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REVIEW

Dermatologic Comorbidities Associated with Atopic Dermatitis Towards a Shared Therapeutical Approach: A Narrative Review

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ABSTRACT

Introduction: Atopic dermatitis (AD) is a chronic inflammatory skin condition commonly associated with other dermatologic comorbidities, which can complicate management and affect treatment outcomes. This review aims to analyse the dermatologic comorbidities of AD, their underlying mechanisms, and therapeutic implications, with a focus on their management in clinical practice.

Methods: A narrative review was conducted by searching PubMed, Embase, Cochrane and ClinicalTrials.gov, using terms related to AD and its comorbidities, including allergic contact dermatitis, alopecia areata, prurigo nodularis, psoriasis, hidradenitis suppurativa and chronic spontaneous urticaria.

Results: The literature highlights a strong association between AD and several dermatologic comorbidities, including allergic contact dermatitis, alopecia areata, chronic spontaneous urticaria, hidradenitis suppurativa, psoriasis, prurigo nodularis and vitiligo. Promising therapeutic effects were observed with JAK inhibitors, dupilumab and other biologics across multiple comorbid condition.

Conclusion: Recognizing comorbidities in AD is critical for effective management. Tailored therapies targeting both AD and its comorbidities, based on shared immunological mechanisms, may improve outcomes. Further research is needed to optimize treatment strategies and explore combination therapies for patients with both AD and comorbid dermatological conditions.

Keywords: Atopic dermatitis; Biologic treatment; Small molecules; Comorbidities

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Key Summary Points

Atopic dermatitis is frequently associated with several dermatologic comorbidities, including vitiligo, alopecia areata, psoriasis, prurigo nodularis and others, which may complicate its clinical management.

These conditions often share immunopathological mechanisms with atopic dermatitis.

Recognizing these associations is crucial to adopt an integrated, personalized therapeutic approach that avoids overlapping treatments.

Novel systemic treatments, such as JAK-inhibitors and biologic agents, offer promising options in managing both atopic dermatitis and its comorbidities.

Further research is needed to optimize shared treatment strategies for patients with atopic dermatitis and coexisting dermatologic diseases.

INTRODUCTION

Atopic dermatitis (AD) is one of the most prevalent inflammatory skin diseases, with both prevalence and incidence steadily increasing over the past decades [1–3]. There is a growing interest in the comorbidities of AD, which can impact the management of affected patients. AD comorbidities can be classified as atopic (e.g. asthma, rhinitis and food allergy) and non-atopic (including, among others, ocular, psychiatric, infectious, endocrine, autoimmune and cardiovascular diseases) [4]. Cutaneous comorbidities, which are part of the non-atopic group, comprise a heterogeneous set of inflammatory skin diseases that are clinically relevant because they can be positively or negatively influenced by the treatment of AD, particularly with biologics or small molecules. Furthermore, some therapeutic agents used to treat these comorbidities can induce or exacerbate AD. Thus, clinicians should be aware of this evidence when planning systemic treatment for AD. This narrative review will focus on allergic contact dermatitis (ACD), alopecia areata (AA), chronic spontaneous urticaria (CSU), hidradenitis suppurativa (HS), psoriasis (Pso), prurigo nodularis (PN) and vitiligo. These diseases partially share common immunological features with AD (Table 1) and, consequently, could be important in the choice of AD therapeutic approach.

MATERIALS AND METHODS

A comprehensive narrative review of the literature was conducted by searching the following electronic databases: PubMed, Embase, Cochrane Skin and ClinicalTrials.gov, up to 30 April 2025. The following keywords were used, alone or in combination: ‘atopic dermatitis’, ‘dermatologic comorbidities’, ‘allergic contact dermatitis’, ‘alopecia areata’, ‘vitiligo’, ‘prurigo nodularis’, ‘psoriasis’, ‘hidradenitis suppurativa’, ‘chronic spontaneous urticaria’, ‘biologic therapy’, ‘JAK inhibitors’. All article types were considered, including narrative and systematic reviews, meta-analyses, clinical trials, real-life studies, case series and relevant case reports. Studies were included if they addressed the pathophysiology, clinical association, or therapeutic management of dermatologic comorbidities in patients with AD. Only articles published in English were included. Editorials, conference abstracts and non-peer-reviewed articles were excluded. A manual review of reference lists from selected articles was also performed to identify additional relevant studies. A total of 80 manuscripts were included in our review. The quality of the selected studies was assessed as reported in Table 2. This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

