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Review Article

Abrocitinib: A comprehensive review of its use in dermatology beyond atopic dermatitis

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ABSTRACT

Janus kinase (JAK) inhibitors are small molecule inhibitors that restrict proinflammatory pathways and are used in various autoimmune and inflammatory conditions, thus gaining significant threshold in dermatology. Abrocitinib, a JAK 1 inhibitor, was first approved in 2021 for use in atopic dermatitis (AD) and has been shown to be effective and safe in most cases. Since its approval, abrocitinib has also been reported to be varyingly successful in a group of other dermatological conditions and is a relatively safe drug. Our comprehensive review is based on an extensive PubMed search of all published literature on the use of abrocitinib in dermatological disorders with the exclusion of AD whereby we identified and included 37 papers. These include diverse eczematous, autoimmune, inflammatory, and keratinization disorders. Beyond AD, the largest number of patients were reported for vitiligo, hand eczema, chronic pruritus, and prurigo nodularis, all of which reported favorable outcomes. Pharmacodynamic studies have shown a dose-dependent decrease in platelet counts and disruption in lipid levels. Other mild adverse effects include nausea and dizziness which do not merit drug discontinuation. Our review highlights the broad usefulness that abrocitinib has as a therapeutic agent in inflammatory and autoimmune dermatoses, often when used as a single therapeutic agent, and has predictable safety and tolerability profiles. A larger number of randomized controlled trials are required to validate the off-label uses of abrocitinib and to optimize dosing strategies. This review also includes information about dosing recommendations, drug monitoring, and the use of oral abrocitinib in special patient groups.

Keywords: Abrocitnib, Eczema, Janus kinase inhibitors, Janus kinase inhibitors in dermatology

INTRODUCTION

The inflammatory cytokine response is mediated by the interaction between the Janus kinase–signal transducers and activators of transcription of the JAK-STAT pathway. The family of JAK includes 4 protein members of the JAK 1/2/3 and tyrosine kinase 2 and the family of STAT includes the STAT 1/2/3/4/5A/5B/6.

When activated, the JAK mediators pair within themselves and set in motion a downstream inflammatory cascade. The cytokine receptors phosphorylate the associated JAKs, with subsequent phosphorylation of the STAT proteins. When the STATs dimerize, they then translocate to the cellular nucleus with subsequent transcription of the genes. A dysregulated pattern of these proinflammatory JAK-STAT pathways has been seen in autoimmune and inflammatory dermatoses.

The development of JAK inhibitors ensured that these agents act on a specific target for curtailing inflammatory processes and suppressing immune-specific responses.[1]

This has led to the development of a host of small molecule inhibitors that act on various components of the JAK-STAT pathway, and there is a growing incorporation of these agents in the treatment of various dermatological conditions.

One such JAK1 inhibitor is abrocitinib (ab "roe sye" ti nib) which has been gaining popularity in the pharmacological arena, with its first approval in the UK in 2021 [Figure 1].[2] Prior to the approval of the drug in the UK, various Phase 3 studies under JAK1 Atopic Dermatitis Efficacy and Safety (JADE) trials and clinical trials were conducted with abrocitinib [Figure 2].[3]

MATERIALS AND METHODS

This review aims to evaluate the available scientific literature to understand the reported off-label uses of abrocitinib in dermatological conditions including atopic dermatitis (AD) and beyond. The authors conducted this review to summarize the literature on using abrocitinib for treating dermatological conditions including AD and other dermatoses, by searching the PubMed database for studies published before October

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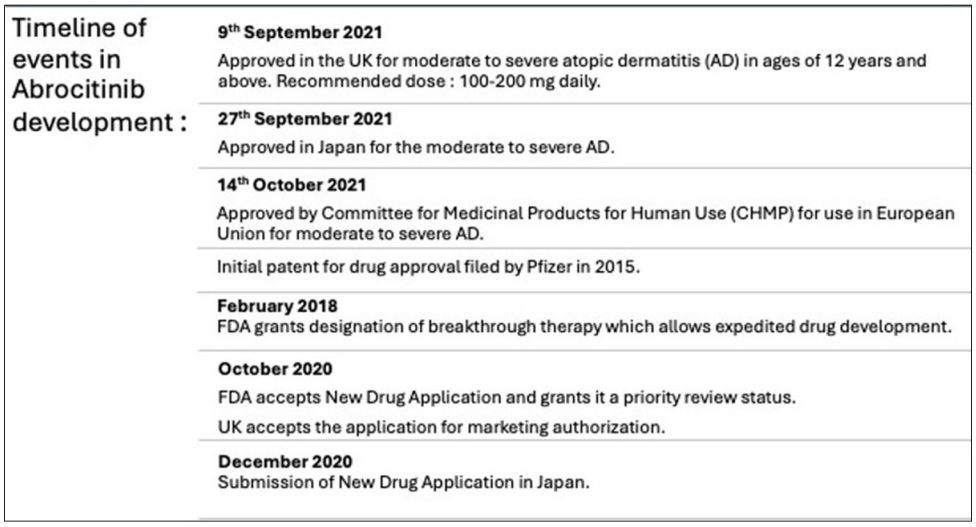

Figure 1: Timeline of development of abrocitinib. FDA: Food and drug administration.

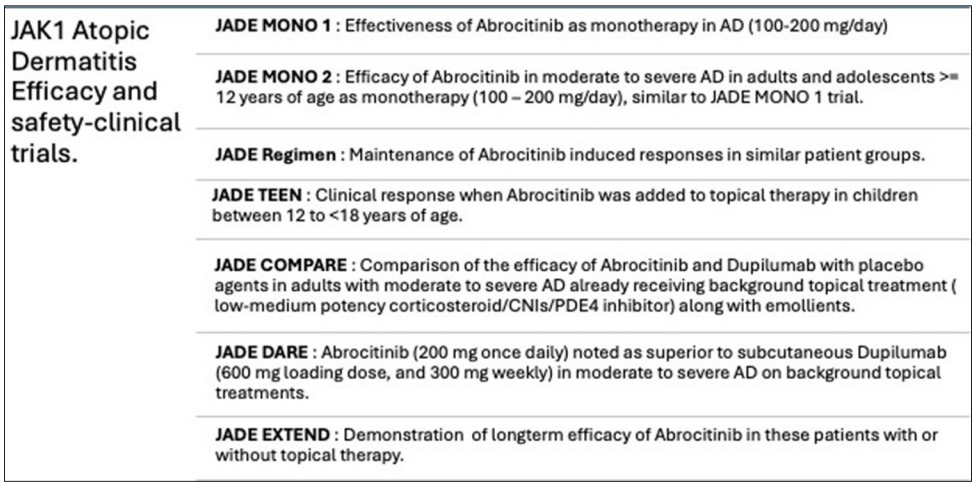

Figure 2: Janus kinase 1 atopic dermatitis efficacy and safety trials to assess the efficacy of abrocitinib in atopic dermatitis. AD: Atopic dermatitis, JADE: Janus kinase 1 atopic dermatitis, CNI : Calcineurin Inhibitors.

15th, 2024. The keywords employed for the database search were "Abrocitinib." Each article was then reviewed for data extraction.

