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CASE SERIES

Abrocitinib for Head and Neck Dermatitis: Case Series and Literature Review

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Background: Head and neck dermatitis (HND) is a recalcitrant subtype of atopic dermatitis (AD). Despite conventional therapies, including topical corticosteroids, calcineurin inhibitors, and biologics such as dupilumab, many patients remain refractory to treatment. Janus kinase (JAK) inhibitors, such as abrocitinib, offer a novel therapeutic approach targeting multiple inflammatory pathways implicated in HND.

Purpose: To evaluate the efficacy of abrocitinib in refractory HND and compare its effectiveness with other JAK inhibitors (upadacitinib, baricitinib).

Patients and Methods: We retrospectively analyzed three patients with HND treated with abrocitinib (100–200 mg/day). Treatment response was assessed using Eczema Area and Severity Index (EASI) scores and the Investigator's Global Assessment (IGA) of the head and neck. A PubMed literature review was conducted to compare abrocitinib with other JAK inhibitors in HND.

Results: All three abrocitinib-treated patients achieved rapid skin lesion relief within 1–2 weeks and near-complete lesion clearance by Week 8. These results are consistent with clinical trial data highlighting the strong early efficacy of JAK inhibitors for head and neck lesions. While abrocitinib and upadacitinib demonstrated robust clinical responses, baricitinib showed comparatively weaker efficacy in the head and neck.

Conclusion: JAK inhibitors, particularly abrocitinib and upadacitinib, may be considered as first-line therapies for refractory HND. They offer both rapid symptom relief and sustained efficacy.

Keywords: head and neck dermatitis, atopic dermatitis, abrocitinib, JAK inhibitors

Introduction

Head and neck dermatitis (HND) is a clinically distinct subtype of AD that predominantly affects seborrheic regions, notably the head, face (with characteristic involvement of eyelids and lips), neck, and upper trunk. It is characterized by persistent and recurrent symptoms, even during periods of general AD remission.[1] Two main phenotypic presentations have been described: a localized, adolescent-onset form frequently associated with Malassezia colonization, and a generalized, adult-onset variant exhibiting diffuse dermatitis, suggesting divergent underlying pathophysiological mechanisms.[2] The pathogenesis of HND arises from multifactorial interactions, including genetic alterations in innate immunity,[3] inherent anatomical susceptibility, and environmental exposures.[4,5] A compromised skin barrier facilitates colonization by Malassezia spp.[6,] which exacerbates inflammation through Th17/Th2-type immune responses.[7,8] These mechanisms collectively contribute to the chronic and refractory course of HND.

Currently, HND remains a therapeutic challenge due to its refractory nature and sensitivity of the affected area. While topical therapies such as corticosteroids and calcineurin inhibitors are limited by efficacy issues or safety concerns, systemic treatments have become increasingly central to the management of refractory HND.

Abrocitinib, a recently approved oral JAK1 inhibitor for moderate-to-severe AD in adults,[9] is characterized by its rapid onset of action—particularly in relieving pruritus, often within the first 1–2 weeks of treatment.[10] Herein, we report three cases of treatment-resistant HND that showed notable improvement following abrocitinib therapy.

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Materials and Methods

We retrospectively collected a case series of 3 patients with HND diagnosed in the outpatient clinic of the Department of the First Affiliated Hospital of Chongqing Medical University, from April 2022 to December 2024. We included patients diagnosed with AD exhibiting a prominent HND phenotype who had been treated with abrocitinib for at least 8 weeks, with the additional requirement that clinical high-resolution photographs of the head and neck region were available from both baseline and at least two follow-up visits to allow for retrospective assessment of EASI and IGA scores. We excluded only those cases with insufficient treatment duration or incomplete key outcomes or images. Clinical information was obtained from medical records and clinical follow-ups. Treatment effectiveness was retrospectively measured by the Investigator Global Assessment (IGA) and Eczema Area and Severity Index (EASI) score of the head and neck by one trained physician based on clinical photographs before and during treatment with abrocitinib. This study included only three cases, with a limited sample size and without statistical analysis, which restricts the generalizability and reliability of the findings. Further studies with larger cohorts are needed to validate these results. To review the current literature concerning the usage of abrocitinib or JAK inhibitors for HND, a PubMed search was conducted with the Mesh terms ("head and neck dermatitis") AND ("abrocitinib" OR "Janus kinase inhibitors"). This resulted in a total of 3 articles about abrocitinib and 14 articles about Janus kinase inhibitors; after reading titles and abstracts, 8 articles were selected as relevant for the topic.

