinical and demographic data. The mean age was 48.2 years (range 24–70 years), with 69.0% (20/29) being female. The mean duration of tofacitinib treatment was 6.9±4.9 months. Among the included patients, 17.2% (5/29) were tobacco users, 10.3% (3/29) were chronic alcohol users, and 13.8% (4/29) had a family history of similar conditions. The mean BMI was 24.25±2.59 kg/m², and the mean disease duration was 2.95 years (range 0.13–20 years). Nail and joint involvement were observed in 24.1% (7/29) and 13.8% (4/29) of patients, respectively; 17.2% (5/29) had

Table I. Demographic and baseline characteristics

Age, median (range), years	48.2 (24–70)
Female, *n* (%)	20 (69.0)
Weight, mean (SD), kg	68.4 (10.31)
Body mass index, (mean±SD), kg/m²	24.25±2.59
Disease duration: mean (range), years	2.95 (0.13–20)
Tobacco use, *n* (%)	5 (17.2)
Chronic alcohol use, *n* (%)	3 (10.3)
Family history, *n* (%)	4 (13.8)
Suspicious substance exposure, *n* (%)[a]	11 (37.93)
Nail involvement, *n* (%)	7 (24.1)
Joint involvement, *n* (%)	4 (13.8)
Concomitant plaque psoriasis, *n* (%)	5 (17.2)
Palmoplantar involvement, *n* (%)	
Both palms and soles	22 (75.86)
Both palms	4 (13.79)
Both soles	3 (10.34)
Palmoplantar Pustulosis Area and Severity Index, mean (SD)	18.62 (6.36)
Palmoplantar Pustulosis Physician Global Assessment score, *n* (%)	
2 Mild	7 (24.1)
3 Moderate	19 (65.5)
4 Severe	3 (10.3)

[a]Suspected substance exposure: dentures, intrauterine device (IUD), special occupations (hairdressing, decorating, printing), orthopaedic or cardiac device implants, etc.
SD: standard deviation.