Allergic contact dermatitis

The risk of ACD in patients with AD is increased, as demonstrated by a retrospective study on 15,737 patients who underwent patch tests, which showed that 54.8% of patients with had a positive test for ACD compared with 47.3% of non-AD patients ($p<0.0001$) [15]. Consequently,

Table 1 Cutaneous comorbidities and their possible immunological link to AD

Comorbidity	Possible immunological link to AD
Allergic contact dermatitis	Th2, Th17 and Th22 responses seem to play a role in ACD, sometimes depending on the allergen (e.g. rubber and fragrance promote Th2 activity with less Th1 and Th17 involvement, nickel is a potent inducer of the innate immune Th1, Th17 and Th22 pathways) [5]
Alopecia areata	Transcriptomic analyses of AA skin biopsies have shown increased Th2-related gene expression and elevated IL-13 levels in AA patients. Filaggrin gene mutations, a key risk factor for AD, and high serum immunoglobulin E (IgE) levels are linked to greater AA severity [6] In AA, clusters of CD8 + T cells accumulate around hair follicles, correlating with disease severity, and may also promote IgE production, worsening AD. Similarly, in AD, CD8 + T cells produce inflammatory cytokines like IL-17, IL-22 and IFN-γ, driving further skin inflammation [7]
Chronic spontaneous urticaria	Both conditions show a dominant Th2 immune response pathway, characterized by increased levels of IL-4, IL-5, IL-13 and IL-31. Similarly, mast cells and basophils play a crucial role in both AD and CSU, as well as elevated IgE being present in both conditions, despite the unclear role of IgE in AD [8, 9]
Hidradenitis suppurativa	Although the pathophysiology of HS currently remains partially obscure, an IL1b-IL23/Th17/IL17 pathway is implicated in the pathogenesis [94], while AD was classically characterized by a Th2 profile. However, Asian, paediatric and intrinsic types of AD involve Th17 [10, 11]
Prurigo nodularis	AD and PN share common pathogenic pathways, particularly Type 2 inflammation and chronic pruritus [12]
Psoriasis	Psoriasis is mainly an IL23-Th17-IL17 disease, while AD is Th2 skewed, associated with IL-4 and IL-13. Nevertheless, Asian, paediatric and intrinsic types of AD involve Th17 as well while the analysis of Pso susceptibility genes identified an odds ratio of 1.18 increase in IL-4/IL-13 signalling loci. Moreover, both diseases involve Th1 and Th22 even though the increased levels of IL-22 in both diseases might not be essential to either psoriasis or AD [11, 13]
Vitiligo	AD is primarily driven by Th2 activation and JAK/STAT signalling, with Th1 and Th17 also playing a role. Vitiligo, dominated by a Th1 response, involves IFN-γ–mediated JAK/STAT1 activation, leading to CD8 + T cell recruitment [14]

AA Alopecia areata, *ACD* Allergic contact dermatitis, *AD* Atopic dermatitis, *CSU* Chronic spontaneous urticaria, *HS* Hidradenitis suppurativa, *PN* Prurigo nodularis, *Pso* Psoriasis

considering that ACD can mimic AD [16], patch testing should be considered in adolescent or adult-onset AD, in patients with with worsening or more generalized dermatitis, when there is a lesional distribution that is atypical for AD, if the dermatitis is recalcitrant to topical therapy, prior to the initiation of systemic immunosuppressive therapy, when AD worsens with therapy, or rebounds quickly upon cessation of therapy [16] (Table 3). As regards treatment, despite allergen avoidance remains the mainstay of ACD therapy [15], a brief cycle of oral corticosteroids

Table 2 To assess the methodological quality of each study included for efficacy aeach study included for efficacy analysis, a grade of evidence was assigned using these criteria

Grade of evidence	
A1	Meta-analysis that includes at least one randomized clinical trial with a grade of evidence of A2; the results of the different studies included in the meta-analysis must be consistent
A2	Randomized, double-blind clinical study of high quality (e.g. sample-size calculation, flow chart of patient inclusion, ITT analysis and sufficient size)
B	Randomized clinical study of lesser quality, or other comparative study (e.g. non-randomized cohort or case–control study)
C	Non-comparative study
D	Expert opinion
Level of evidence	
1	Studies assigned a grade of evidence of A1, or studies that have predominantly consistent results and were assigned a grade of evidence of A2
2	Studies assigned a grade of evidence of A2, or studies that have predominantly consistent results and were assigned a grade of evidence of B
3	Studies assigned a grade of evidence of B, or studies that have predominantly consistent results and were assigned a grade of evidence of C
4	Little or no systematic empirical evidence; extracts and information from the consensus conference or from other published guidelines

In addition, a grading of the levels of evidence were used to provide an overall rating of the available efficacy data for the different treatment options

or systemic immunosuppressants can be used in severe ACD in patients with AD [17, 18] (Fig. 1). Concerning AD indicated treatment, the efficacy of dupilumab on ACD is controversial: some studies reported cases of refractory ACD improved following dupilumab therapy but, others reported development or worsening of ACD during dupilumab use [19–22].