Results

A total of three patients were included in this study. Table 1 summarizes their baseline characteristics, along with post-treatment improvements in EASI and IGA scores of the head and neck, and records of adverse events.

Case Series

A 20-year-old female presented with refractory periorbital and neck eczema with effusion, conjunctivitis, and unbearable pruritus for more than 15 years. She was diagnosed with AD in infancy and had received systemic antihistamines, potent topical corticosteroids (TCS), and topical calcineurin inhibitors (TCI). Still, no improvement was achieved in facial and

Table 1 Clinical Data of HND Patients Treated with Abrocitinib

	Patient 1	**Patient 2**	**Patient 3**
Sex	Female	Male	Female
Age (Years)	20	21	30
Course of disease (Years)	Infancy (15+)	Middle Childhood (10+)	Middle Childhood (20+)
Comorbidities	None	None	None
Allergic comorbidities	Allergic conjunctivitis	None	Allergic conjunctivitis
			Allergic rhinitis
			Asthma
Previous treatments	Topical treatments (TCS and TCI)	Topical treatments (TCS and TCI)	Topical treatments (TCS)
	Systemic antihistamines	Systemic antihistamines	Systemic antihistamines
		Systemic corticosteroids	Systemic corticosteroids
		Dupilumab	Tofacitinib
Abrocitinib Treatment Duration (weeks)	8	80	90

(*Continued*)

Table 1 (Continued).

	Patient 1	**Patient 2**	**Patient 3**
EASI scores/ IGA scores			
Baseline	1.3/2	3.8/3	3/3
2w	0.3/1	0.25/1	None
8w	0/0	1.05/2	0/0
Adverse events	None	Urinary Tract Infection	Folliculitis
		Upper respiratory tract infection	Upper respiratory tract infection
			Herpes simplex virus

neck redness and itch (shown in Figure 1a). Abrocitinib (100 mg/d) was started, with rapid control of itch. Face and neck lesions showed significant improvement within 2 weeks (shown in Figure 1b), and remission persisted after 8-week follow-up (shown in Figure 1c). Finally, the patient discontinued abrocitinib due to financial reasons. Owing to the patient's decision to decline laboratory monitoring, no adverse events or discomfort were reported throughout the course of therapy.

A 21-year-old male with moderate AD and no history of allergy presented with pruritus, edema, and scales on his face, neck, hands, front of the thigh, and popliteal flexures. His condition had been present since childhood, and systemic antihistamines and TCI had been unsuccessful. The patient started with dupilumab 1 year before, with a good response, but face and neck redness with severe itch did not improve. Treatment with TCS, TCI, and systemic corticosteroids induced only a minimal response for 6 months. Subsequently, the patient was switched to abrocitinib therapy. Changes in disease severity scores (EASI and IGA) during treatment and the details of drug dose reduction are shown in Figure 2A