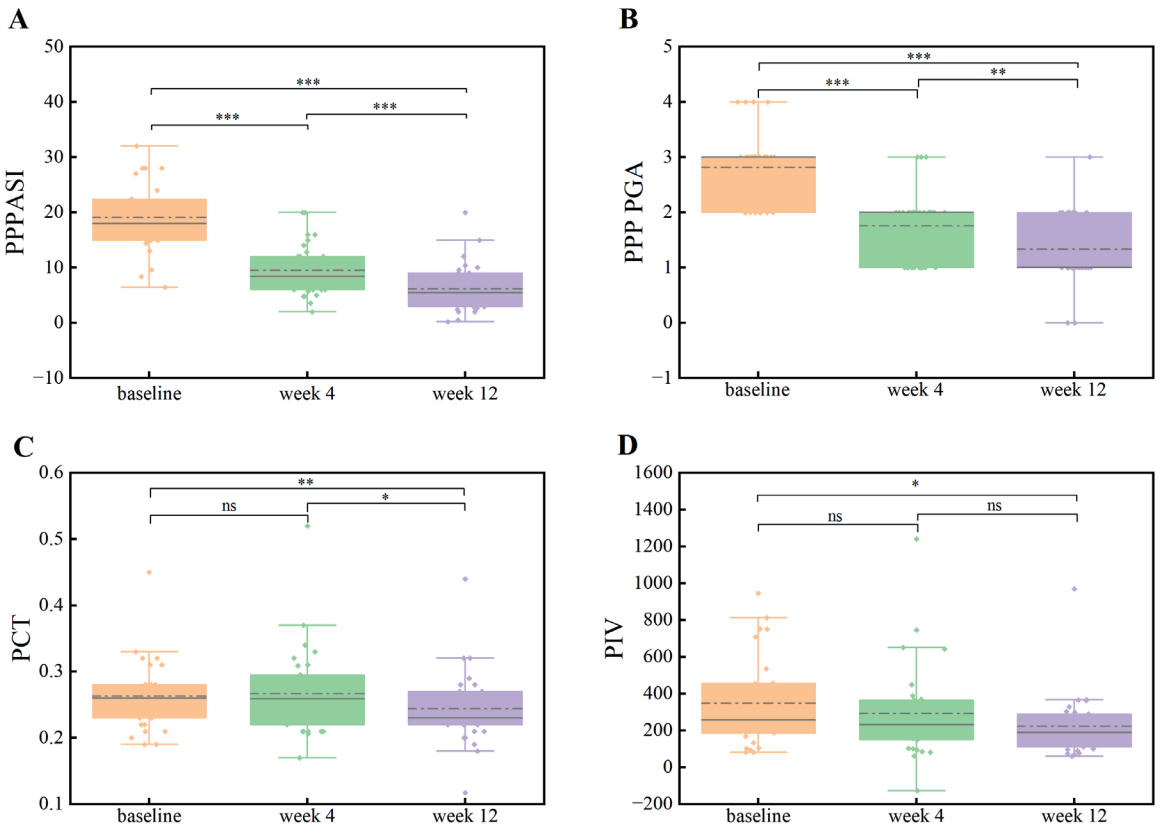

Fig. 1. Changes in Palmoplantar Pustulosis Area and Severity Index (PPPASI) score. (A) Palmoplantar Pustulosis Physician Global Assessment (PPP PGA) score, (B) plateletcrit (PCT), and (C) pan-immune-inflammation value (PIV), (D) in each patient before and after Janus kinase inhibitor treatment (Friedman test). *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$.

concomitant psoriasis. At baseline, the mean PPPASI score was 18.62±6.36, and the PPP PGA scores were distributed as follows: 24.1% (7/29) had mild disease (score 2), 65.5% (19/29) had moderate disease (score 3), and 10.3% (3/29) had severe disease (score 4). Most patients (75.86%, 22/29) had lesions on both palms and soles.

Treatment efficacy assessment

After 12 weeks, both PPPASI and PPP PGA scores improved significantly (**Fig. 1**A, B). The mean (SD) PPPASI score decreased from (18.62±6.36) to (6.17±4.42) (**Table II**), and the difference was statistically significant ($p < 0.001$). At 4 and 12 weeks, the percentage of patients achieving PPPASI-50 was 55.17% and 72.41%, respectively (**Fig. 2**A). At week 12, PPPASI-75 and PPPASI-90 responses were observed in 41.38% (12/29) and 10.3% (3/29), respectively (Fig. 2A); 69.0% (20/29) had a PGA score ≤1, only 1 patient (3.45%) had a PPP PGA score ≥3 (Table II). PCT and PIV, as disease severity-related haematological indicators, decreased significantly after 12 weeks of treatment (Fig. 1C, D). NLR and MLR changes were not statistically significant. No significant association was detected between PPPASI-50 achievement and gender, BMI ≥24.0 kg/m², age ≥60 years, smoking, alcohol use, family history, or exposure to suspicious substances such as dentures.

Table II. Treatment and follow-up outcomes

Patients with PPP PGA scores ≤1, *n* (%) (at week 12)	18 (62.1)
Patients with PPP PGA scores ≥3, *n* (%) (at week 12)	1 (3.45)
PPPASI: mean (SD) (at week 12)	6.17 (4.42)
Duration of follow-up (*n* =29): mean (range), month	12.2 (6–28)
Follow-up outcome, *n* (%)	
Relapse	8 (27.6)
Clearance	10 (34.5)
Lower dosage maintenance	8 (27.6)
Standard-dose maintenance	3 (10.3)
Adverse reactions during treatment, *n* (%)	
Gastric discomfort	2 (6.9%)

PPPASI: Palmoplantar Pustulosis Area and Severity Index; PPP PGA: Palmoplantar Pustulosis Physician Global Assessment; SD: standard deviation.

Safety assessments

Adverse events were mild and included 2 cases (6.9%) of gastric discomfort (see Table II).

Follow-up outcomes

During a mean follow-up of 12.2 months (ranging from 6 to 28), 27.6% (8/29) of patients experienced relapse. Among the rest, 34.5% (10/29) remained clear without medication; 27.6% (8/29) required lower dosage maintenance, and 10.3% (3/29) needed continued standard-dose maintenance (see Table II).