Finally, since JAK inhibitors block the JAK–STAT pathway and regulate Th1, Th2 and Th17 responses, they may interfere with the elicitation phase of allergens [23]. As a result, JAK inhibitors could serve as a potential therapeutic option for ACD but may also contribute to false-negative results in patch testing [24].

Alopecia areata

AA is an autoimmune disorder, typically characterized by well-demarcated patches of non-scarring hair loss on the scalp and other hair-growing regions. The lifetime incidence of AA is reported to be around 2% globally, with the onset usually occurring before the age of 40 years, and an unpredictable course. While the exact pathophysiology remains unclear, a combination of genetic predisposition, immune system dysregulation and environmental triggers is believed to play a key role in its development [25]. Holmes et al. have shown

Table 3 Clinical scenarios suggesting concomitant allergic contact dermatitis (ACD) and atopic dermatitis (AD) or ACD mimicking AD

Clinical Scenario	Description	Rationale
Atypical onset of AD during adolescence or adulthood	AD developing or worsening at ≥ 18 years of age	Classical AD typically begins in childhood; adult-onset should raise suspicion for ACD
Unusual AD lesion distribution	Predominantly involving hands, feet, face, periocular areas, or photo-exposed sites	ACD often affects contact or photo-exposed areas
Recalcitrant dermatitis despite standard topical therapy	Lack of response to topical corticosteroids or calcineurin inhibitors	Concomitant ACD may impair therapeutic response
AD worsening with standard therapies or rapid rebound after treatment cessation	Flare after immunosuppressive therapy withdrawal or paradoxical worsening	ACD may reactivate independently from AD inflammation control
Suspected association with environmental or occupational exposure	Dermatitis worsening with cosmetics, metals, textiles, gloves or other allergens	Typical feature of exposure-driven ACD

ACD Allergic contact dermatitis, *AD* Atopic dermatitis

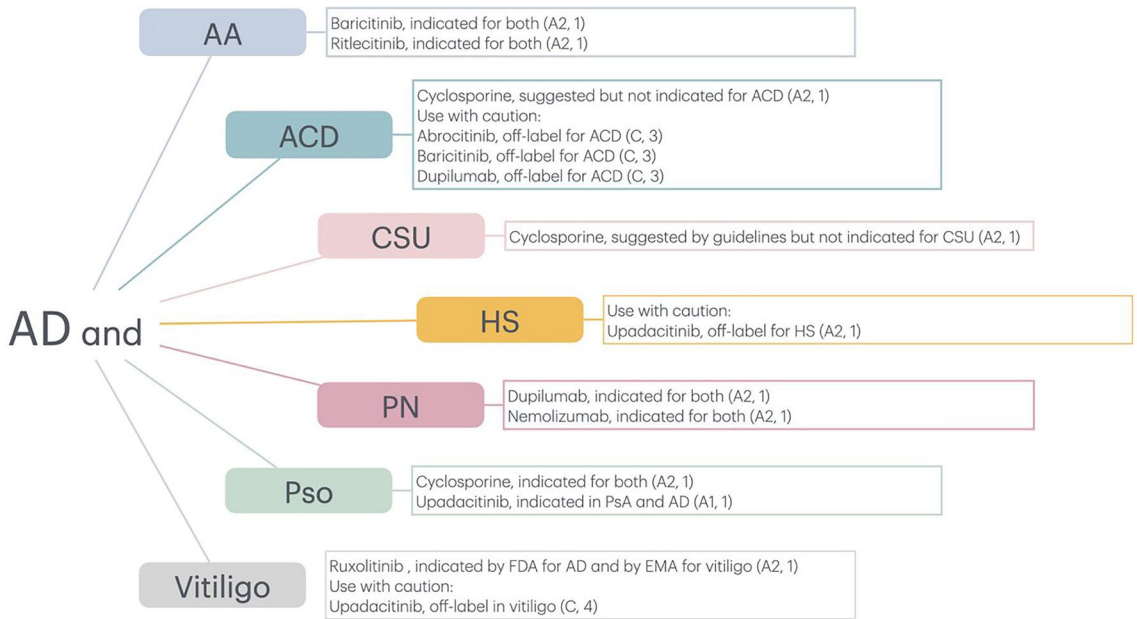

Fig. 1 Therapeutical management of AD and skin comorbidities according to EMA/FDA approval and recent literature evidence

