

Mollusum Contagiosum, Warts and Condylomas during JAK Inhibitor Therapy for Atopic Dermatitis: a Retrospective Series of 17 Cases

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Janus kinase inhibitors (JAK-i) represent a major therapeutic advancement for inflammatory dermatoses such as atopic dermatitis (AD), alopecia areata and vitiligo. By blocking intracellular signal transduction of multiple cytokines, including type I and II interferons, they exert potent anti-inflammatory effects but may also impair antiviral immune defence (1). Although herpes-zoster reactivation and other varicella-zoster virus (VZV) infections are well-recognized adverse events, data on nonherpetic cutaneous viral infections remain limited (2). We report a multicentre retrospective case series of 17 patients who developed cutaneous viral infections (mollusum contagiosum (MC), warts or condylomas) during JAK-i treatment for AD.

Our cohort comprised 17 patients (13 males, 4 females; mean age 35.6±11.4 years, median 35 years, range 13–63 years) treated for AD with JAK-i in 9 dermatology centres in France and Switzerland. Upadacitinib was the most prescribed agent (94.1%, $n=16$; 14 patients at 30 mg/day, 2 at 15 mg/day). Nine patients had received previous immunosuppressive

therapy, most commonly ciclosporin and prior JAK-i. Patients were not systematically screened for underlying immunodeficiency; however, no clinical history suggestive of primary or secondary immunodeficiency was reported. Among the cohort, 6 patients (35.3%) had a documented history of MC, warts or condylomas prior to JAK-i initiation. Cutaneous viral infections developed at a median of 6 months (range: 1–24 months, mean 9.2±7.1 months) after JAK-i initiation. The predominant infection types were condylomas (47.1%, $n=8$), warts (47.1%, $n=8$) and mollusum contagiosum (41.2%, $n=7$), with 23.8% ($n=5$) of patients exhibiting multiple concurrent infections (**Table I**). Lesion burden varied considerably according to infection type, with most patients presenting either 10–50 lesions ($n=5$) or fewer than 10 lesions; however extensive disease (>50 lesions) occurred in 3 patients, predominantly in those with condylomas or mollusum contagiosum (**Fig. 1**).

JAK-i therapy was modified in 11 patients (64.7%), including discontinuation ($n=5$, 29.4%), dose reduction ($n=3$, 17.6%) or switching to another JAK-i ($n=3$,

Table I. Clinical characteristics and outcomes of 17 patients with cutaneous viral infections during Janus kinase inhibitor (JAKI) therapy for atopic dermatitis

Patient	Age/ Sex	Prior immunosuppression	Prior viral infection	JAKI (dose, mg/day)	Time to infection (months) ^a	Infection type	Lesion count	JAKI management
1	32 /M	CsA, BARI, ABRO	No	UPA (30)	6	Mollusum contagiosum	Unknown	Switched JAKI
2	63 /M	None	Mollusum contagiosum	UPA (30)	3	Mollusum contagiosum	Unknown	Continued
3	30 /F	None	No	UPA (30)	15	Mollusum contagiosum	>50	Stopped
4	44 /M	None	Mollusum contagiosum, condyloma, warts	UPA (30)	6	Condyloma, warts	Unknown	Stopped
5	36 /M	None	Herpes zoster, warts	UPA (30)	6	Mollusum contagiosum, warts	10–50	Reduced
6	39 /M	None	No	UPA (30)	9	Condyloma	10–50	Reduced
7	28 /M	None	No	UPA (15)	2	Mollusum contagiosum	>50	Continued
8	44 /M	BARI, ABRO	No	UPA (30)	11	Condyloma	>50	Stopped
9	35 /M	CsA, BARI, ABRO	No	ABRO (200)	24	Condyloma, warts	<10	Stopped
10	31 /M	CsA	Condyloma	UPA (30)	1	Condyloma	<10	Continued
11	53 /M	CsA, MTX	Herpes zoster, Condyloma	UPA (30)	1	Condyloma, warts	10–50	Switched JAKI
12	22 /F	CsA	No	UPA (30)	15	Condyloma	10–50	Stopped
13	13 /F	MTX, OCS	Warts	UPA (15)	12	Warts	<10	Continued
14	31 /M	BARI	No	UPA (30)	9	Mollusum contagiosum	10–50	Switched JAKI
15	31 /F	BARI	No	UPA (30)	6	Warts	<10	Reduced
16	35 /M	None	No	UPA (30)	6	Warts	<10	Continued
17	38 /M	None	No	UPA (30)	24	Mollusum contagiosum, condyloma, warts	10–50	Continued

^aTime to infection represents the interval from JAK-i initiation to viral infection onset. Reduced: Upadacitinib (UPA) switched to 15 mg/day.