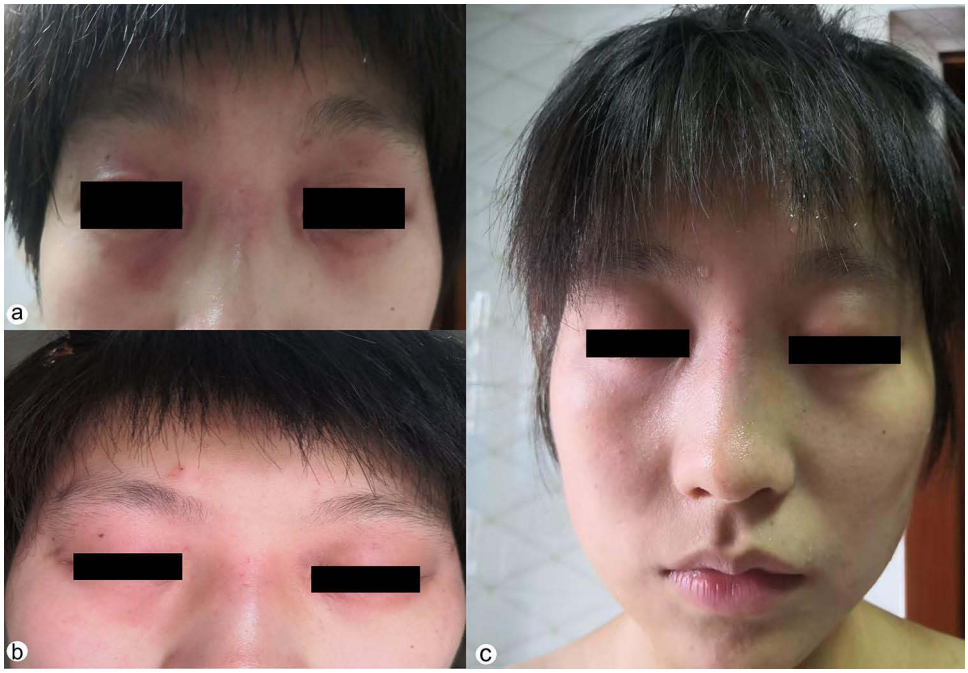

Figure 1 Clinical improvement of patient 1 with HND on abrocitinib. (**a**) before treatment: Refractory Periorbital eczema in HND patients. (**b** and **c**) Clinical remission after 2 weeks (**b**) and 8 weeks (**c**) of abrocitinib.

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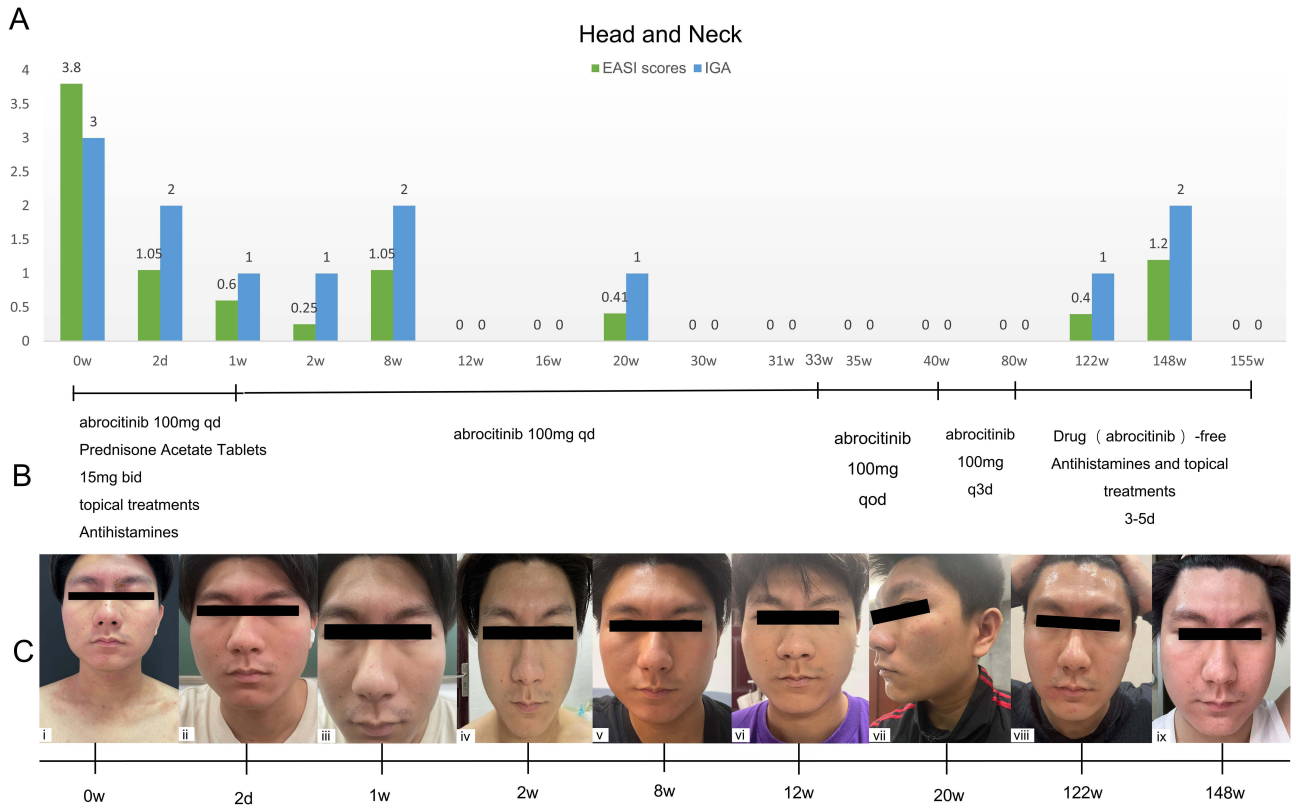