a clinically significant increase in the burden of atopic and autoimmune comorbidities among patients with AA [26]. Atopic conditions, such as AD, allergic rhinitis and asthma, are frequently reported comorbidities in AA [27]. Goh et al. highlighted that patients with AA who also had AD, or a family history of AD were statistically significantly more likely to develop severe forms of the disease, such as alopecia totalis and alopecia universalis, compared with those without a history of AD [28].

Currently, two JAK inhibitors are approved for the treatment of either AA and AD: baricitinib (JAK1/JAK2 inhibitor) and ritlecitinib (JAK3 and TEC kinase inhibitor) (Fig. 1). Recently, Seneschal et al. published a systematic literature review, on the real-world use of baricitinib in the treatment of AD and AA demonstrating that this agent consistently improved the signs and symptoms of AD and was effective for the treatment of AA in both adult and paediatric patients with a favourable safety profile [29]. Finally, there are few case reports on the efficacy of upadacitinib in the treatment of both conditions [6, 7, 30] (Table 4) and a Phase 3 randomized, placebo-controlled, double-blind clinical trial is currently underway to assess the efficacy and safety of this agent in adult and adolescent patients with severe alopecia areata (Clinical Trial NCT06012240).

Chronic spontaneous urticaria

The prevalence of CSU among patients with AD varies across studies, with pediatric data indicating a higher risk of developing CSU in children with a history of AD [31]. Moreover, a higher prevalence of AD in subjects with CSU compared with the general population has been reported [32–34]. While AD and CSU have distinct clinical presentations, their pathogenesis overlaps may explain some shared treatments (Table 1). Regarding therapies, antihistamines are commonly used for managing CSU, but they are not recommended in AD [35]. Cyclosporine is recommended for severe forms of AD and as an add-on treatment to second-generation antihistamine in CSU management [36] (Fig. 1). As regards biologic drugs, dupilumab has been investigated for CSU management in two randomized, double-blind, placebo-controlled, phase 3 trials (LIBERTY-CSU CUPID) demonstrating its efficacy to reduce urticaria activity by reducing itch and hives severity in omalizumab-naive patients with CSU that is uncontrolled with first-generation H1-antihistamines [37]. However, the primary endpoint (change from baseline over 7 days in the Urticaria Activity Score or Itch Severity Score at week 24) for CUPID Study B (omalizumab-intolerant/incomplete responders) was not met. Summarizing, dupilumab can be an alternative to omalizumab in patients who are intolerant/unresponsive to this drug, or in subjects with atopic comorbidities [37]. Omalizumab, a monoclonal antibody targeting IgE, is indicated and recommended for the management of CSU. Data on its effectiveness in AD are conflicting. Indeed, although some studies suggest its effectiveness, other experiences showed that omalizumab use did not improve AD symptoms, despite the reduction of free serum IgE [38–40]. Other treatments are currently under investigation. Among these, JAK Inhibitors are currently approved for AD and under investigation for CSU [34], and Bruton's Tyrosine Kinase (BTK) Inhibitors are currently being explored for both conditions [41]. To sum up, due to their partially shared immunopathogenesis, some drugs, such as dupilumab or cyclosporine, should be preferred in patients affected by both AD and CSU. Certainly, further studies on the pathogenesis of these diseases will allow the development of new drugs, leading to a more personalized approach in subjects with both conditions.