RESULTS AND DISCUSSION

A database search showed 307 articles with the keyword abrocitinib. On excluding publications that had abrocitinib used for the treatment of AD, 37 articles remained and they were included in the review. The chosen articles were then analyzed to assess the use of abrocitinib in dermatological conditions other than AD, which gave us an outline of its off-label applications for sake of completion a synopsis of role of abrocitinib in AD was also prepared.

Mechanism of action

Abrocitinib is a selective JAK 1 inhibitor. It inhibits JAK1 binding by blocking the ATP binding site. Abrocitinib is selective for JAK1 over JAK2 (28-fold), JAK 3 (>340-fold), and tyrosine kinase 2 (43-fold). *In vitro*, the parent compound and its active metabolite show similar selectivity levels for JAK 1.

JAK 1/2/3/TyK 2 is expressed in multiple cell types. JAK3 is commonly seen in hematopoietic cells. Various domains facilitate the binding of JAKs to intracellular receptors. Pseudokinase domains play a regulatory role.

- Step 1: When the ligands bind to the cell receptors, there is dimerization which brings the associated JAKs close together. The JAK phosphorylates each other on tyrosine residues and then increases the activity of kinase domains. Then, they phosphorylate the tyrosine residues on the receptor subunit.
- Step 2: Once phosphorylation occurs, it creates a binding site for the SH2 domains of the intracytoplasmic transcription factors. This is known as STAT

- Step 3: The STATs are phosphorylated by JAKs which form hetero/homodimers and the dimer translocates into the nucleus which regulates the target gene expression of inflammatory mediators.

JAK-STAT is the common signal transduction pathway of cellular proliferation, migration, differentiation, and apoptosis [Figure 3].[4]

Pharmacokinetics

Abrocitinib has 91% oral bioavailability. Systemic drug absorption is not influenced by food. Peak concentrations of the drug are reached within 1 hour of drug intake. The t ½ of the drug is 5 hours.

When taken once daily, the drug reaches steady-state plasma concentrations within 48 hours.

The following are the drug metabolites of abrocitinib –

1. Pyrrolidinone pyrimidine – inactive
2. 2-hydroxypropyl – active
3. 3-hydroxypropyl – active.

The drug undergoes enzymatic metabolism through mostly CYP2C19 (53%), and CYP2C (30%), and a small percentage of the drug is metabolized by CYP3A4 and CYP2B6. When administered with strong CYP2C19 inhibitors, the dose of the drug should be reduced by half. When administered by CYP2C119/2C9 inducers, no dose adjustment is needed.[4]

Patient characteristics including age, weight, sex, race, and CYP2C19/CYP2C9 genotype have no apparent effect on the metabolism of the drug. In adolescent patients, no difference in metabolism has been seen as compared to adult patients.

There is no clinically significant difference in the active moiety of the drug in mild or moderate hepatic impairment or in mild renal impairment.

It is contraindicated in patients with severe hepatic impairment, considering the absence of studies in this population.

It has not been evaluated in patients with end-stage renal disease or renal replacement therapy.[3]

Pharmacodynamics

Reduction in JAK1 activity reduces downstream thrombopoietin production which consequently shows a fall in platelet count peaking at 4 weeks of treatment. The platelet count gradually rises back up starting at 4 weeks of treatment. After 4 weeks of abrocitinib treatment, there is also a dose-dependent increase in low-density lipoprotein (LDL) cholesterol, high-density lipoprotein cholesterol, and an increase in total cholesterol levels. A 10% rise in LDL cholesterol is seen with 100 mg of abrocitinib and a 15% rise is seen with 200 mg of abrocitinib.[4,5]

ABROCITINIB IN ATOPIC DERMATITIS

Abrocitnib has been approved for the treatment of moderate-to-severe AD in adults and adolescents of 12 years and older in patients who are candidates for systemic therapy. Approval status has been given for use in the UK, Japan, European Union, and US-FDA.

The only approved indication of abrocitinib use is AD, with oral and topical JAK inhibitors gaining rapid leverage in the treatment of AD. Second-generation JAK inhibitors act

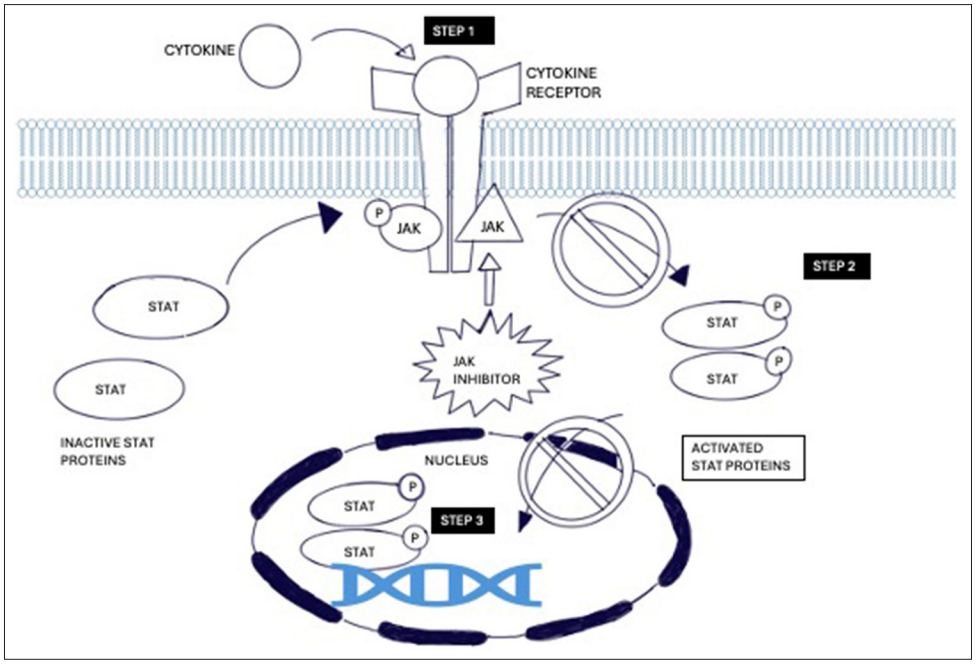

Figure 3: Mechanism of action of Janus kinase signal transducers and activators transcription inhibitors in cytokine mediation. JAK: Janus Kinase, STAT: Signal transducers and activators of transcription

against a single isoform of JAK. This selective inhibition sidesteps the undesirable effects of JAK2 inhibition such as neutropenia and anemia. Multiple studies were done to assess the effectiveness of abrocitinib in AD and its safety in different age groups and to compare it to pre-existing treatment agents like dupilumab. Comprehensive details of abrocitinib usage in AD are covered in Table 1 and details are as follows:

JADE MONO-1, Simpson *et al.* (July 2020)

A multicenter, double-blind, placebo-controlled, phase 3 trial was conducted in patients with moderate-to-severe AD of age 12 years or above, with a body weight of 40 kg or more. Both 200 mg and 100 mg doses of abrocitinib demonstrated significant efficacy in improving the signs and symptoms of moderate-to-severe AD compared to placebo. By week 12, a significantly greater proportion of patients in the abrocitinib 100 mg and 200 mg groups achieved an Investigator Global Assessment response and at least a 75% improvement in their Eczema Area and Severity Index scores compared to the placebo group. Notable reductions in pruritus were observed as early as the first post-baseline assessment. The 12-week study highlighted a favorable safety profile for abrocitinib, with no reported cases of venous thromboembolism, malignancy, major adverse cardiovascular events, or deaths.[6]