Figure 2 Clinical improvement of patient 2 with HND on abrocitinib. (**A)** The timeline of the EASI scores and IGA of head and neck during treatment with abrocitinib. (**B**) The detailed of tapering protocol. (**C**) The Clinical appearance of the patient 2 before and after of abrocitinib treatment. **(i)** Clinical appearance of the head and neck before treatment. **(ii–vii)** Clinical appearance of the head and neck during treatment. **(viii–ix)** Clinical appearance of the head and neck following treatment discontinuation.

and B, respectively. The patient was initially treated with a combination of abrocitinib (100 mg/day) and prednisone (30 mg/day); facial atopic lesions and itch were dramatically improved within 1 week (shown in Figure 2C.i–iii). The patient was then maintained with abrocitinib alone for 2 weeks with EASI90 remission (shown in Figure 2C.iv). The patient exhibited disease recurrence at weeks 8 and 20. Following continued treatment with abrocitinib 100 mg once daily, clinical remission was successfully achieved (shown in Figure 2C.v–vii). After 33 weeks, the dosage was tapered to every other day (QOD) administration. Further dose reduction to every third day (Q3D) was implemented following 40 weeks. Complete discontinuation occurred after 80 weeks; the patient remained on sustained improvement in HND symptoms. The patient has maintained clinical remission for 75 weeks with only occasional mild recurrences (shown in Figure 2C.viii–ix) that were effectively managed by antihistamines and topical medications for a few days and did not significantly impact the quality of life. During the treatment period, the patient experienced one episode of urinary tract infection and acute upper respiratory tract infection. No abnormalities in complete blood count, liver function, or coagulation parameters were observed, and no hepatitis B virus infection was detected.

A 30-year-old female presented with a 20-year history of refractory pruritus, erythema, and xerosis predominantly affecting the head, neck, and upper chest. Similar but less severe cutaneous manifestations were also noted on the trunk and extremities. She had comorbid allergic rhinitis, asthma, and conjunctivitis, along with a positive family history of eczema in both parents. She was diagnosed with AD and previously treated with topical corticosteroids, oral antihistamines, tofacitinib, and systemic glucocorticoids, but the condition remained poorly controlled. She achieved complete disease control after 4 months of oral abrocitinib 100 mg once daily (QD) at another hospital, but the disease relapsed after self-discontinuation. Subsequently, the patient re-initiated abrocitinib therapy at our outpatient clinic. Changes in disease severity scores (EASI and IGA) during treatment and the details of drug dose reduction are shown in Figure 3A and B, respectively. We administered abrocitinib 200 mg QD, observing significant improvement in erythema and pruritus within 1 week (shown in Figure 3C.i–ii). Complete remission was maintained by week 8 (shown in Figure 3C.