Hidradenitis suppurativa

HS is a chronic recurrent inflammatory disease affecting skin-bearing apocrine glands, typically in the axillae, breasts, groin and perineum [42]. The main classic manifestations are painful, deep-seated, inflamed lesions, including nodules and abscesses, in addition to sinus tracts and scarring. Few studies investigate the association between HS and AD. A possible association between AD and HS was firstly suggested by Kaakati et al. that observed that that patients

Table 4 Effectiveness of systemic treatments in patients with concurrent AD and AA

Author, year	Study population	Observation period	Results	Quality of the study
Asfour et al., 2022 [6]	59-year-old woman affected by relapsing–remitting multifocal alopecia areata with associated recession of the frontotemporal hair line and severe eyebrow loss (SALT = 22) and moderate-severe AD (EASI = 37.4) treated with upadacitinib	8 weeks	Regrowth in her chronic preauricular AA patches within 4 weeks and partial response in her eyebrows. She is tolerating the treatment well with no side effects at 8 weeks	C4
Cantelli et al., 2022 [30]	24-year-old patient with an history of AD since childhood (EASI = 45.1) and 10 years of AA (SALT = 91.5) treated with upadacitinib 30 mg	3 months	Clinical improvement of both AD and AA. Trichoscopy shows regrowing hair all over the scalp without any sign of disease activity. No adverse events have reported	C4
Liu et al., 2024 [7]	12-year-old boy with severe AD (EASI = 34) and a coin-sized patch of hair loss in the occipital scalp, treated with abrocitinib 200 mg	12 weeks	Complete resolution of his AA symptoms with only mild relapse of the AD lesions	C4

AA Alopecia areata, *AD* Atopic dermatitis, *DLQI* Dermatology Life Quality Index, *EASI* Eczema Area and Severity Index, *P-NRS* Pruritus Numerical Rating Scale, *QoL* quality of life, *SALT* Severity of Alopecia Tool

with a diagnosis of AD have an approximately 5.57-fold increased OR of having HS as compared with those who do not have AD in a retrospective cohort study on 28,780 patients with and 48,383 controls [43]. Subsequently, this observation was confirmed by another large epidemiologic study on 6671 patients with HS and 32,994 control individuals which demonstrated a statistically significant bidirectional association between HS and AD [44]. Patients with HS experienced a twofold elevated risk of developing subsequent AD, whereas a history of AD was associated with a 40% increase in the odds of having subsequent HS. Patients with HS with comorbid AD were more likely to be younger, female and nonsmoker and have a lower body mass index than patients with HS without AD [44]. In the last years, few reports described the effectiveness of dupilumab in the treatment of patients with AD and HS [45–47] (Table 5) even thou Kluger described the therapeutic failure of dupilumab in a patients affected by HS alone [48]. Recently, the results of the phase II clinical trial on the efficacy of upadacitinib in the treatment of moderate-to-severe HS were published (NCT04430855) [49] and it could be speculated that the future perspective of the treatment of concurrent AD and HS may be represented by the phase I trial on the IRAK4 degrader (NCT04772885) [50].

Prurigo nodularis

PN is a distinct clinical condition characterized by persistent pruritus and multiple elevated,

Table 5 Effectiveness of systemic treatments in patients with concurrent AD and HS

Author, year	Study population	Observation period	Results	Quality of the study
Gambardella et al., 2020 [45]	50-year-old man affected by AD (EASI = 20, P-NRS = 10, DLQI = 16) and HS (Hurley II, AN count = 16) treated with dupilumab	16 weeks	Achievement of HiSCR with an AN count = 6	C4
Molinelli et al., 2022 [46]	43-year-old man with a childhood-onset AD (EASI = 30, P-NRS = 10, DLQI = 25) and HS (Hurley II, IHS4 = 8, pain VAS = 6) treated with dupilumab	24 weeks + 24 weeks of follow-up	EASI = 5.4, P-NRS = 1 and DLQI = 3. No acute flare of HS was reported for the next 6 months	C4
Cho et al., 2022 [47]	25-year-old man with a childhood-onset AD (EASI = 33.4) and concomitant HS (IHS4 = 30) treated with dupilumab and oral clindamycin	16 weeks	At week 16, achievement of EASI75 and partial improvement of HS. At 1 year, EASI < 4 and IHS4 < 3	C4

EASI Eczema Area and Severity Index, *P-NRS* Pruritus Numerical Rating Scale, *DLQI* Dermatology Life Quality Index, *AN* abscesses nodules, *HiSCR* Hidradenitis Suppurativa Clinical Response, *IHS4* International Hidradenitis Suppurativa Severity Scoring System