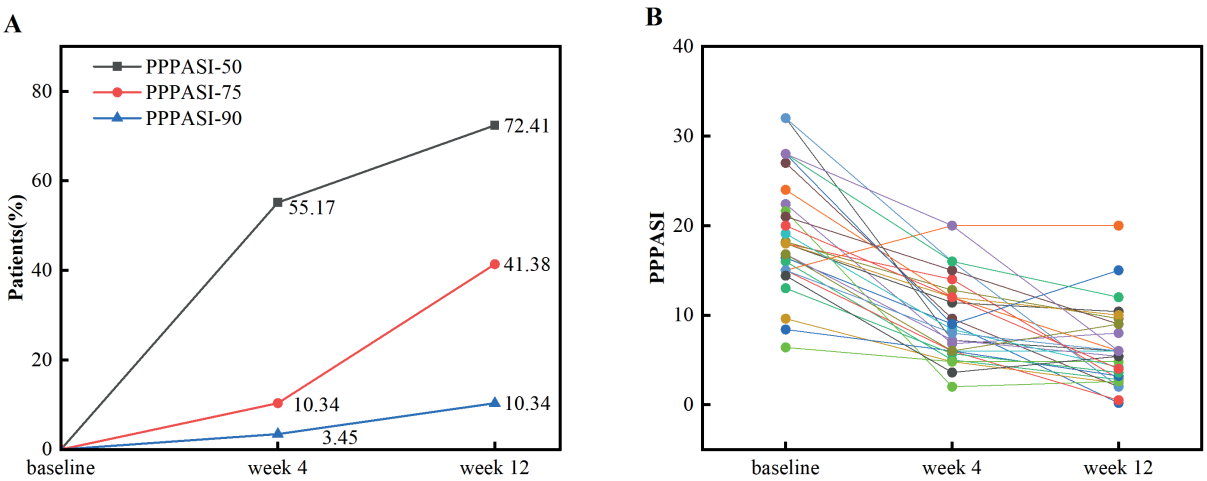

Fig. 2. Progression of Palmoplantar Pustulosis Area and Severity Index (PPPASI) scores during Janus kinase inhibitors treatment. (A) Percentage of patients with 50%, 75%, and 90% reductions in PPPASI scores (PPPASI-50, PPPASI-75, and PPPASI-90, respectively) at baseline, week 4, and week 12. (B) Change in PPPASI score between baseline, week 4, and week 12 among all patients.

DISCUSSION

This study demonstrates that the JAKi tofacitinib provides clinically meaningful improvements in PPP, evidenced by significant improvements in PPPASI and PPP PGA scores, and haematological inflammatory parameters were observed by week 12. The relapse rate of 27.6% and 34.5% drug-free remission after discontinuation highlight both the potential and limitations of JAKi treatment.

The treatment of PPP remains challenging, with refractory cases and recurrences despite various therapeutic approaches. Previous studies have reported PPPASI-50 and PPPASI-75 responses rates ranging from 14.5–78.3% and 8.1–43.5%, respectively (15, 18, 20, 31, 32). Among available treatments, guselkumab and apremilast appear relatively more effective. Specifically, guselkumab achieved PPPASI-50 in 60% of patients at week 16 (19), while apremilast reached 78.3% and 43.5% for PPPASI-50 and PPPASI-75 responses at week 16, respectively (33). In our study, 72.41% of patients achieved PPPASI-50 and 41.38% achieved PPPASI-75 at week 12, indicating faster onset and efficacy comparable to existing effective treatments. The JAK-STAT pathway, which mediates proinflammatory cytokine signalling in PPP pathogenesis (1), has prompted increasing reports of JAKi in PPP. Case studies further support JAKi efficacy in refractory PPP, including responses in patients unresponsive to biologics or with drug-induced PPP (24, 25, 28). Tofacitinib and upadacitinib have demonstrated reductions in both PPPASI and DLQI, with some achieving complete or near-complete clearance (22, 23, 27). Notably, no statistically significant associations were observed between PPPASI-50 responses and demographic or clinical variables, including sex, age ≥60 years, BMI ≥24.0 kg/m², alcohol consumption, exposure to suspicious substances (e.g., dentures, metal prostheses, printed materials), or family history. These findings suggest that the efficacy of tofacitinib in PPP may not be affected by the above-mentioned factors. However, large-scale studies are needed to further explore the impact of these variables on treatment outcomes.

Relapse rates of PPP have rarely been evaluated in most previous studies of therapeutic agents. For JAKi in PPP, some cases did not report relapse data, while others, with follow-up durations ranging from 12 weeks to 1 year, lacked detailed relapse information (22–28). During a mean follow-up period of 12.2 months, 27.6% of patients relapsed, indicating a need for improved strategies to sustain disease control beyond initial JAKi efficacy. Some 34.5% of patients maintained remission after discontinuation, suggesting the potential for long-term remission. In addition, 27.6% of patients required a reduced maintenance dose and 10.3% continued on the standard dose to preserve therapeutic response. These findings highlight the importance of developing individualized relapse prevention and maintenance protocols in future research.