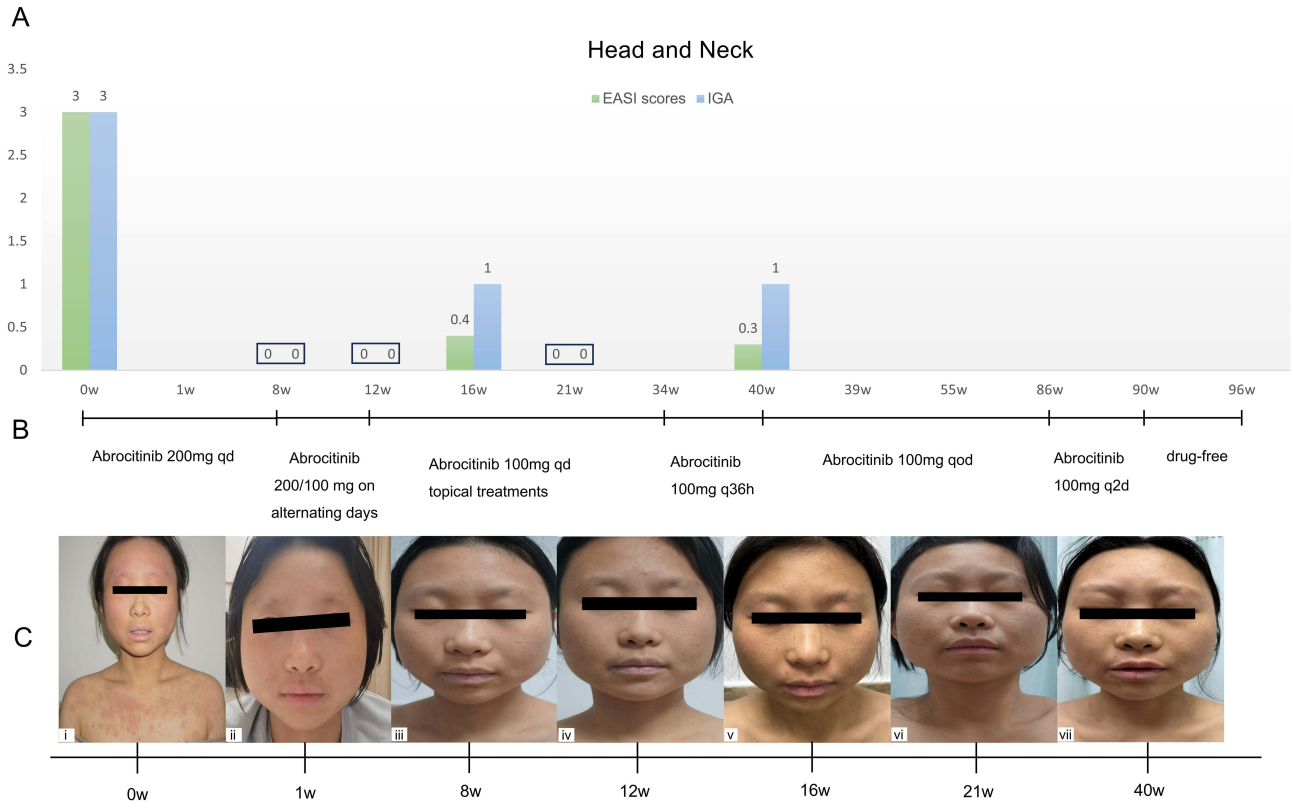

Figure 3 Clinical improvement of patient 3 with HND on abrocitinib. (**A**) The timeline of the EASI scores and IGA of head and neck during treatment with abrocitinib. (**B**) The detailed of tapering protocol. (**C**) The Clinical appearance of the patient 3 before and after of abrocitinib treatment. **(i)** Clinical appearance of the head and neck before treatment. (ii–vii) Clinical appearance of the head and neck during treatment.