firm nodular lesions that may be localized or generalized [51]. Pathogenetically, immune and neural dysregulation seem to be involved [52]. A recent meta-analysis showed an increased risk of AD in patients suffering from PN as compared with control groups (OR: 16.85; 95% CI 6.13–46.31; $I2 = 100\%$) [53]. Similarly, an increased prevalence of PN was identified in subjects with AD (2.00%; 95% CI 1.62–2.37%) [51]. Since AD and PN share common pathogenic pathways (Table 1), several treatment strategies overlap and, in line with AD, the use of antihistaminergic agents is generally ineffective, as PN is a non-histaminergic itch condition [12]. Regarding the use of biologic drugs, dupilumab and nemolizumab have been authorized for the management of both diseases. Dupilumab in LIBERTY-PN PRIME and PRIME2 phase 3 trials including adults with PN with ≥ 20 nodules and severe itch uncontrolled with topical therapies, showed an improvement ≥ 4-point Worst Itch Numeric Rating Scale (WI-NRS) at week 24 in 60.0% of patients vs 18.4% of subjects receiving placebo, respectively, in PRIME1 (95% CI 27.8–57.7 for the difference, $p < 0.001$) and at week 12 by 37.2% and 22.0% of patients, respectively, in PRIME2 (95% CI, 2.3–31.2; $p = 0.022$) [54]. Finally, regarding JAK inhibitors, interesting data on their use in PN are emerging [12], with an ongoing phase II trial on the use of abrocitinib in 10 patients with PN and 10 patients with chronic pruritus of unknown origin (Clinical Trial NCT05038982), and other ongoing trials on povorcitinib (phase II) and topical ruxolitinib (phase III) [55]. To sum up, PN can be defined as a distinct clinical entity compared with AD, thus requiring major knowledge on pathogenesis and diagnostic/management. Except for dupilumab and nemolizumab, all treatments for PN are currently used off-label, and all data supporting the use of these therapies are based on anecdotal or small clinical trials [56–62] (Table 6).

Psoriasis

Psoriasis (Pso) is a common immune-mediated, genetic, inflammatory disease manifesting in the skin or joints or both with a prevalence in Europe and North America of about 2% [63]. Plaque-type Pso is the most common form of the disease, and it is characterized by monomorphic, sharply demarcated erythematous plaques covered by silvery lamellar scales. Plaques can be few or extend over larger areas and, despite psoriasis can affect any skin site, typical locations include the extensor surfaces of forearms and shins, sacral and retro-auricular regions and scalp [64]. Pso prevalence increases approximately linearly over the life course, from 0.12% at age 1 year to 1.2% at age 18 years [65] while AD prevalence in children is up to 20% [64]. Retrospective and case studies about concomitant AD and Pso have been increasingly reported either as diseases with overlapping features (the so-called psoriasis dermatitis (PD), atopic psoriasis or psorema) or as coexisting diseases on different body regions in the same individual. The prevalence of PD is estimated at 2% among atopic patients [66] but ranges from 0.2 to 16.7%, depending on the definition and methods [13]. In addition, few cases also report sudden phenotypic AD/Pso switches in the course of disease, in several cases under biological treatment (so-called paradoxical reactions). In particular, the incidence of a switch from AD towards Pso in dupilumab-treated patients is 1.7% [67] and it has been described to occur 2–18 months after the initiation of dupilumab and in patients with infancy or early childhood-onset AD [13]. The suggested mechanism was that the inhibition of IL-4 activates Th1 and Th17 cells because IL-4 negatively regulates Th1 and Th17 cells, both of which are involved in Pso [13]. Conversely, switches from Pso to AD have been described in patients treated with IL-17i, IL-12/–23i and IL-23i agents after a treatment of 5 weeks to 22 months [68]. Concerning the cases of transformation from AD to Pso or vice versa during biologics treatment, discontinuation of the induced biologic agents and switching to a different mode of action is a generally accepted strategy because transformation or paradoxical reaction is regarded as a class effect, not merely a drug-specific effect [67]. Nonetheless, there has been no consensus on the optimal treatment of coexisting AD and Pso. Conventional systemic therapies (including methotrexate, azathioprine and cyclosporine) and phototherapy can be used

Table 6 Real-life evidence of systemic shared treatment in patients with concurrent AD and PN, or in PN-like AD