Silverberg *et al.* (October 2020)

This phase 3, double-blind, placebo-controlled, parallel-group randomized clinical trial involved patients aged 12 years and older who had moderate-to-severe AD for at least 1 year and an inadequate response to topical therapies administered for a minimum of 4 weeks within the past 6 months. The findings demonstrated that abrocitinib is an effective treatment for AD, with a low incidence of treatment-related adverse effects, making it a promising and safe therapeutic option.[7]

JADE REGIMEN, Blauvelt *et al.* (May 2021)

A phase 3 trial investigated the effects of randomized withdrawal of abrocitinib in patients undergoing treatment. This study assessed the necessity of maintaining a consistent dose and the flexibility of the treatment regimen. Patients initially received 200 mg of abrocitinib daily for 12 weeks. Those achieving clear or nearly clear skin then entered a 40-week phase, during which their dose was adjusted to either 200 mg, 100 mg, or a placebo. Patients who experienced an AD flare during this period entered a rescue phase with 200 mg of abrocitinib and a topical treatment to regain disease control. A high rate of relapse was seen with treatment discontinuation but the treatment response seen previously was recaptured with 200 mg of abrocitinib.[8]

JADE TEEN, Eichenfield *et al.* (June 2021)

A phase 3 trial was conducted to understand the safety and efficacy of oral abrocitinib in patients between 12 and 17 years of age, with moderate-to-severe AD, not responsive to topical therapy. At week 12, 41.6% in the 100 mg group and 46.2% in the 200 mg group achieved over 75% clearance in dermatitis. 2.1% in the 200 mg group had a serious adverse effect and none in the 100 mg group. The study confirmed that abrocitinib is an effective treatment for AD in adolescents, with a very low incidence of serious adverse effects, consistent with previous results.[9]

JADE COMPARE, Bieber *et al.* (October 2021)

The study was a multicenter, randomized, double-blind, placebo-controlled trial evaluating the efficacy and safety of two abrocitinib doses in adults with moderate-to-severe AD. Patients were allowed to use more than one topical therapy (low-to-medium potency topical glucocorticoids, topical calcineurin inhibitors, and topical phosphodiesterase 4 inhibitors). Patients 18 years of age or older with at least a 1-year history of AD were included.

The secondary objective was to evaluate and compare the efficacy of abrocitinib, with dupilumab based on reduction in itch at 2 weeks.

Abrocitinib 200 mg or 100 mg once daily had significantly greater reductions in moderate-to-severe atopic compared to placebo at weeks 12 and 16. The 200-mg dose of abrocitinib was superior to dupilumab with respect to relief of pruritus at week 2.

More adverse events were seen with Abrocitinib (Ab) 200 mg dose, compared to the lesser dose. 100 mg of Ab had adverse effects comparable to dupilumab.

The main adverse events with abrocitinib were nausea, acne, nasopharyngitis, and headache.[10]

JADE EXTEND, Shi *et al.* (December 2022)

An extended phase 3 study was conducted to assess the effectiveness of abrocitinib in patients who had earlier been treated with dupilumab. After the 12-week study, 76.9% of patients in the 100 mg abrocitinib group who had previously achieved the endpoint with dupilumab also met the endpoint with abrocitinib. Similarly, 83.3% of those in the 200 mg abrocitinib group who initially achieved the endpoint with dupilumab maintained their response. Among patients who did not respond to the initial dupilumab trial, 35.2% in the 100 mg abrocitinib group and 47.2% in the 200 mg group successfully reached the endpoint. These findings demonstrate that abrocitinib is effective in nearly half of the cases where dupilumab was previously ineffective. This could be attributed to abrocitinib's ability to target cytokines that are more directly involved in the pathophysiology of AD compared to dupilumab.[11]

JADE EXTEND, Reich *et al.* (February 2023)

This Phase 3 long-term extension study enrolled patients who had participated in previous abrocitinib trials for AD providing long-term safety information for abrocitinib in

Table 1: Phase 3 studies to assess the safety and efficacy of abrocitinib in different population groups.

No.	Author	Study design	Sample size	Intervention	Outcome	Result
1	Simpson *et al.* (July 2020)[6]	A multicenter, double-blind, randomized phase 3 trial (JADE MONO -1), patients (aged≥12 years) with moderate-to-severe AD with a body weight of 40 kg or more (JADE MONO-1).	387	387 participants with moderate to severe AD were selected and randomly assigned to 200 mg abrocitinib, 100 mg abrocitinib, or placebo in a 2:2:1 ratio. 154 participants were assigned to 200 mg abrocitinib group, 156–100 mg abrocitinib group, and 77 were assigned to placebo.	The primary endpoint of study was 75% improvement in symptoms after 12 weeks.	40% of the 100 mg group saw at least a 75% improvement in symptoms with 63% of the 200 mg group reaching the same endpoint. 12% of the placebo group also saw a 75% improvement in symptoms over 12 weeks.
2	Silverberg *et al.* (October 2020)[7]	A phase 3, double-blinded, placebo-controlled, parallel-group randomized clinical trial included patients 12 years or older with a clinical diagnosis of moderate-to-severe AD for at least 1 year and inadequate response to topical medications given for at least 4 weeks within 6 months.	391	155 participants were assigned to the 200 mg abrocitinib group, 158 to the 100 mg abrocitinib group, and 78 were assigned to the placebo group.	The primary endpoint of the study was a 75% improvement in symptoms after 12 weeks.	In the 12-week trial, 44.5% of the 100 mg group and 61% of the 200 mg group achieved at least a 75% improvement in symptoms, compared to 10.4% in the placebo group. More patients in the placebo group also discontinued treatment.
3	Blauvelt *et al.* (May 2021)[8]	A multicenter, double-blind, randomized phase 3 trial (JADE REGIMEN), patients (aged ≥12 years) with moderate-to-severe AD with a bodyweight of 40 kg or more. (JADE REGIMEN)	798	Patients received 200 mg of abrocitinib daily for 12 weeks. Those achieving clear or nearly clear skin entered a 40-week phase, where they were randomized (1:1:1) to continue 200 mg (*n*=266), switch to 100 mg (*n*=265), or receive a placebo (*n*=267). Patients experiencing AD flares during this period entered a rescue phase with 200 mg of abrocitinib and topical treatment to restore disease control.	The primary endpoint was the need for rescue medication due to loss of response during the maintenance phase.	During the rescue period, flares occurred in 16.2% of the 200 mg group, 39.2% of the 100 mg group, and 76.4% of the placebo group. Response was regained with 200 mg of abrocitinib and topical treatments in 36.6%, 58.8%, and 81.6% of these groups, respectively, highlighting the effectiveness of 200 mg abrocitinib in restoring response after relapse.
4	Eichenfield *et al.* (June 2021)[9]	A randomized clinical trial, phase 3, randomized, double-blind, placebo-controlled study was conducted in patients aged 12–17 years with moderate-to-severe AD and an inadequate response to topical medication or requiring systemic therapy.	287	Participants with moderate-to-severe AD were randomly assigned to receive either 200 mg of abrocitinib, 100 mg of abrocitinib, or a placebo in a 1:1:1 ratio.	The primary endpoint of the study was achieving clear or almost clear skin and/or a 75% improvement in symptoms after 12 weeks.	Over 12 weeks, 41.6% of the 100 mg group and 46.2% of the 200 mg group achieved at least a 75% improvement in symptoms, compared to 24.5% in the placebo group

(*Contd...*)

Table 1: (*Continued*).