Mechanistically, JAKi inhibit the signalling activity of key cytokines involved in PPP pathogenesis, including IL-6, IL-17, and IL-36 (1). After 12 weeks of tofacitinib treatment, PCT and PIV were significantly reduced, suggesting that tofacitinib may inhibit platelets and inflammatory cells. The elevated PCT in PPP patients may result from the upregulation of IL-6, a thrombopoiesis-promoting cytokine (29). Thus, the reduction in PCT and PIV after tofacitinib treatment might be due to the inhibition of IL-6 upregulation. However, the exact relationship between these indices and treatment requires further investigation.

Despite their promise, JAKi have side effects in treatment, such as infections (including reactivation of varicella zoster virus), altered lipid metabolism, occasional transaminase, creatinine elevations and even tumour risk (34). In our cohort, no serious adverse events were

observed, but vigilant monitoring remains essential. JAKi offer advantages over biologics in terms of cost and oral administration, potentially improving adherence.

Limitations

Limitations of our study include small sample size, lack of a control group, and retrospective design. Additionally, drug-induced PPP – with low incidence (≈5% for anti-TNF-induced cases) and substantial clinical heterogeneity – was excluded in our study to avoid confounding the results (35). However, given that JAK inhibitors can provide unique therapeutic benefits by simultaneously targeting multiple cytokine pathways (e.g., IL-6, IFN-γ) (35), it is reasonable to hypothesize that JAK inhibitors may also exhibit significant efficacy in drug-induced PPP.

Conclusion

In conclusion, tofacitinib exhibited a significant improvement in PPPASI and PPP PGA scores as well as haematological markers such as PIV and PCT, with a favourable safety profile in PPP patients. This study provides preliminary evidence of tofacitinib's efficacy and safety in treating PPP, highlighting its potential as a therapeutic option. However, the small sample size and retrospective design limit the generalizability of the findings. Larger, prospective, controlled studies are warranted to further validate these findings and assess long-term outcomes.

ACKNOWLEDGEMENTS

The authors would like to thank all the patients who participated and the doctors who provided cases.

Funding information: This work was supported by the Joint Innovation Team for Clinical & Basic Research (202410) and Shandong Provincial Clinical Research Center for Dermatovenerology.

Data availability statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics declarations: Reviewed and approved by the Review Committee of Hospital for Skin Diseases, Shandong First Medical University; approval number: 20240718KYKTKS002.

The authors have no conflicts of interest to declare.