ABRO: abrocitinib; ADA: adalimumab; BARI: baricitinib; CsA: ciclosporine; HSV: herpes simplex virus; MTX: methotrexate; OCS: oral corticosteroids.



Fig. 1. Cutaneous viral infections arising during treatment with upadacitinib in patients with atopic dermatitis. Giant plantar warts (left); extensive molluscum contagiosum eruption (right)

17.6%) in response to viral infections. Therapeutic interventions included cryotherapy, topical imiquimod, topical keratolytic agents and surgical excision. Among the 5 patients in whom JAK-i were discontinued, cutaneous viral lesions completely regressed in 4 cases, including 3 patients with disseminated MC and 1 with condylomas.

JAK-i may increase susceptibility to viral infections through suppression of type I interferon signalling in epidermal and mucosal tissues, a pathway critical for keratinocyte antiviral responses and cytotoxic lymphocyte activation (3).

A cohort study of 1793 AD patients reported higher overall infection rates with JAK-i treatment compared to biologic treatment, particularly for herpes infections (4). Beyond herpesviruses, isolated case reports have described MC and human papillomavirus (HPV)-related warts arising during JAK-i therapy. Notably, a case of disseminated MC was reported in a patient receiving ruxolitinib, a JAK1/2 inhibitor, highlighting the role of interferon pathway blockade in impaired viral clearance (5).

Most recently, several reports documented the occurrence of HPV-related warts following JAK-i initiation (6–8). Collectively, these observations support a mechanistic link between JAK inhibition and reduced cutaneous antiviral immunity beyond herpesvirus reactivation.

Our retrospective series suggests that JAK-i may predispose patients to opportunistic cutaneous viral infections particularly HPV-related condylomas, warts, and MC. Patients with AD and/or a prior history of viral skin infections may represent a population at increased risk. Clinicians should therefore remain alert to the development of new or progressive viral skin lesions during JAK-i therapy.

The main limitations of this study are the relatively young cohort and the predominance of upadacitinib

exposure, limiting the generalizability of our findings to older patients and to other JAK inhibitors.

We recommend patient education, baseline assessment of viral infection history and periodic skin examination during JAK-i therapy. Reporting such cases is essential to better define their incidence, risk factors, and outcomes across dermatologic indications.

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Ethics committee: All procedures adhered to the ethical standard set by the Helsinki Declaration. All patients provided written informed consent for images. Identifying information has been removed.

Conflict of interest: MJ and EL have received personal fees for consulting and/or speaking activities or travel grants and congress registration support from AbbVie, Almirall, AstraZeneca, Galderma, Incyte, LEO Pharma, Lilly, Pfizer, Sanofi. These relationships are outside the submitted work. GR has received personal fees for speaking activities and/or travel grants and congress registration support from Sanofi, Johnson & Johnson, Pfizer and UCB. These relationships are outside the submitted work. EM has undertaken activities as a paid consultant, adviser or speaker for AbbVie, Almirall, Amgen, Bioline, Janssen-Cilag, Leo Pharma, Lilly, Novartis, Sanofi, and UCB. AN has served as an investigator, speaker, and/or advisor for AbbVie, Almirall, Sanofi Regeneron, Pierre Fabre, Janssen Cilag, Celgene, Lilly, Leo Pharma, L'Oréal, Galderma, Leo Pharma, Medac, Novartis. NRP has served as an investigator for AbbVie and Pierre Fabre and has received personal fees for speaking activities and/or for travel grants and congress registration support from Sanofi and ALK. AS has received support or personal fees for consulting and/or speaking activities from Novartis, Sanofi, AbbVie, Lilly, LEO Pharma, Pfizer, Bioprojet, Almirall and research grants from Sanofi. MT has served as an investigator, speaker, and/or advisor for AbbVie, Almirall, Sanofi Regeneron, Pierre Fabre, Janssen Cilag, Lilly, Leo Pharma, Galderma, Leo Pharma, Medac, Novartis.

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