No.	Author	Study design	Sample size	Intervention	Outcome	Result
5	Bieber *et al.* (October 2021)[10]	Phase 3, multicenter, randomized, double-blind, placebo-controlled trial unresponsive to topical therapy or needed systemic therapy -with secondary objective of comparison of efficacy with Dupilumab on the basis of reduction of itch JADE COMPARE trial.	838	Patients were randomly assigned to be administered 200 mg or 100 mg daily oral dose of abrocitinib, 300 mg of dupliumab, which is a competing therapy, subcutaneously every other week, or a placebo for a period of 16 weeks in a 2:2:2:1 ratio	A reduction in the severity of AD to clear or mild after 16 weeks of treatment.	Reduction in severity of AD was seen in 48.4% of the 200 mg abrocitinib group, 36.6% of the 100 mg abrocitinib group, 36.5% of the 300 mg Dupilumab group, and 14% of the placebo group by week 12. A greater reduction in itch was seen with 200 mg oral abrocitinib at week 2 as compared to Dupilumab.
6	Reich *et al.* (July 2022)[12]	Efficacy and safety of abrocitinib vs Dupilumab in moderate to severe AD - a randomized, double-blind, multicenter phase 3 trial. Randomized, double-blind, active-controlled, parallel-treatment (JADE COMPARE).	727	The abrocitinib group was given 200 mg of abrocitinib orally daily while the dupilumab group was given 300 mg of dupilumab every 2 weeks, after a 600 mg loading dose, subcutaneously.	Reduction in severity from moderate to mild or clear AD.	48% of the abrocitinib group reached the primary endpoint by week 2 of the study while only 26% of the dupilumab group reached the primary endpoint in the same amount of time. At the end of the 26-week trial, 68% of the abrocitinib group reached the primary endpoint while 63% of the dupilumab group reached the primary endpoint.
7	Shi *et al.* (August 2022) [11]	Efficacy and safety of abrocitinib in adults with moderate-to-severe AD after switching from dupilumab (JADE EXTEND) - Patients with moderate-to-severe AD received abrocitinib 200 mg or 100 mg once daily in JADE EXTEND (phase 3 extension) after dupilumab in double-blind, placebo-controlled phase 3 JADE COMPARE.	203	Focused on the dupilumab-treated patient population from JADE COMPARE and 203 patients from the dupilumab group were used in the study. 73 were given 200 mg of abrocitinib orally and the other 130 were given 100 mg of abrocitinib orally.	Reduction in severity from moderate to mild or clear AD.	Patients who responded to prior dupilumab in JADE COMPARE maintained most of these clinical benefits with abrocitinib treatment for 12 weeks in JADE EXTEND. Additionally, large proportions of patients not responsive to dupilumab in JADE COMPARE achieved clinical benefit with abrocitinib treatment for 12 weeks, with clearance of dermatitis and itch relief.

(*Contd...*)

Table 1: (*Continued*).

No.	Author	Study design	Sample size	Intervention	Outcome	Result
8	Reich *et al.* (February 2023)[12]	Phase 3, long- term extension study with patients from previous abrocitinib AD trials, to evaluate the abrocitinib efficacy up to 48 weeks and long- term safety in patients with moderate- to- severe AD. This analysis includes data from adolescent (12 to <18 years of age) and adult (≥18 years of age) patients who received prior treatment with abrocitinib in JADE MONO- 1, JADE MONO- 2 and JADE COMPARE.	1116	Patients who had previously participated in JADE MONO- 1, JADE MONO- 2 or JADE COMPARE, were continued and the dose in JADE EXTEND remained consistent with the qualifying study, in the 200 mg or 100 mg groups.	To evaluate the abrocitinib efficacy up to 48 weeks and long- term safety in patients with moderate- to-severe AD.	About 70% and 45% of patients received a≥48 weeks, respectively. At week 48, almost to complete clearance was seen in 52% patients receiving 200 mg abrocitinib and 39% patients on 100 mg abrocitinib. Significant improvement in skin lesions and pruritus was seen in moderate to severe AD.

AD: Atopic dermatitis, JADE: JAK1 atopic dermatitis efficacy and safety

a population with a median drug exposure of 10.4 months, where approximately 45% of patients had been exposed to the drug for ≥48 weeks. The most frequent adverse events included nasopharyngitis, AD, nausea, upper respiratory tract infections, acne, increased creatine phosphokinase, and herpes simplex.[12]

Long-term efficacy data showed:

1. Sustained control of AD was seen with abrocitinib without the need for concomitant topical medications.
2. A significant proportion of patients who did not meet formal response criteria by week 12 showed clinically meaningful improvements by week 24.

Serious adverse effects occurred in 7% of patients receiving abrocitinib 200 mg and 5% of those on 100 mg, with study discontinuation in 9% and 7% of patients receiving 200 mg and 100 mg, respectively.

ABROCITINIB IN OTHER DERMATOSES

However, considering the role of JAK inhibitors in other inflammatory and autoimmune dermatoses, this review examines other published data where abrocitinib has been used in the treatment of other dermatoses, which are frequently refractory to other treatments as reported.

Demonstration of therapeutic use has mostly been restricted to isolated case reports and small groups of patients [Table 2].

Alopecia areata (AA)

In AA, there is a T-cell-mediated autoimmune destruction of hair follicles with resultant nonscarring patchy hair loss. The extent of involvement varies in the patient and may be restricted to the scalp or involve other hair-bearing areas. The condition carries significant detriment to the patient's psychological health.

Tofacitinib, a JAK 1 and JAK 3 inhibitor, has been increasingly popular in the treatment of AA. Several case reports and case series have reported successful treatment of AA and alopecia universalis with abrocitinib at 100 mg daily and 200 mg daily doses.

In comparison to other dermatological conditions, a long median time to complete resolution (about 10 months) was noted in AA.[13-15]

Lichen sclerosus (LS)

LS is a non-specific inflammatory genital dermatosis, and a key regulator is interleukin (IL)-6 which has a pro-inflammatory role. It has been hypothesized that LS has more of an autoimmune disease with a preferable Th1 immune response and increased expression of proinflammatory cytokines specific to Th1-interferon (IFN) gamma-induced immune response. Abrocitinib, as an inflammatory agent, also is considered to reduce the release of IL-6.

Table 2: List of conditions in which oral abrocitinib has been used.