iii), prompting dose reduction to 200 mg/100 mg. Alternating days for 4 weeks (shown in Figure 3C.iv) then 100 mg QD (weeks 13–46) before progressive tapering until discontinuation at week 90, as the detailed tapering protocol is shown in Figure 3B. The patient exhibited disease recurrence at weeks 16 and 40. Following continued treatment with abrocitinib, clinical remission was successfully achieved (shown in Figure 3C.v–vii). During the 6-week post-treatment follow-up, only minor self-limited recurrences (2–3 days) occurred, all managed with topical therapies without significant quality of life impact. During the treatment period, the patient developed herpes simplex virus infection, acute upper respiratory tract infection, and folliculitis. No abnormalities were detected in laboratory tests, including liver function, renal function, coagulation parameters, T-SPOT.TB, hepatitis B virus infection, syphilis, or HIV.

Literature Review

To our knowledge, no studies have been reported specifically investigating the efficacy of abrocitinib or other JAK inhibitors (JAKi) in HND,but. To further elucidate therapeutic strategies for HND, we systematically reviewed clinical trials and real-world evidence evaluating the improvement of head and neck lesions in AD patients receiving abrocitinib or JAK inhibitors.

The available data suggest distinct efficacy profiles among JAK inhibitors (abrocitinib, upadacitinib, and baricitinib) and the IL-4/IL-13 inhibitor dupilumab in treating head and neck atopic dermatitis (Table 2). The Phase 3 JADE COMPARE trial revealed distinct therapeutic profiles among abrocitinib 100 mg (n=238), 200 mg (n=226), and dupilumab 300 mg (n=242) in atopic dermatitis management.[11] Early efficacy analysis demonstrated abrocitinib 200 mg's superiority, with significantly greater improvement in EASI head and neck scores at Week 2 (52.5% vs 39.9% for dupilumab and 47.8% for abrocitinib 100 mg), attributable to its potent JAK1-mediated anti-inflammatory effects.[12] This contrasts with dupilumab's delayed onset, reflecting the gradual immunomodulation of IL-4/IL-13 pathway inhibition.[13] Long-term outcomes at Week 16 showed comparable efficacy between abrocitinib 200 mg (76.6%) and dupilumab

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Table 2 Studies Describing Jaki in Head and Neck Lesions of AD Found During Literature Search

Article	Diagnosis	Previous Therapy	Name	Drug Target	Patients, n	Management and Dose	Combination Therapy	Time (W)	Reduction of EASI (%)	EASI-50 (%)	EASI-75 (%)	EASI-90 (%)	EASI-100 (%)
Jacob P Thyssen[13] 2024	mod-sev AD	NA	Upadacitinib	JAK1	342	30 mg/d	NA	1	44.8		25.3	13.6	8.9
								2	65.5		51.9	27.8	14.6
								4	72.7		66.2	44.1	25.5
								8	77.4		69.5	54.7	37.2
								12	79.6		67.5	53.0	36.9
								16	80.4		66.0	54.2	40.4
								20	80.5		63.5	53.7	41.3
								24	79.8		57.2	47.9	36.6
			Dupilumab	IL-4 and IL-13	331	300mg/2w	NA	1	25.0		11	5.3	3.7
								2	46.6		24.0	12.1	8.2
								4	57.3		39.0	21.8	14.3
								8	65.1		47.4	32.5	20.6
								12	70.4		51.1	37.0	23.0
								16	78.1		54.0	38.9	26.1
								20	78.4		55.2	40.5	27.0
								24	80.6		53.4	39.7	29.7
Teppei, Hagino[14] 2024		Upadacitinib 15mg: responses insufficient	Upadacitinib	JAK1	21	30mg/d	Topical treatments	4	65.2*				
								12	72.1*				
Teppei Hagino[15] 2023		Baricitinib 4mg: responses insufficient	Upadacitinib	JAK1	20	30mg/d	Topical treatments	4	42.2*				
								12	69.3*				
Teppei, Hagino[16] 2023		NA	Upadacitinib	JAK1	72	15mg/d	Topical treatments	4			45.3		
								12			42.2		
Teppei, Hagino[17] 2023		NA	Upadacitinib	JAK1	65	15mg/d	Topical treatments	4	75*		50.7*	23.7*	8.4*
								12	74.7*		52.5*	30.6*	10.2*
								24	86*		60.4*	33.1*	12.4*