REFERENCES

1. Misiak-Galazka M, Zozula J, Rudnicka L. Palmoplantar pustulosis: recent advances in etiopathogenesis and emerging treatments. Am J Clin Dermatol 2020; 21: 355–370. https://doi.org/10.1007/s40257-020-00503-5
2. Mrowietz U, van de Kerkhof PC. Management of palmoplantar pustulosis: do we need to change? Br J Dermatol 2011; 164: 942–946. https://doi.org/10.1111/j.1365-2133.2011.10233.x
3. Kubota K, Kamijima Y, Sato T, Ooba N, Koide D, Iizuka H, et al. Epidemiology of psoriasis and palmoplantar pustulosis: a nationwide study using the Japanese national claims database. BMJ Open 2015; 5: e006450. https://doi.org/10.1136/bmjopen-2014-006450
4. Michaëlsson G, Kristjánsson G, Pihl Lundin I, Hagforsen E. Palmoplantar pustulosis and gluten sensitivity: a study of serum antibodies against gliadin and tissue transglutaminase, the duodenal mucosa and effects of gluten-free diet. Br J Dermatol 2007; 156: 659–666. https://doi.org/10.1111/j.1365-2133.2006.07725.x
5. Akiyama T, Seishima M, Watanabe H, Nakatani A, Mori S, Kitajima Y. The relationships of onset and exacerbation of pustulosis palmaris et plantaris to smoking and focal infections. J Dermatol 1995; 22: 930–934. https://doi.org/10.1111/j.1346-8138.1995.tb03948.x
6. Yamamoto T. Triggering role of focal infection in the induction of extra-palmoplantar lesions and pustulotic arthro-osteitis associated with palmoplantar pustulosis. Adv Otorhinolaryngol 2011; 72: 89–92. https://doi.org/10.1159/000324620
7. Muro M, Kawakami H, Matsumoto Y, Abe N, Tsuboi R, Okubo Y. Topical combination therapy with vitamin D3 and corticosteroid ointment for palmoplantar pustulosis: a prospective, randomized, left-right comparison study. J Dermatolog Treat 2016; 27: 51–53. https://doi.org/10.3109/09546634.2015.1052036
8. Umezawa Y, Nakagawa H, Tamaki K. Phase III clinical study of maxacalcitol ointment in patients with palmoplantar pustulosis: a randomized, double-blind, placebo-controlled trial. J Dermatol 2016; 43: 288–293. https://doi.org/10.1111/1346-8138.13064
9. Engin B, Oguz O. Evaluation of time-dependent response to psoralen plus UVA (PUVA) treatment with topical 8-methoxypsoralen (8-MOP) gel in palmoplantar dermatoses. Int J Dermatol 2005; 44: 337–339. https://doi.org/10.1111/j.1365-4632.2004.02153.x
10. Su LN, Ren J, Cheng SM, Liu JL, Ding YF, Zhu NW. UVA1 vs. narrowband UVB phototherapy in the treatment of palmoplantar pustulosis: a pilot randomized controlled study. Lasers Med Sci 2017; 32: 1819–1823. https://doi.org/10.1007/s10103-017-2280-0
11. Sevrain M, Richard MA, Barnetche T, Rouzaud M, Villani AP, Paul C, et al. Treatment for palmoplantar pustular psoriasis: systematic literature review, evidence-based recommendations and expert opinion. J Eur Acad Dermatol Venereol 2014; 28: 13–16. https://doi.org/10.1111/jdv.12561
12. Skov L, Beurskens FJ, Zachariae CO, Reitamo S, Teeling J, Satijn D, et al. IL-8 as antibody therapeutic target in inflammatory diseases: reduction of clinical activity in palmoplantar pustulosis. J Immunol 2008; 181: 669–679. https://doi.org/10.4049/jimmunol.181.1.669
13. Tauber M, Viguier M, Alimova E, Petit A, Lioté F, Smahi A, et al. Partial clinical response to anakinra in severe palmoplantar pustular psoriasis. Br J Dermatol 2014; 171: 646–649. https://doi.org/10.1111/bjd.13012
14. Cornelius V, Wilson R, Cro S, Barker J, Burden D, Griffiths CEM, et al. A small population, randomised, placebo-controlled trial to determine the efficacy of anakinra in the treatment of pustular psoriasis: study protocol for the APRICOT trial. Trials 2018; 19: 465. https://doi.org/10.1186/s13063-018-2841-y
15. Mrowietz U, Bachelez H, Burden AD, Rissler M, Sieder C, Orsenigo R, et al. Secukinumab for moderate-to-severe palmoplantar pustular psoriasis: results of the 2PRECISE study. J Am Acad Dermatol 2019; 80: 1344–1352. https://doi.org/10.1016/j.jaad.2019.01.066
16. Pinter A, Wilsmann-Theis D, Peitsch WK, Mössner R. Interleukin-17 receptor A blockade with brodalumab in palmoplantar pustular psoriasis: report on four cases. J Dermatol 2019; 46: 426–430. https://doi.org/10.1111/1346-8138.14815
17. Bissonnette R, Maari C, Tsianakas A, Reid D, McCutchan S, Baumgartner S, et al. A randomized, double-blind, placebo-controlled, phase 2a study to evaluate the efficacy and safety of RIST4721 in subjects with palmoplantar pustulosis. Dermatol Ther (Heidelb) 2021; 11: 2179–2193. https://doi.org/10.1007/s13555-021-00632-7
18. Okubo Y, Kobayashi S, Murakami M, Sano S, Kikuta N, Ouchi Y, et al. Efficacy and safety of brodalumab, an anti-

