ORIGINAL REPORT

MJS PUBLISHING

Cutaneous Lymphoma Associated with JAK Inhibitors: A Pharmacovigilance Analysis of the FAERS Database and Literature Review

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The increasing use of JAK inhibitors in clinical practice is raising concerns regarding the potential risk of cutaneous lymphoma. This study aimed to conduct a comprehensive search for cases of cutaneous lymphoma associated with JAK inhibitors in the Food and Drug Administration's Adverse Event Reporting System. The clinical characteristics of cases from January 2004 to September 2023 were retrieved from the FAERS database. Disproportionality and Bayesian analyses were performed to detect signals for cutaneous lymphoma associated with JAK inhibitors. In total, 24 cases of cutaneous lymphoma were identified associated with JAK inhibitors, including tofacitinib, ruxolitinib, baricitinib, upadacitinib, and abrocitinib. The majority of patients (64%) were aged 60 or older, with no significant difference in incidence between genders. The average onset time was 8.64 months. One patient with ruxolitinib experienced a fatal outcome, and 1 patient with tofacitinib had a life-threatening event. Cutaneous lymphoma associated with baricitinib has the highest reporting odds ratio (23.91, 95% confidence interval 10.71–53.4), proportional reporting ratio (23.88, $\chi^2 = 103.67$), information component (4.57, IC025 = 2.05), and empirical Bayes geometric mean (23.73, EBGM05 = 12.12). The occurrence of cutaneous lymphoma associated with JAK inhibitors highlights the importance of pharmacovigilance studies to deepen our understanding of both the medications and associated conditions.

Key words: cutaneous lymphoma; JAK inhibitors; FAERS; pharmacovigilance studies.

Submitted Aug 4, 2025. Accepted after revision Dec 10, 2025

Published Jan 8, 2026. DOI: 10.2340/actadv.v106.44546

Acta Derm Venereol 2026; 106: adv44546.

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SIGNIFICANCE

We searched the FAERS database for cases of lymphoma after application of JAK inhibitors and analysed the clinical features of these cases. The possibility of lymphoma induced by JAK inhibitors was explored through literature review and summarization. The importance of pharmacovigilance studies and the need to be alert to JAK inhibitor-related adverse effects in clinical applications were also emphasized.

Cutaneous lymphomas, comprising cutaneous T-cell lymphoma (CTCL) and B-cell lymphoma (CBCL), are heterogeneous malignancies originating in the skin (1). The clinical presentations of cutaneous lymphoma are diverse, including skin patches, plaques, tumours, or systemic involvement, which can affect life expectancy (2). Mycosis fungoides (MF), the most common CTCL, typically evolves through patch, plaque, and tumour stages in sun-protected areas. Sézary syndrome (SS), characterized by erythroderma, lymphadenopathy, and circulating malignant T cells, follows an aggressive course (3). The development of MF and SS is associated with genetic, microenvironmental, and immunological factors. The pathogenesis of MF/SS involves mutations in genes including p16 and p53, as well as loss of Janus kinase-signal transducers and activators of transcription (JAK-STAT) pathway inhibitors in early MF (4, 5). Subcutaneous panniculitis-like T-cell lymphoma (SPTCL) presents as subcutaneous nodules and plaques, with HAVCR2 mutations in 51% of cases characterized by an enriched IL6-JAK-STAT3 signalling pathway (6). Lymphomatoid papulosis (LyP), a CD30+ lymphoproliferative disorder, manifests as solitary or localized tumours or nodules (2), with a favourable prognosis (5-year survival > 50%) (1). LyP may be associated with mutations in the TGF-beta type 1 receptor gene (7), and viral infections like HTLV-1 (8). Treatment strategies for different types of cutaneous lymphomas vary, including local therapies such as topical corticosteroids, phototherapy, radiation therapy, and surgery, as well as systemic treatments like chemotherapy, biological response modifiers such as IFN-α, targeted therapy, and haematopoietic stem cell transplantation (9).

The JAK/STAT signalling pathway regulates proliferation, differentiation, and apoptosis via 4 subtypes of JAK: JAK1, JAK2, JAK3, and TYK2. Cytokine-induced JAK phosphorylation activates STAT proteins, driving gene transcription (10). JAK inhibitors are now widely

used for inflammatory diseases and myeloproliferative neoplasms (11).

With the widespread use of JAK inhibitors, associated cutaneous lymphoma cases have emerged. Therefore, we analysed the United States Food and Drug Administration Adverse Event Reporting System (FAERS) and literature to systematically evaluate this potential association.