Teppei, Hagino[18] 2023		NA	Baricitinib	JAK 1/2	36	4mg/d	Topical treatments	12	56.9				
Andreas, Wollenberg[19] 2022		NA	Baricitinib (BREEZE-AD1 and AD2)	JAK 1/2		1mg/d	NA	16		21	14		
						2mg/d		16		26	20		
						4mg/d		16		32	20		
			Baricitinib (AD5)			1mg/d		16		17	13		
						2mg/d		16		30	25		
			Baricitinib (AD4)			1mg/d		16		42	25		
						2mg/d		16		49	27		
						4mg/d		16		42	28		
			Baricitinib (AD7)			2mg/d		16		59	39		
						4mg/d		16		59	40		
Andrew Alexis[10] 2022		NA	Abrocitinib	JAK1	238	100mg/d	Topical treatments				57d	110d	
								2	47.8				
								16	67.6				
			Abrocitinib	JAK1	226	200mg/d					29d	57d	
								2	52.5				
								16	76.6				
			Dupilumab	IL-4 and IL-13	242	300 mg/2w					57d	112d	
								2	39.9				
								16	74.2				

Notes: *As the original literature did not provide numerical data, graphical data were extracted and analyzed using WebPlotDigitizer (version 4.6). Each measurement was repeated three times, and the mean value was calculated.
Abbreviations: mod-sev AD, Moderate-to-Severe AD; NA, Not available.

(74.2%), both numerically exceeding abrocitinib 100 mg (67.6%), indicating dose-dependent JAK inhibitor effects while achieving biologic-level efficacy. Notably, the median time to EASI-75 response in head and neck regions was 29 days for abrocitinib 200 mg versus 57 days for both abrocitinib 100 mg and dupilumab, with similar patterns observed for EASI-90 responses (57 vs 110[15mg] and 112[dupilumab] days, respectively). These kinetics demonstrate abrocitinib 200 mg's rapid onset advantage, whereas dupilumab's eventual therapeutic parity suggests late compensatory mechanisms.

Similarly, Upadacitinib demonstrated dose-dependent efficacy in the treatment of head and neck, with the 30 mg dose (n=342) showing superior early response compared to both the 15 mg (n=137 pooled) and dupilumab (n=331).[14,16,17] At Week 4, EASI-75 response rates were 66.2%[16] for upadacitinib 30 mg versus 45.3[17]-50.7[14] % for the 15 mg dose and 39.0%[16] for dupilumab. This early advantage was maintained at Week 12, with response rates of 67.5%,[16] 42.2[17]–52.5[14] %, and 51.1%,[16] respectively. However, by Week 24, the therapeutic profiles converged, showing comparable efficacy between upadacitinib 30 mg (57.2%[16]), 15 mg (60.4%[14]), and dupilumab (57.2%[16]). Notably, upadacitinib 30 mg showed particular effectiveness in refractory cases, achieving EASI reductions of 65.2% at Week 4 and 72.1% at Week 12 in Upadacitinib 15mg-refractory patients (n=21),[15] and 42.2% to 69.3% over the same period in baricitinib 4mg-refractory cases (n=20).[19] While the 15 mg dose initially showed lower response rates, its long-term performance at 24 weeks (60.4% EASI-75 response with 86% mean EASI reduction) was comparable to the higher 30mg dose (57.2% EASI-75 response with 79.8% reduction),[14,16] suggesting that the dose-dependent differences may become less pronounced over extended treatment periods. These findings indicate that while the 30 mg dose provides a more rapid initial improvement, both doses ultimately achieve similar long-term outcomes. However, as these observations come from non-comparative studies, further head-to-head clinical trials are needed to confirm these dose-response relationships.