Author, year	Study population	Observation period	Results	Quality of the study
Napolitano et al., 2020 [56]	90 patients with PN-like AD treated with dupilumab	9 months	A significant improvement in EASI and DLQI was reported	C3
Ferrucci et al., 2021 [57]	11 patients with PN-like AD treated with dupilumab	16 weeks	All patients showed rapid clinical improvement of cutaneous lesions	C4
Yew et al., 2023 [58]	36 patients with PN received dupilumab and 13 patients had oral JAK-inhibitors. Of these, 75.5% had concomitant AD	12–16 weeks	At week 12–16, the ≥ 4-point reduction in WI-NRS of the dupilumab group was achieved by 60.0%, versus 58.3% in the JAK-inhibitors group	C3
Mitsuyama et al., 2023 [59]	Four patients with AD and PN treated with dupilumab	4–8 weeks	EASI90 was achieved 4—8 weeks after initiating dupilumab treatment	C4
Pezzolo et al., 2023 [60]	17 patients with PN-like AD treated with tralokinumab	32 weeks	All patients reached EASI50 within 4 weeks, EASI75 within 12 weeks and EASI90 within 32 weeks	C4
Lou et al., 2024 [61]	12-year-old male with PN-like AD and 28-year-old female with PN-like AD treated with upadacitinib	5 months for the first case and 3 months for the second case	Significant reduction in, EASI, DLQI and NRS at weeks 4 and 8 in both cases	C4
Pezzolo et al., 2024 [62]	21 patients with PN ($n = 4$) or PN-like AD ($n = 17$) treated with upadacitinib	52 weeks	The average IGA, PP-NRS, Sleep Disturbance-NRS and DLQI values significantly decreased from baseline up to week 52 by 78.7%, by 93.3%, by 90.9% and by 89.9%, respectively	C4

EASI Eczema Area and Severity Index, *DLQI* Dermatology Life and Quality Index, *WI-NRS* Worst Itch Numerical Rating Scale, *IGA* Investigator Global Assessment, *PP-NRS* Peak Pruritus-Numeric Rating Scale

for both diseases, but their long-term toxicity could represent an evident limit in the context of a continuous disease control. While biologic agents targeting one specific T cell line appear to be non-beneficial for co-occurring diseases, the inhibition of the JAK-STAT pathway has been a safe and effective strategy for both conditions. While no JAK inhibitor-induced Pso during AD treatment was identified in literature, several case-series describe the efficacy of JAK inhibitors, particularly upadacitinib, in the treatment of PD or AD/Pso paradoxical reactions (Table 8).

Vitiligo

Vitiligo is an autoimmune condition characterized by patches of skin depigmentation, resulting from destruction of melanocytes. Vitiligo is mainly driven by a Th1-mediated immune response, but recent studies suggest that Th2 and Th17 pathways may also be involved. These immune responses could contribute to melanocyte damage and skin inflammation, adding more complexity to the disease's underlying mechanisms. The prevalence of vitiligo has been

Table 7 Effectiveness of systemic treatments in patients with concurrent AD and vitiligo

Author, year	Study population	Observation period	Results	Quality of the study
Shao et al., 2024 [14]	65-year-old male patient with generalized vitiligo and concomitant AD, treated with 100 mg of abrocitinib	6 months	Erythema and papules significantly improved, and subjective itching symptoms disappeared. In addition, significant improvement and repigmentation of vitiligo lesions on the face and body were observed	C4
Pan et al., 2023 [72]	16-year-old boy affected by childhood-onset AD (EASI = 10.5, P-NRS = 6) with rapidly progressive vitiligo (VIDA = 2, VASI = 0.35) treated with 15 mg of upadacitinib halved after 4 months	16 weeks	Nearly 90% repigmentation of face and neck, 60% repigmentation of chest and only a little repigmentation of the extremities P-NRS = 0 and EASI = 4.1	C4
Magnanimi et al., 2025 [73]	25-year-old woman affected by childhood-onset AD (EASI = 18, P-NRS = 10) and progressive vitiligo at the age of 16 years (VASI = 0.45) with high impact on QoL (DLQI = 17) treated with upadacitinib 15 mg	1 year	At week 1 EASI = 0, P-NRS = 0, DLQI = 0 and VASI = 0.25). An almost complete remission of vitiligo was achieved at week 28 and maintained up to 1 year	C4

DLQI Dermatology Life Quality Index, *EASI* Eczema Area and Severity Index, *P-NRS* Pruritus Numerical Rating Scale, *QoL* quality of life, *SALT* Severity of Alopecia Tool, *VASI* Vitiligo Area Scoring Index, *VIDA* Vitiligo Disease Activity Score

estimated to be 0.5–1% of the world population. Vitiligo usually appears before the age of 30 years and is frequently associated with other skin diseases and autoimmune disorders [69]. Previous studies indicate a notable association between AD and vitiligo, suggesting a shared underlying immunological mechanism [70, 71]. Given the shared immune pathways (Table 1), patients with concomitant AD and vitiligo require an integrated approach. JAK inhibitors, which target the JAK-STAT signaling cascade, have emerged as a promising therapeutic option for both diseases [14, 72–74] (Table 7). Currently, topical ruxolitinib is the only JAK inhibitor approved for the treatment of vitiligo in patients aged ≥ 12 years. Meanwhile, upadacitinib, an oral selective JAK1 inhibitor, approved for the treatment of moderate/severe AD, has recently been proposed as an effective treatment in recalcitrant vitiligo [75] (Fig. 1). Ongoing research and clinical trials will be crucial in optimizing therapeutic strategies and improving outcomes for patients affected by both conditions.