METHODS

Data source

FAERS, the FDA's post-market surveillance database for drugs and therapeutic biologics, contains information on all adverse events and medication errors collected by the FDA. It holds significant value for pharmacovigilance research. FAERS comprises 7 data tables: Patient Demographics and Administrative Information (DEMO), Drug Information (DRUG), Adverse Events (REAC), Patient Outcomes (OUTC), Report Sources (RPSR), Therapy Start and End Dates (THER), and Use/Diagnostic Indications (INDI).

We conducted a retrospective review of data from January 2004 to September 2023 and obtained 20,352,838 reports from the DEMO data table. After removing duplicates, we included 17,035,801 reports for analysis (**Fig. 1**).

Data searching

Adverse events in the REAC table are coded using the Medical Dictionary for Regulatory Activities (MedDRA 26.21), with cutaneous lymphoma (code: 10079945), cutaneous T-cell lymphoma (code: 10079946), and cutaneous B-cell lymphoma (code: 10079944) included. The drugs retrieved for analysis include tofacitinib, ruxolitinib, baricitinib, upadacitinib, and abrocitinib.

Descriptive analysis

Our study provides a descriptive analysis of the characteristics of patients who developed cutaneous lymphoma following the use of JAK inhibitors. These characteristics include age, gender, reporter, reporting region, indications, and outcomes.

Statistical analysis

The onset times of the JAK inhibitors-associated cutaneous lymphoma among different kinds of drugs were compared using a Kruskal–Wallis test and Dunn's multiple comparisons test, with the threshold for statistical significance set at $p<0.05$ and a 95% confidence interval (CI). Statistical analysis was conducted using GraphPad Prism version 10 (GraphPad Software, San Diego, CA, USA).

To explore adverse reaction signals, we utilized the FAERS database and employed disproportionality analysis and Bayesian analysis methodologies to identify potential signals for cutaneous lymphoma adverse events associated with JAK inhibitors. We employed the following methods for signal detection: reporting odds ratio (ROR), proportional reporting ratio (PRR), Bayesian confidence propagation neural network, and multi-item gamma Poisson shrinker. For ROR analysis, if the lower

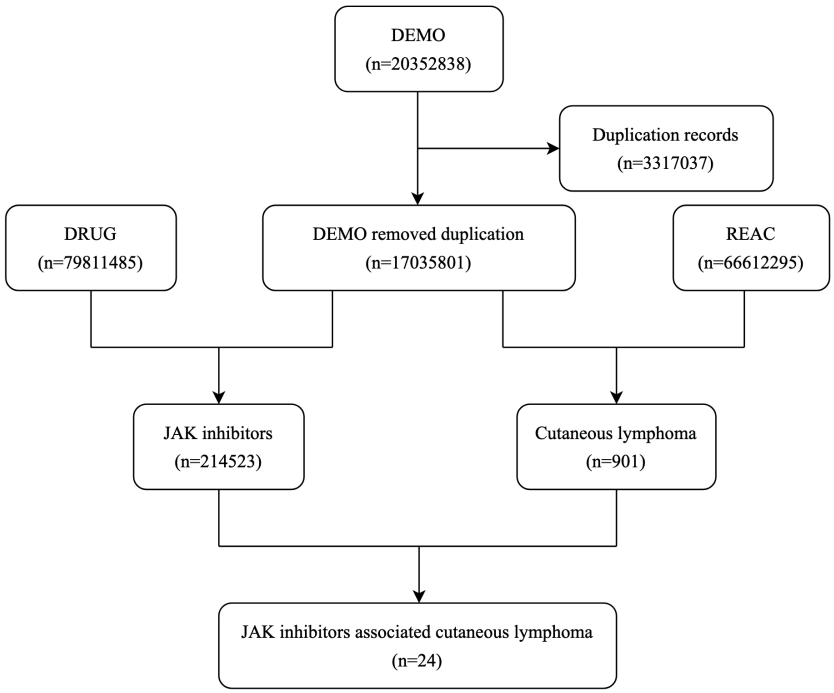

Fig. 1. Selection process for cases of JAK inhibitors-associated cutaneous lymphoma from the Food and Drug Administration Adverse Event Reporting System database.