Condition	Level of evidence
Eczema	
Moderate to severe AD in adults.*	Level 2
Occupational airborne allergic contact dermatitis (ABCD)	Level 4
Nipple and areola eczema	Level 4
Hand eczema	Level 4
Autoimmune conditions	
AA	Level 4
LS	Level 4
PG	Level 4
LV	Level 4
Oral LP	Level 4
Vitiligo	Level 4
Inflammatory conditions	
HS	Level 4
Prurigo nodularis	Level 4
Necrobiosis lipoidica	Level 4
Lichenoid amyloidosis	Level 4
Disorders of keratinization	
PPK	Level 4
HHD	Level 4
Netherton syndrome	Level 4
Others	
Pruritus of unknown origin	Level 4

*Approved indications (US-FDA): Moderate to severe AD in adults, when the condition is refractory, has not responded to systemic immunosuppressants including biologics. AD: Atopic dermatitis, AA: Alopecia areata, LS: Lichen sclerosus, PG: Pyoderma gangrenosum, LV: Livedoid vasculopathy, LP: Lichen planus, HS: Hidradenitis suppurativa, PPK: Pruritic papular porokeratosis, HHD: Hailey–Hailey disease

A demonstrable effective response was seen in a 50-year-old male with plasma cell balanitis and LS where there was complete remission within 1 month of abrocitinib 100 mg daily.[16]

Abrocitinib was also used in 10 patients, 7 females, and 3 males, mean age 35.4 years, who had been treated with different topical agents for LS and had an inadequate response. They were given a 100 mg daily dose of abrocitinib, which was continued for 4 months after all patients showed good outcomes. One patient developed dyslipidemia and had a fall in their platelet count at the end of the treatment period. The rest showed no adverse effects.[17]

Pyoderma gangrenosum (PG)

Abrocitinib has been effectively used in a 16-year-old male with perianal PG. After worsening symptoms with doxycycline, isotretinoin, oral steroids, and cyclosporine A, the patient was started on abrocitinib 100 mg daily. Improvement was seen within a week, and within a month, the ulcer size reduced significantly, with alleviation of swelling and discharge. During the 4-month follow-up period, further improvement was maintained.[18]

Livedoid vasculopathy (LV)

In a 31-year-old female with LV for over 2 years, when adequate response was not seen with glucocorticoids, thalidomide, hydroxychloroquine, doxycycline, or cyclosporine, the patient was started on 100 mg abrocitinib. There was rapid improvement in pain, swelling, and ulceration with complete remission in 6 weeks.

During the next 12-week follow-up period, when the dose was tapered to 100 mg once every 2 days, there was no recurrence. No adverse effect was noted.[18]

Hidradenitis suppuritiva (HS)

In a 17-year-old male, with axillary HS with recurrent inflammatory lesions over the 3-year duration, minimal resolution was seen despite prolonged medical and surgical treatment. Following such a poor clinical outcome, the patient was started on abrocitinib at 100 mg once daily.

With treatment, there was a significant reduction in the pain and size of abscesses. Excellent improvement was seen in 2 weeks, and the drug was continued for 6 weeks, then reduced to a dose of 100 mg every 2 days. No lesions were reported during a follow-up period of 10 weeks.[18]

Lichenoid amyloidosis

Significant lesional improvement with symptomatic betterment was seen in 2 patients (53-year-old female and 59-year-old female) with daily abrocitinib 100 mg on treatment for 2–4 months, and sustained improvement was seen when the drug was tapered to once in 2–3 days.[19]

Porokeratosis

Abrocitinib has been effective in the treatment of eruptive pruritic papular porokeratosis with rapid resolution of pruritus. Increased IL-31 cells are seen in the lesional skin. Dysregulated keratinocyte proliferation also causes a Th2 inflammatory response which further upregulated IL-31. Abrocitinib through JAK1 blockage inhibits IL-31 by over 85%. In a 75-year-old male with porokeratosis for 60 years, rapid relief in itching was seen in 24 hours followed by subsidence of skin lesions within 30 days of treatment.[20]

Hailey–Hailey disease (HHD)

HHD is a rare genodermatosis with a mutation in the ATP2Cl gene that causes impaired Ca+2 transport signaling. It presents as painful erythematous, blistering skin at sites of friction, that will then erode with crusting, scaling, and hypertrophic vegetative growths. The primary skin barrier defects in HHD lead to cytokine-mediated secondary Th2 inflammation. The use of JAK inhibitors inhibits the Th2- mediated IL-4 and IL-13 signaling, which also helps in Ca+2 mobilization in keratinocytes.[21]

A 41-year-old male with HHD was successfully treated with 4 weeks of abrocitinib 100 mg daily.[22]

Necrobiosis lipoidica

A 53-year-old female with extensive NBL for over 10 years, treated previously with steroids, calcineurin inhibitors, psoralens, and hydroxychloroquine, was treated with oral abrocitinib. Treatment was started with 200 mg abrocitinib once daily for 11 months.[23]

Vitiligo

Vitiligo is due to the autoimmune destruction of melanocytes by self-reactive CD8+ T lymphocytes. CD8+ T cells produce IFN gamma which then causes JAK-STAT phosphorylation. JAK inhibitors, both in topical form (ruxolitinib 1.5%, tofacitinib 2%, ifidancitinib 0.46%, delgocitinib) and oral form (tofacitinib, baricitinib), have proven to be effective in treatment.[1]

A 61-year-old male with active acrofacial vitiligo, inadequate response to topical tacrolimus 0.1%, was treated with 100 mg daily abrocitinib and had significant repigmentation. After 2 months, there was no recurrence of the patches, and was successfully maintained on topical Tacrolimus, with repigmentation developing 4 months after abrocitinib stoppage.[24,25]

In a case series, Xu *et al*. treated 11 patients of refractory progressive vitiligo, between 26 and 59 years of age (mean age 35.9 years, mean disease duration 17.7 years) – 2 males and 9 females – with Abrocitinib 100 mg daily for 16 weeks. Favorable clinical outcomes were seen in all, with disease stability achieved. 10 patients continued treatment with 100 mg every alternate day for 2 months, along with narrow-band ultraviolet B therapy. Headache, dizziness, and nausea were noted in 3 patients.[26]

Oral lichen planus (LP)

LP is an inflammatory condition with T-cell-predominant lymphocytic reactions. Reticulated white patches are seen over the oral mucosa associated with burning or increased gustatory sensitivity in oral LP.

JAK-STAT cytokines have been proposed to play a definitive role in disease pathogenesis. JAK inhibitors have demonstrated effectiveness in the treatment of oral LP, including tofacitinib, baricitinib, and upadacitinib.[26]

A 58-year-old male, who had failed to show effective response to other immunosuppressants, responded well to abrocitinib 200 mg once daily for 7 days, followed by 100 mg daily, with improvement in 3 months.[27]

Allergic contact dermatitis (ACD)

ACD is a delayed type IV hypersensitivity reaction where diffuse eczematous lesions manifest within hours to days after exposure to a contact allergen.[28] Complete clearance was reported in a 37-year-old male with ACD to *Compositae species*, where 100 mg abrocitinib was given daily for 8 weeks.[29]

Nipple and areola eczema

Nipple eczema is seen in patients with AD, with a pathogenic role of the same Th2 inflammatory pathways. A 28-year-old male, recently recovered from severe acute respiratory syndrome coronavirus 2 infection, with background atopy, presented with complaints of nipple eczema. Dermatitis had limited response to emollients, steroids, calcineurin inhibitors, and phototherapy. He was started on 100 mg abrocitinib once daily and pruritus reduced remarkably within 24 hours. Treatment was continued for 12 weeks and the lesions completely cleared. No relapse was seen in 3 months after treatment cessation.[30]

Prurigo nodularis (PN)

PN occurs with hyperplasia of intraepidermal neuronal fibers with proinflammatory cytokine release such as IL-4, IL-17, IL-22, and IL-31. An increase in expression of STAT 3/6 has been seen in lesional biopsies which underline the therapeutic role of JAK-STAT inhibitors. Tofacitinib and baricitinib have been demonstrated to be useful in the treatment of PN.[31]