Baricitinib, while effective, showed more modest improvements in head and neck, with response rates varying across trials (eg, 20–40% EASI-75 improvement at Week 16 with 4 mg/day, depending on study design).[18,20] Notably, the proportion of patients achieving EASI-75 was significantly lower compared to upadacitinib, abrocitinib, and dupilumab.

Discussion

Head and neck dermatitis, a severe and refractory subtype of AD, poses unique therapeutic challenges due to its complex pathophysiology and limited response to conventional therapies. Our study provides the first case-series evidence demonstrating the rapid and sustained efficacy of abrocitinib, a selective JAK1 inhibitor, in patients with multidrug-resistant HND who had previously failed therapies including topical calcineurin inhibitors, systemic corticosteroids, tofacitinib, and biologics such as dupilumab. These findings align with emerging evidence supporting JAK inhibitors as promising therapeutic options for HND, particularly in cases resistant to IL-4/IL-13 pathway modulation.

JAK inhibitors represent an effective option for HND, broadly suppressing cytokine signaling (eg, IL-4, IL-13, IL-31, IL-33), and may disrupt this inflammatory cascade more effectively than targeted biologics.[21] Our patients' rapid relief (within 1–2 weeks) and lesion resolution (within 2–8 weeks). Notably, patients maintained stable efficacy and favorable safety profiles throughout 70–80 weeks of continuous therapy, with one case sustaining basic remission for 75 weeks post-treatment discontinuation. Our observations are supported by clinical trial data highlighting the superior early efficacy of JAK1 inhibitor (abrocitinib, upadacitinib) over dupilumab for the head and neck. These findings suggest that JAK1 inhibitor's rapid onset—attributed to direct intracellular inhibition of pro-inflammatory signals—may be particularly advantageous for HND, where pruritus and visible lesions significantly impact the quality of life.[22]

The dose-dependent efficacy of JAK inhibitors correlates with their superior early efficacy compared to dupilumab in HND, highlighting the importance of optimal dosing for initial symptom control. However, long-term (16–24 weeks) outcomes reveal diminishing therapy differences. Notably, all three therapies - abrocitinib 200 mg, upadacitinib, and dupilumab - achieve comparable clinical endpoints with extended treatment. This pattern suggests that higher JAK inhibitor doses accelerate initial response, but their long-term efficacy may ultimately approximate dupilumab and the low-dose group.[14,16,17,20] In contrast, baricitinib 4 mg showed more modest efficacy for the head and neck, may stems from its dual JAK1/JAK2 inhibition profile, which results in less potent suppression of Th2-driven inflammation than selective JAK1 inhibitors like abrocitinib and upadacitinib.[18,23] The pathogenesis of HND is dominated by robust type 2 immunity coupled with frequent *Malassezia* colonization. These fungi thrive in sebum-rich environments and can further

aggravate skin barrier dysfunction and amplify local Th2/Th17 inflammation. Notably, emerging evidence indicates that *Malassezia* spp. (eg, *M. restricta*) may diminish the efficacy of both anti-IL4Rα agents and JAK1/2 inhibitors such as ruxolitinib.[24] Therefore, in HND—where type 2 inflammation and fungal superinfection are central—targeting the key cytokines via highly selective JAK1 inhibition may constitute a more rational and effective first-line therapeutic strategy. In contrast, dupilumab may remain a viable treatment for long-term HND due to its sustained efficacy profile.

Our study is limited by its small sample size and retrospective design. Future randomized controlled trials directly comparing these agents are warranted to establish their comparative efficacy and safety profiles. In parallel, mechanistic studies elucidating the effects of JAK inhibitors on the cutaneous microbiome and localized immune dysregulation are needed to refine patient-specific therapeutic strategies.

Ethics Approval and Informed Consent

Institutional approval was not required to publish the case details. The patient provided written informed consent for the publication of the case details, including the images, which were obtained from the patient.

Consent for Publication

The patients provided written informed consent for the publication of the case details, including the images, which were obtained from the patients.

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Disclosure

The authors report no conflicts of interest in this work.

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