Table I. Clinical features of patients who experienced JAK inhibitors-associated cutaneous lymphoma (source: FAERS database, January 2004–September 2023)

Features	Tofacitinib	Ruxolitinib	Baricitinib	Upadacitinib	Abrocitinib
Age					
18–44 years	0	0	1	3	0
45–59 years	0	0	0	1	0
60–74 years	4	0	1	2	1
75–89 years	0	0	1	0	0
Unknown	1	3	3	3	0
Sex					
Female	4	0	1	3	0
Male	1	0	3	6	1
Unknown	0	3	2	0	0
Area					
Asia	0	1	3	2	0
Europe	1	1	3	1	0
North America	4	1	0	6	1
Indications					
Chronic graft vs host disease	0	1	0	0	0
Colitis, ulcerative	2	0	0	0	0
Dermatitis, atopic	0	1	4	4	1
Eczema	0	0	0	2	0
Myeloproliferative neoplasm	0	1	0	0	0
Rheumatoid arthritis	3	0	1	0	0
Unknown	0	0	1	3	0

limit of the ROR 95% CI was > 1, it indicated a potential adverse reaction signal. For PRR analysis, if PRR > 2.0, the event count > 2, and the associated $\chi^2 > 4.0$, this suggested a potential adverse reaction signal. In Bayesian confidence propagation neural network analysis, IC > 0 and a lower limit of the 95% CI of IC > 0 indicated a potential adverse reaction signal. In multi-item gamma Poisson shrinker analysis, if the lower limit of the 95% CI of EBGM was ≥ 2, this suggested a potential adverse reaction signal. If any of the 4 algorithms met the criteria, we considered the drug to be associated with cutaneous lymphoma adverse reactions (12).

RESULTS

General characteristics

A total of 214,523 adverse event reports related to the use of JAK inhibitors were retrieved from the FAERS database. Cutaneous lymphoma adverse events accounted for only a small fraction of the total adverse reactions in all of the JAK inhibitor reports, with a case number of 24, which accounted for 0.01% (24/214,523) of the overall cases. Among these, 5 cases reported cutaneous lymphoma without specifying the subtype, 19 cases reported cutaneous T-cell lymphoma, and no cases of cutaneous B-cell lymphoma were reported. The breakdown of cutaneous lymphoma reports by specific inhibitor is as follows: 5 cases (20.83%) for tofacitinib, 3 cases (12.50%) for ruxolitinib, 6 cases (25.00%) for baricitinib, 9 cases (37.50%) for upadacitinib, and 1 case (4.17%) for abrocitinib. Age was not reported for 10 cases. There was a median age of 61 years (interquartile range 44.25–69.25) among those with a reported age. The majority of patients (64%) were aged 60 or older ($n = 9$). Gender was not reported for 5 cases, with a reported male-to-female ratio of 11:8 (no statistical difference). The majority of cases originated from the North America ($n = 12$, 50%). Most cases ($n = 10$, 41.67%) occurred during treatment for atopic dermatitis (**Table I**).

Onset time analysis

A total of 11 cases reported onset time (3 for tofacitinib, 2 for ruxolitinib, 3 for baricitinib, 3 for upadacitinib and 0 for abrocitinib). Most cases of cutaneous lymphoma occur within 12 months of JAK inhibitors' application ($n = 10$, 90.90%). The average onset time was 8.64 months, with a median onset time of 8 months (interquartile range 1.5–8). The Kruskal–Wallis test indicated no significant statistical difference in onset time among the different drugs ($p = 0.49$). Dunn's multiple comparisons test further confirmed that there were no significant statistical differences in onset times between the drugs (**Fig. 2**).

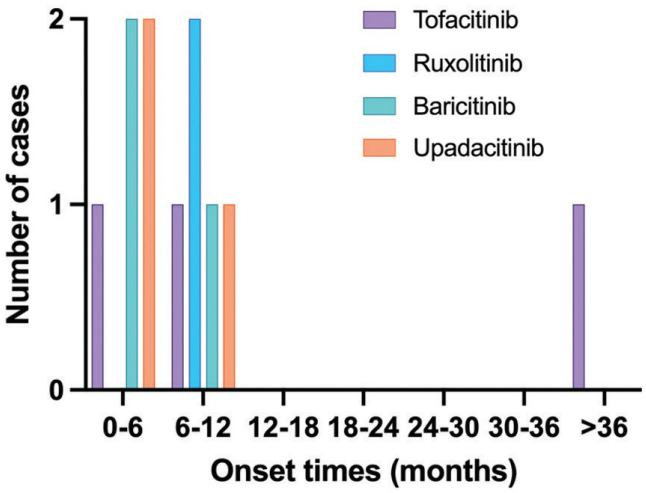

Fig. 2. Onset time of cutaneous lymphoma after the use of JAK inhibitors.