A 62-year-old female, with a 2-year history of PN, had complete resolution with 2 months of 100 mg abrocitinib and topical ruxolitinib, triamcinolone, and crisaborole. Unsatisfactory responses had been seen with dupilumab.[32]

Hand eczema

Eczema of the hands is a chronic and recurrent dermatoses that significantly affect the quality of life. JAK inhibitors are considered beneficial in the treatment of hand eczema. In a case series of 12 patients, 7 males and 5 females, mean age 46.3 ± 14.1 years, abrocitinib 100 mg once daily was administered and significant improvement was seen at week 16, with over 90% clearance in the lesions. 7/12 patients experienced adverse effects including nausea, headache, dizziness, and blurred vision – none of which required stoppage of the drug.[33] Other similar instances of abrocitinib use include a case of refractory hand and feet eczema treated successfully,[34] and a series of 17 patients, of which 13, showed over 90% clearance by week 16.[35]

Pruritus of unknown origin

Chronic pruritus of unknown origin or CPUO is a condition where there is persistent itching for over 6 weeks, without any identifiable cause. It involves both local and systemic type 2 inflammation which targets the type 2 cytokines (IL-4 and IL-13) thereby causing itch sensation over the sensory neurons. Dupilumab has been used to treat CPUO successfully. Kwatra *et al*. reported a case series wherein 10 patients with CPUO were treated with 200 mg abrocitinib once daily for 12 weeks, and all had favorable clinical

outcomes. However, 2/10 patients developed mild adverse effects including scalp folliculitis, acneform eruptions, and herpes labialis.[35]

Netherton syndrome

Netherton syndrome is a genodermatosis involving the skin, hair, and immune system. An underlying increase in serum immunoglobulin E levels and hypereosinophilia is seen, which is similar to AD. It is regulated by activated kallikreins which are serine proteases that cause overexpression of proallergic and proinflammatory cytokines. Persistent increased levels of kallikrein 5 induce an overexpression of proinflammatory Th2 and Th17 activity.[36,37]

A 28-year-old female with Netherton syndrome was treated with dupilumab but had a recurrence of lesions. She was switched to abrocitinib, and there was rapid and complete

Table 3: Overview of dermatoses (other than AD) treated by Abrocitinib

No.	Dermatological Indication	Type of Study	Patient Population	Dose and duration of Abrocitinib	Concomitant treatment.	Results	Adverse Effects	Author
1	Alopecia Areata after DRESS	Case Report	30 y/Female	100 mg OD x 2 months, then 200 mg OD for 2 months, then tapered down to 100 mg OD for 6 months	None	Significant improvement	None	Zhang *et. al.*[14]
		Case Report	40y/Female	100 mg OD	None	Improved at 1 week, rebound at 8 weeks stopping, good results at 12 weeks.	None	Cai L *et. al.*[51]
2	Pediatric Alopecia Areata	Case Report	11 y/Male	100 mg OD for 4 months	None	Hair regrowth on the scalp	None	Huang *et. al.*[13]
3	Alopecia Areata with Atopic Dermatitis	Case Report	14 y/Female	200 mg OD for 3 months	None	Complete regrowth of the hair with subsidence of AD lesions	None	Zhao J *et. al.*[15]
		Case Report	13 y/Male	100 mg OD for 2 months followed by 200 mg OD for 2 months then reduced to 100 mg once daily for 6 months.	None	Significant hair regrowth was seen at 6 months	None	
4	Lichen Sclerosus	Case Series	7 females and 3 males between 22 to 48 years of age	100 mg OD	None	Disease control seen in 3 months	None	Bao *et. al.*[16]

(*Contd...*)

Table 3: *(Continued)*

No.	Dermatological Indication	Type of Study	Patient Population	Dose and duration of Abrocitinib	Concomitant treatment.	Results	Adverse Effects	Author
5	Lichenoid Amyloidosis	Case Report	1 male and 1 female	100 mg once daily for 2-4 months, tapered to once in 2-3 days	None	Marked improvement in 8 weeks	None	Bai *et. al.*[19]
6	Hailey Hailey Disease	Case Report	41 y/Male	100 mg OD	Topical zinc oxide	Evident clinical improvement after 4 weeks	None	Mitroi *et. al.*[23]
7	Necrobiosis Lipoidica	Case Report	53 y/Female	200 mg OD x 11 weeks, then 100 mg OD	NR	Improvement after 11 months	Mild abdominal pain	Arnet *et. al.*[24]
8	Allergic Contact Dermatitis - occupational airborne	Case Report	37 y/Male	100 mg OD	None	Marked clearance in 2 months	None	Baltazar *et. al.*[30]
9	Pyoderma Gangrenosum	Case Report	16 y/Male	100 mg OD	Cyclosporine 50 mg BD for 3 weeks	Significant improvement after 4 weeks	None	Chen *et. al.*[18]
10	Livedoid Vasculopathy	Case Report	31 y/Female	100 mg OD x 4 weeks, followed by 100 mg every 2 days.	None	Complete remission after 6 weeks	None	Chen *et. al.*[18]
11	Hidradenitis Suppuritiva	Case Report	17 y/Male	100 mg OD x 4 weeks, followed by 100 mg every 2 days.	Doxycycline 100 mg BD for 2 weeks	Almost complete clearance after 6 weeks	None	Chen *et. al.*[18]
12	Netherton Syndrome	Case Report	28 y/Female	200 mg OD x 1 week, tapered to 100 mg OD x 3 weeks	None	Significant improvement after 3 weeks	Light-headedness, nausea	Zheng *et. al.*[39]
13	Oral Lichen Planus	Case Report	58y/Male	200 mg OD x 12 weeks, tapered to 100 mg once daily	None	Good response after 12 weeks	None	Solimani *et. al.*[28]
14	Nipple-Areola Eczema	Case Report	28 y/Male	100 mg OD x 12 weeks	None	Marked improvement in 12 weeks and no relapse at 20 weeks	None	Teng *et. al.*[31]
15	Vitiligo - Progressive	Case Series	11 patients - 2 Males and 9 Females - Mean Age 35.9 years	100 mg OD x 16 weeks tapered to 100 mg every 2 days	Phototherapy	Favourable outcomes	Headache, dizziness, nausea, abdominal discomfort (27.27%)	Xu Z *et. al.*[26]

(*Contd...*)