interleukin-17 receptor A monoclonal antibody, for palmoplantar pustulosis: 16-week results of a randomized clinical trial. Am J Clin Dermatol 2024; 25: 837–847. https://doi.org/10.1007/s40257-024-00876-x
19. Terui T, Kobayashi S, Okubo Y, Murakami M, Hirose K, Kubo H. Efficacy and safety of guselkumab, an anti-interleukin 23 monoclonal antibody, for palmoplantar pustulosis: a randomized clinical trial. JAMA Dermatol 2018; 154: 309–316. https://doi.org/10.1001/jamadermatol.2017.5937
20. Bissonnette R, Poulin Y, Bolduc C, Maari C, Provost N, Syrotuik J, et al. Etanercept in the treatment of palmoplantar pustulosis. J Drugs Dermatol 2008; 7: 940–946.
21. Chapman S, Kwa M, Gold LS, Lim HW. Janus kinase inhibitors in dermatology: Part I. A comprehensive review. J Am Acad Dermatol 2022; 86: 406–413. https://doi.org/10.1016/j.jaad.2021.07.002
22. Xu Q, Wang X, Yang A, Wei G. Refractory palmoplantar pustulosis successfully treated with JAK inhibitor tofacitinib: a case series. Infect Drug Resist 2023; 16: 5165–5172. https://doi.org/10.2147/idr.S421299
23. Li C, Li Z, Cao Y, Li L, Li F, Li Y, et al. Tofacitinib for the treatment of nail lesions and palmoplantar pustulosis in synovitis, acne, pustulosis, hyperostosis, and osteitis syndrome. JAMA Dermatol 2021; 157: 74–78. https://doi.org/10.1001/jamadermatol.2020.3095
24. Koga T, Sato T, Umeda M, Fukui S, Horai Y, Kawashiri SY, et al. Successful treatment of palmoplantar pustulosis with rheumatoid arthritis, with tofacitinib: impact of this JAK inhibitor on T-cell differentiation. Clin Immunol 2016; 173: 147–148. https://doi.org/10.1016/j.clim.2016.10.003
25. Fan Y, Yin R. Janus kinase inhibitor tofacitinib successfully treated palmoplantar pustulosis after switching from secukinumab: a case report and review of the literature. Int Immunopharmacol 2024; 139: 112762. https://doi.org/10.1016/j.intimp.2024.112762
26. Haynes D, Topham C, Hagstrom E, Greiling T. Tofacitinib for the treatment of recalcitrant palmoplantar pustulosis: a case report. Australas J Dermatol 2020; 61: e108–e110. https://doi.org/10.1111/ajd.13117
27. Du N, Yang J, Zhang Y, Lv X, Cao L, Min W. Successful treatment of refractory palmoplantar pustulosis by upadacitinib: report of 28 patients. Front Med (Lausanne) 2024; 11: 1476793. https://doi.org/10.3389/fmed.2024.1476793
28. Wang YA, Rosenbach M. Successful treatment of refractory tumor necrosis factor inhibitor-induced palmoplantar pustulosis with tofacitinib: report of case. JAAD Case Rep 2020; 6: 115–118. https://doi.org/10.1016/j.jdcr.2019.12.006
29. Ning X, Wu C, Song B, Wang HM, Jin HZ. Evaluation of hematological inflammatory parameters in patients with palmoplantar pustulosis. Int J Dermatol 2024; 10.1111/ijd.17230. https://doi.org/10.1111/ijd.17230
30. Watanabe T, Yamaguchi Y. Author reply to "regarding "retrospective study of the clinical significance of the neutrophil-to-lymphocyte ratio in 79 patients with palmoplantar pustulosis"". J Dermatol 2025; 52: e187–e188. https://doi.org/10.1111/1346-8138.17440
31. Terui T, Okubo Y, Kobayashi S, Sano S, Morita A, Imafuku S, et al. Efficacy and safety of apremilast for the treatment of Japanese patients with palmoplantar pustulosis: results from a phase 2, randomized, placebo-controlled study. Am J Clin Dermatol 2023; 24: 837–847. https://doi.org/10.1007/s40257-023-00788-2
32. Wilsmann-Theis D, Kromer C, Gerdes S, Linker C, Magnolo N, Sabat R, et al. A multicentre open-label study of apremilast in palmoplantar pustulosis (APLANTUS). J Eur Acad Dermatol Venereol 2021; 35: 2045–2050. https://doi.org/10.1111/jdv.17441
33. Terui T, Okubo Y, Kobayashi S, Sano S, Morita A, Imafuku S, et al. Author correction: Efficacy and safety of apremilast for the treatment of Japanese patients with palmoplantar pustulosis: results from a phase 2, randomized, placebo-controlled study. Am J Clin Dermatol 2024; 25: 165–167. https://doi.org/10.1007/s40257-023-00825-0
34. Banerjee S, Biehl A, Gadina M, Hasni S, Schwartz DM. JAK-STAT signaling as a target for inflammatory and autoimmune diseases: current and future prospects. Drugs 2017; 77: 521–546. https://doi.org/10.1007/s40265-017-0701-9
35. Maronese CA, Valenti M, Moltrasio C, Romagnuolo M, Ferrucci SM, Gilliet M, et al. Paradoxical psoriasis: an updated review of clinical features, pathogenesis, and treatment options. J Invest Dermatol 2024; 144: 2364–2376. https://doi.org/10.1016/j.jid.2024.05.015