Table II. Outcome event of cutaneous lymphoma after the use of JAK inhibitors

	Reports, *n* (%)				
Outcome event	Tofacitinib	Ruxolitinib	Baricitinib	Upadacitinib	Abrocitinib
Death	0	1 (33.33)	0	0	0
Hospitalization	1 (20)	2 (66.67)	1 (16.67)	1 (11.11)	0
Life-threatening	1 (20)	0	0	0	0
Other serious effects (important medical event)	5 (100)	3 (100)	5 (83.33)	9 (100)	1 (100)

Outcome analysis

The reported adverse reaction events included death, hospitalization, life-threatening conditions, and other serious effects (important medical event). Among the reported cases, most (n=23, 95.83%) involved important medical events. Only 1 patient who underwent ruxolitinib treatment for myeloproliferative neoplasm had a fatal outcome, and 1 patient receiving tofacitinib for rheumatoid arthritis experienced a life-threatening event (**Table II**).

Disproportionality analysis and Bayesian analysis

According to the 4 data-mining algorithms, ruxolitinib, baricitinib, upadacitinib, and abrocitinib produced positive signals for cutaneous lymphoma. Among the 4 drugs, baricitinib exhibited the most significant association with cutaneous lymphoma (n=6; ROR=23.91, 95% CI 10.71–53.4; PRR=23.88, χ^2=103.67; IC=4.57, IC025=2.05; EBGM=23.73, and EBGM05=12.12), whereas abrocitinib and upadacitinib had relatively weaker associations with cutaneous lymphoma. Ruxolitinib had the weakest association with cutaneous lymphoma, with only Bayesian confidence propagation neural network analysis suggesting a weak adverse reaction signal (IC=0.11, IC025=0.03) (**Table III**).

DISCUSSION

Our study represents the largest real-world analysis of cutaneous lymphoma associated with JAK inhibitors. Through a comprehensive analysis of cases in the FAERS database, we elucidated the clinical characteristics and risk factors for adverse drug reactions. Disproportionality and Bayesian analyses identified 4 JAK inhibitors that showed signals for cutaneous lymphoma-related adverse drug reactions.

The relationship between JAK inhibitors and cutaneous lymphoma has long been controversial, with evidence supporting both therapeutic and lymphomagenic roles. Previous studies indicate that JAK inhibitors may be effective in CTCL, with clinical responses documented in 58 cases; in contrast, 5 case reports describe the emergence of CTCL following JAK inhibitor therapy (13). Regarding B-cell lymphomas, cerdulatinib (SYK/JAK inhibitor) has shown efficacy *in vitro* and in clinical trials for refractory B-cell lymphomas (14, 15), though its role in primary cutaneous B-cell lymphoma requires further study.

The differential effects of JAK inhibitors may relate to lymphoma subtype. In cases of CTCL treated with JAK inhibitors, the majority of patients have MF, SS, or SPTCL. Early-stage MF has been associated with the loss of JAK-STAT signalling pathway inhibitors SOCS1 and HNRNPK (5). In advanced MF, abnormal activation of STAT3 has been observed, which is associated with large-cell transformation (16). Mutations in JAK1, JAK3, STAT3, and STAT5B, as well as copy number variations (CNVs) in JAK2, STAT3, and STAT5B, have also been identified in MF and SS (17), supporting the rationale for using JAK inhibitors in their treatment. SPTCL frequently harbours HAVCR2 mutations with IL6-JAK-STAT3 pathway enrichment (6). Additionally, mutations in the JAK-STAT pathway have been found in other CTCL, such as cutaneous CD30+ lymphoproliferative disorders (18). Recently, JAK inhibitors have been found to promote the efficacy of immune checkpoint inhibitors in the treatment of small-cell lung cancer and non-Hodgkin's lymphoma, and the combination therapy was able to increase the number of anti-tumour T cells and NK cells, and reverse the state of T-cell depletion (19, 20). JAK inhibitors may be potential drugs for the treatment of cutaneous lymphoma.