Table 3: *(Continued)*

No.	Dermatological Indication	Type of Study	Patient Population	Dose and duration of Abrocitinib	Concomitant treatment.	Results	Adverse Effects	Author
16	Vitiligo - Acrofacial Progressive	Case Report	61 y/Male	100 mg OD	None	Significant repigmentation and no progression at 2 months follow up, was restarted on topical tacrolimus 0.1% daily	None	Satkunathan *et. al.*[25]
17	Hand Eczema	Case Series	12 patients - 7 Males and 5 Females, Mean Age 46.3 y	100 mg OD	None	Significant improvement at 16 weeks follow up	Nausea (58%), headache, acne, dizziness, blurred vision in others.	Li Y *et. al.*[36]
18	Chronic Pruritus of Unknown Origin	Case Series	10 patients - 8 Males and 2 Females, Mean Age 70.7 y	200 mg OD x 12 weeks	None	Significant improvement at 12 weeks; treatment discontinued thereafter.	Scalp folliculitis, acneiform eruption, herpes labialis	Kwatra SG *et. al.*[35]
19	Pityriasis Rubra Pilaris	Case Series	5 patients - 2 Males and 3 Females, Mean Age 47.7 y	100 mg OD	None	At 4 weeks, PASI 24.6 ± 19.61 became 4.6 ± 4.03, and BSA of 39.6 ± 33.41 became 13.6 ± 11.89.	None	Li Y *et. al.*[40]
20	Erythematotelangiectatic Rosacea	Case Series	4 females, Mean age = 37.5 y	100 mg OD for 16 weeks followed by once every 4 days		Significant improvement after 4 weeks in 1 patient, Mild improvement in 2 patients, and no improvement in 1 patient.	None were reported, and 1 patient was discontinued due to Hepatitis B detection.	Zhang T *et. al.*[41]
21	Granulomatous Rosacea	Case Report	53 y/Female	100 mg OD for 20 weeks followed by 100 mg once weekly	None	Significant improvement at 20 weeks follow-up	None	Ren M *et. al.*[42]
22	Steroid induced Rosacea	Case Series	4 Females, Mean Age = 40 y.	100 mg OD	Topical azelaic acid and a skin barrier protection	Significant improvement.	None	Xu B *et. al.*[43]
23	Chronic Actinic Dermatitis	Case Report	70 y/Male	100 mg OD for 6 weeks followed by twice a week.	None	Significant improvement after 6 weeks.	None	Jin X *et. al.*[44]

(*Contd...*)

Table 3: *(Continued)*

No.	Dermatological Indication	Type of Study	Patient Population	Dose and duration of Abrocitinib	Concomitant treatment.	Results	Adverse Effects	Author
24	Bullous Pemphigoid	Case Report	52 y/ Female,	100 mg OD	60 mg Methylprednisolone reduced to 2 mg daily after 1 month	Significant improvement	None	Jiang W *et. al.*[45]
		Case Report	83 y/Male	100 mg OD	4 mg Methylprednisolone	Significant improvement	None	Jiang W *et. al.*[45]
25	Tattoo granuloma with uveitis	Case Report	41 y/Male	100 mg OD	Prednisone 30 mg daily with pranoprofen eye drops for uveitis	Significant improvement with no new lesions at 6 weeks follow-up	None	Yang Y *et. al.*[46]
26	Granuloma annulare	Case Report	29y/Female	150 mg OD	NR	Significant improvement after 6 weeks	None	Liu W *et. al.*[47]
		Case Report	77y/Female	200 mg OD	NR	Complete clearance in 3 months	Nausea, herpes labialis	Michels A *et. al.*[48]
27	Psoriasis	Case Report	69y/Female	100 mg OD interrupted at 12 weeks	None	Marked remission of skin lesions	None	Mao J *et. al.*[49]
28	Post-zygomatic arch hyaluronic acid filler: cheek and jawline edema at 6 weeks	Case Report	55y/Female	100 mg OD	None	Complete clearance in 2 months	None	Lopez MHP *et. al.*[50]
29	Eosinophilic pustular folliculitis	Case Report	50y/Male	100 mg OD	None	Improved at 4 weeks, and cleared by 6 months	None	Cai L *et. al.*[51]
30	Dissecting cellulitis of the scalp	Case Report	27y/Male	100 mg OD	NR	Improvement after 4 months	None	Jin S *et. al.*[52]
31	Mucous Membrane Pemphigoid	Case Report	62y/Female	100 mg OD for 4 weeks, followed by once in 2 days, for 2 months	NR	Significant improvement after 3 days, complete clearance in 4 weeks	None	Teng Y *et. al.*[53]
32	Porokeratosis ptychotropica	Case Report	45y/Male	100 mg OD for 4 weeks	None	Improvement after 4 weeks, with good outcome at 4 months	None	Zhang X *et. al.*[21]
33	Pruritic Papular Porokeratosis	Case Report	75y/Male	100 mg OD	None	Favorable outcome at 1 month follow-up	None	Xia J *et. al.*[20]
34	Perioral dermatitis	Case Report	26y/Female	100 mg OD	None	Favourable outcome at 1 month follow-up	None	Teng Y *et. al.*[54]

(*Contd...*)

Table 3: *(Continued)*

No.	Dermatological Indication	Type of Study	Patient Population	Dose and duration of Abrocitinib	Concomitant treatment.	Results	Adverse Effects	Author
35	Pityriasis Rosea	Case Report	25y/Female	100 mg OD	NR	Significant improvement after 2 days; Complete clearance in 14 days	NR	Wu H et.al.[55]
36	Prurigo Nodularis	Case Report	62y/Female	100 mg OD	Ruxolitinib, Triamcinolone topical for 2 months	Complete clearance after 2 months	None	Vander Does A *et. al.*[33]
		Case Report	56y/Male	100 mg OD	NR	Almost complete clearance after 2 months	NR	Sun F *et. al.*[34]
		Case Series	10 female patients, mean age 58.6y	200 mg OD	None	Favourable clinical outcome in all cases	4 cases of headache, nausea, acneiform eruption, sore throat, and nasal congestion	Kwatra SG *et. al.*[35]

clearance in 3 weeks with 200 mg daily for 1 week. Since the patient complained of nausea and lightheadedness, it was tapered to 100 mg daily.[38]

Previous treatment included steroids, methotrexate, cyclosporine, and secukinumab.

Table 3 gives an overview of the various dermatoses in which oral abrocitinib has been used with the clinical outcome and observed adverse effects.[39-55]

Dosage recommendations, prescribing information

- Available dosage forms: 50 mg, 100 mg, 200 mg
- Recommended dosage: 100 mg orally once daily.

If not responding to 100 mg, can be increased to 200 mg once daily (CIBINQO Prescribing Information).[56]

Dosage recommendations for special groups are listed in Table 4.

Baseline investigations and follow-up

Recommendations for baseline investigations and follow-up monitoring of oral abrocitinib are listed in Table 5.[2]

Treatment discontinuation recommendations

Recommendations for discontinuing oral abrocitinib in patients are listed in Table 6.[2]

Instructions about administration

Abrocitinib is administered with or without food at approximately the same time each day.

Tablets must be swallowed whole with water. They should not be crushed, split, or chewed (CIBINQ prescribing information).[56]

Immunization

Before treatment, all age-appropriate vaccinations as recommended by current guidelines should be fulfilled.
Avoid vaccination with live vaccines immediately before, during, and immediately after abrocitinib (CIBINQ prescribing information).[56]

Contraindications

Abrocitinib is contraindicated in patients taking antiplatelet therapies, except for low-dose aspirin (≤81 mg daily), during the first 3 months of treatment.

Drug interactions

Abrocitinib dose should be reduced when it is co-administered with CYP2C19 inhibitors and it should be avoided with drugs that are both CYP2C19/CYP2C9 inhibitors.
Abrocitinib should be avoided with strong CYP2C19 or CYP2C9 inducers.[56]

Side effects

Adverse effects noted with oral abrocitinib include those ranging from serious infections, malignancies, cardiac events, and increased prothrombotic events, as listed in Table 7.[56]
Among AD patients, the frequency of side effects is similar with doses of 100 mg and 200 mg. Common adverse effects (AEs) in clinical practice are headache, nausea, nasopharyngitis, and acne, and these are mostly transient and mild to moderate in nature. Black box warnings for Janus Kinase inhibitors (JAKi) should prompt careful patient selection.[56]

Cost of treatment

The upfront price of abrocitinib is presumably more than other drugs for moderate-to-severe AD, but treatment with abrocitinib has shown that it saves 11000–13000 dollars annually per patient while factoring in the finances saved through reduction in doctors' visits and improved work quality of life. Overall, this increases the cost-effectiveness of abrocitinib in moderate-to-severe AD.