Our FAERS analysis reveals varying lymphoma risks among JAK inhibitors. Baricitinib (JAK1/JAK2),

Table III. Signal detection for JAK inhibitor-associated cutaneous lymphoma

		ROR	PRR	IC	EBGM
Drug	*n*	(95% two-sided CI)	(χ^2)	(IC025)	(EBGM05)
Tofacitinib	5	0.81 (0.33,1.94)	0.81 (0.23)	–0.31	0.81 (0.39)
Ruxolitinib	3	1.08 (0.35,3.34)	1.08 (0.02)	0.11 (0.03)	1.08 (0.42)
Baricitinib	6	23.91 (10.71,53.4)	23.88 (130.67)	4.57 (2.05)	23.73 (12.12)
Upadacitinib	9	5.02 (2.6,9.69)	5.02 (28.7)	2.32 (1.2)	4.98 (2.88)
Abrocitinib	1	18.55 (2.61,131.95)	18.53 (16.57)	4.21 (0.59)	18.51 (3.58)

n: number of reports; ROR: reporting odds ratio; CI: confidence interval; PRR: proportional reporting ratio; χ^2: chi-square; IC: information component; IC025: the lower limit of the 95% two-sided CI of the IC; EBGM: empirical Bayesian geometric mean; EBGM05: lower 90% one-sided CI of EBGM.

abrocitinib (JAK1), and upadacitinib (JAK1) showed stronger signals, while ruxolitinib (JAK1/JAK2) showed only a weak signal, and tofacitinib (JAK1/JAK3) exhibited no risk signals. This suggests that the risk of inducing lymphoma may be significantly related to the specific type of JAK inhibitor used. Ruxolitinib has shown benefit in SPTCL (21, 22); however, it showed limited efficacy in MF, with a clinical trial reporting only a 14% clinical benefit rate (CBR, defined as the combination of complete response, partial response, and stable disease lasting at least 6 months) (23). *In vitro* studies have demonstrated that ruxolitinib, in combination with resminostat, exerts antitumor effects in the chick embryo chorioallantoic membrane model (24), suggesting a potential new direction for treating refractory CTCL. Tofacitinib has been suggested to potentially induce LyP in 1 case report (25), with no documented cases of its efficacy as a treatment. Notably, abnormal expression of JAK3 has been observed in CTCL (26), yet our pharmacovigilance study did not detect a risk signal for tofacitinib, suggesting its potential safety in this context. Upadacitinib has been associated with both the treatment and emergence of cutaneous lymphoma (27–29). To date, no cases of cutaneous lymphoma have been linked to abrocitinib in the literature. Baricitinib has been associated with a few cases of induced cutaneous lymphoma (30, 31). Therefore, it is important to regularly monitor skin lesions in patients using JAK inhibitors and, if necessary, to perform skin biopsy for further evaluation (32). Beyond their distinct JAK inhibition profiles, we propose that differences in underlying patient diseases, depth of immunosuppression, and tissue distribution may also account for the varying lymphoma signals among these agents. However, validating these hypotheses requires further investigation.

Tumour stage may further influence drug effects. In early-stage MF and SS, the skin lesion microenvironment often shows high expression of Th1-related markers such as STAT4 and IFN-γ (33). Conversely, in advanced-stage MF and SS, there is typically high expression of Th2-related markers like GATA3, IL-4, and IL-13 (34–36). Based on the signalling biology, JAK inhibitors effectively suppress both Th1 and Th2 pathways. However, their therapeutic impact is particularly pronounced in Th2-dominant contexts (e.g., atopic dermatitis). Therefore, we hypothesize that JAK inhibitors would demonstrate superior efficacy in advanced-stage MF and SS.

This study has limitations inherent to the FAERS database. As a spontaneous reporting system, it is susceptible to reporting biases and incomplete data, which prevents confirmation of diagnoses and limits subgroup analyses. Furthermore, for serious outcomes such as death or hospitalization, the data rarely specify the precise immediate clinical cause, hindering a deeper pathophysiological interpretation. Although our analysis identifies a potential signal, it cannot establish causality. Confounding factors, including indication bias and concomitant medications, cannot be fully accounted for.

In summary, our analysis of the FAERS database evaluated the relationship between JAK inhibitors and cutaneous lymphoma and found that baricitinib exhibited the strongest signal for adverse drug reactions. Clinicians should exercise caution when considering the use of JAK inhibitors in treatment.

ACKNOWLEDGEMENTS

Funding sources: This work was supported by National High Level Hospital Clinical Research Funding (2022-PUMCH- A-164) and Beijing Municipal Natural Science Foundation (Z210017) to Tao Wang, Beijing Key Clinical Specialty Construction Project, and National Key Clinical Specialty Project of China.

The authors have no conflicts of interest to declare.

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