Table 4: Dosage recommendations for oral abrocitinib in special groups.

Special group	Recommendation
Moderate renal impairment	50 mg orally once daily. If not responding, increase to 100 mg once daily.
CYP2C19 poor metabolizer	50 mg orally once daily. If not responding, increase to 100 mg once daily.
Pregnancy	Effects of Abrocitnib on pregnancy are unknown. It is advised to avoid in pregnancy. Patients are encouraged to contact a pregnancy register if they are on abrocitinib.
Lactation/ Breastfeeding	• Not recommended. Abrocitinib may be started 1 day after the last dose (approximately 5–6 elimination half-lives).
Pediatric patients	• Not approved for use in under 12 years of age. • Reported to be effective in refractory AA in 11-year-old child, without any adverse effects.[6]
Geriatric patients (>65 years and older)	Higher percentage of patients, who are 65 years and older, have been discontinued from trials since a higher reduction in lymphocyte count (<500/mm^3) and platelet count (<7500/mm^3) is seen in this group. Higher incidence of herpes zoster is seen.
Severe hepatic impairment	Not recommended.
Severe renal impairment (eGFR 15–29 mL/min)	Mild renal impairment (60-89 mL/min) 100 mg daily Abrocitnib. Moderate renal impairment (30-59 mL/min) 50 mg daily Abrocitinib. Severe renal impairment (15-29 mL/min) Not recommended.
End-stage renal disease (eGFR<15 mL/min)	Not recommended.

eGFR: estimated Glomerular Filtration Rate

Table 5: Baseline investigations and monitoring suggestion for oral abrocitinib.

Parameter	Baseline investigations	Periodic follow-up
CBC	Not recommended in patients with: - Platelet count <150,000/mm^3 - Absolute lymphocyte count <500/mm^3 - Absolute neutrophil count <1,000/mm^3 - Hemoglobin <8 g/dL	Repeat at 4 weeks- Repeat 4 weeks after dose increase
RFT	Not recommended in patients with eGFR <15 mL/min or severe renal impairment.	Baseline RFT to be done to rule out renal dysfunction.
LFT	Not recommended in severe hepatic impairment.	Baseline LFT to rule out hepatic dysfunction.
T evaluation	- Rule out active TB; abrocitinib not recommended in active TB. - For latent TB or high-risk patients, start preventive anti-TB therapy. - Monitor for TB signs and symptoms yearly.	Monitor yearly for TB signs and symptoms.- Patients with high latent TB risk should have continued preventive measures.
Viral hepatitis screening	- Not recommended in active Hepatitis B or C. - Monitor HBV DNA levels in inactive Hepatitis B; consult hepatologist if DNA levels rise.	Periodically monitor HBV DNA levels in inactive Hepatitis B.
Skin examinations	- Perform skin examinations before initiating therapy. - Assess for signs of malignancies, including NMSC.	Repeat periodically for signs of malignancies, especially NMSC and lung cancers in current or past smokers.
Cardiovascular Risk (MACE)	- Use with caution in patients with cardiovascular risk factors or smoking history. - Inform patients of serious cardiovascular symptoms.	Monitor for signs of MACE at 4 weeks and 4 weeks after dose increase.
Thrombosis risk	Avoid use in patients with an increased risk of thrombosis.	Monitor for signs of thrombosis periodically.
Blood lipid levels	Check baseline levels.	Repeat at 4 weeks and 4 weeks after dose increase.
Signs of infection	Baseline evaluation not explicitly mentioned but consider in context of active infections.	Monitor for signs of infection at 4 weeks and 4 weeks after dose increase.

CBC: Complete blood count, RFT: Renal function test, LFT: Liver function test, TB: Tuberculosis, NMSC: Non-melanoma skin cancers, HBV: Hepatitis B virus, MACE: Major adverse cardiovascular events, T: Tuberculosis

Table 6: Recommendations for discontinuation of oral abrocitinib in patients.

Criteria	Action	Condition for reinitiation
Serious or opportunistic infection	Discontinue treatment.	Reinitiation must be carefully considered after controlling the infection.
Hematologic Abnormalities	Condition	
- Platelet Count <50,000/mm^3	Discontinue abrocitinib and follow with CBC until platelet count >100,000/mm^3.	Do not initiate Abrocitinib in platelet count less than <150,000/mm^3
- ALC <500/mm^3	Temporarily discontinue treatment.	Restart when ALC returns above 500/mm^3.
- ANC <1,000/mm^3	Temporarily discontinue treatment.	Restart when ANC returns above 1,000/mm^3.
- Hb value <8 g/dL	Temporarily discontinue treatment.	Restart once Hb returns above 8 g/dL.

CBC: Complete blood count, Hb: Hemoglobin, ALC: Absolute Leucocyte Count, ANC: Absolute Neutrophil Count

Table 7: Adverse effects noted with oral abrocitinib.

Side effect	Details	Recommendations
Serious infections	Vulnerable to serious infections, including increased risk of herpes zoster.	Counsel patients about infection signs and advise contacting healthcare providers immediately.
Malignancies (including skin cancers)	Increased risk of malignancies, including skin cancers.	Periodic skin examinations; counsel on wearing protective clothing and using broad-spectrum sunscreen.
MACE	Increased risk of MI, stroke, and other cardiovascular issues.	Baseline monitoring; counsel on smoking cessation and cardiovascular risk factors. Warn about symptoms like chest discomfort, slurred speech, and lightheadedness.
DVT and PE	Increased risk of DVT and PE.	Counsel on signs such as leg swelling or pain, chest or back pain, and shortness of breath.
Retinal detachment	Retinal detachment reported during drug trials for AD.	Advise immediate reporting of vision blurring or difficulty.
Live vaccines	Live vaccines are not recommended during treatment, or immediately before starting the drug.	Avoid live vaccines while taking abrocitinib.

MACE: Major adverse cardiovascular events, DVT: Deep vein thrombosis, PE: Pulmonary embolism, AD: Atopic dermatitis, MI: Myocardial Infarction

CONCLUSION

This review suggests that abrocitinib holds potential as a treatment for a range of dermatological conditions beyond AD. However, it is important to recognize the limitations of the available evidence. Most of the current data come from case reports and small case series, which limits the ability to generalize findings and draw firm conclusions regarding its safety and effectiveness. Therefore, these preliminary results should be viewed with caution. To gain a more thorough understanding of abrocitinib's potential in treating various skin conditions, future research should prioritize large-scale, randomized controlled trials. Critical areas for further investigation include assessing the long-term efficacy and safety of abrocitinib, as well as comparing it directly to established treatments. Such studies are essential to confirm these initial findings and assess the broader applicability of abrocitinib in clinical dermatology. Until such evidence becomes available, clinicians should use their clinical judgment carefully when considering the off-label use of abrocitinib, given its exploratory nature.

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