Table 8 Possible interventions and strength of recommendation

Comorbidity	Agent	Treatment target	Results and quality of the study
Allergic contact dermatitis	Abrocitinib	iJAK-STAT pathway	Reported effective C, 3
	Baricitinib	iJAK-STAT pathway	Reported effective C, 3
	Dupilumab	iIL4/13	Controversial, C, 3
Alopecia areata	Abrocitinib	iJAK-STAT pathway	Reported effective C, 3
	Baricitinib	iJAK-STAT pathway	EMA and FDA approved, A2, 1
	Ritlecitinib	iJAK-STAT pathway	EMA and FDA approved A2, 1
	Upadacitinib	iJAK-STAT pathway	Reported effective B, 1
Hidradenitis suppurativa	Dupilumab	iIL4/13	Controversial C, 3
	Upadacitinib	iJAK-STAT pathway	Reported effective A2, 1
Prurigo nodularis	Baricitinib	iJAK-STAT pathway	Reported effective C, 3
	Dupilumab	iIL4/13	EMA and FDA approved A1, 1
	Nemolizumab	iIL31	EMA and FDA approved A2, 1
	Tralukinumab	iIL13	Reported effective C, 3
	Upadacitinib	iJAK	Reported effective A2, 1
Plaque-type psoriasis	Upadacitinib	iJAK	Reported effective A2, 1
Psoriatic arthritis	Upadacitinib	iJAK	EMA and FDA approved A1, 1
Vitiligo	Roxolitinib	iJAK	EMA approved A2, 1
	Upadacitinib	iJAK	Reported effective A2, 1

CONCLUSIONS

Recognizing comorbidities in AD is critical for effective management. Tailored therapies targeting both AD and its comorbidities, based on shared immunological mechanisms, may improve outcomes. To date, some agents are indicated for AD and some comorbidities, others are still under investigation (Table 8).

The coexistence in a patient with AD and other dermatological diseases could represent a problem in clinical practice, particularly when a biological agent or a small molecule is needed to manage disease manifestations.

Cumulative effects of different traditional treatments when added to treat different dermatological conditions associated with AD could be inappropriate and harmful.

In the actual scenario, apart from AD and psoriatic arthritis (upadacitinib is indicated and approved by EMA and FDA for their treatment and the treatment of AD) and, partially vitiligo (topical ruxolitinib is EMA-approved for the treatment of vitiligo and FDA-approved for the treatment of AD), the choice of a treatment could be inappropriate and negatively influence AD or the associated skin disease. Furthermore, although upadacitinib has been reported to be effective, it remains off-label for the treatment of AA and HS. Results from ongoing phase 3 trials and published phase 2 trials suggest that this molecule could represent an effective therapeutic approach when these diseases occur with AD. In fact, upadacitinib is EMA/FDA approved for the treatment of AD and the quality of the studies reporting upadacitinib efficacy in AA and HS is B, 1 and A2, 1, respectively. This latter observation is appropriate even when AD coexist with vitiligo (quality of the research of upadacitinib in the treatment of vitiligo: A2, 1) or psoriasis. In fact, upadacitinib is EMA/FDA approved for psoriatic arthritis but has been reported to be effective on the skin domain in high quality studies. Unfortunately, in CSU and ACD there are few low-quality studies the efficacy of biological agents or small molecules approved for the treatment of AD in their management (Fig. 1).

The joint therapeutic opportunities in the dermatological field are now numerous and require the clinician to constantly and continuously update his scientific knowledge.

Further research is needed to optimize treatment strategies and explore combination therapies and algorithms for patients with both AD and comorbid dermatological conditions.

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Declarations

Conflict of interest. Maria Esposito has served as speaker/consultant for Abbvie, Amgen, Almirall, Boehringer Ingelheim, Eli Lilly, Janssen, Leopharma, Novartis, Pfizer, Sanofi Regeneron and UCB. Alessandro Giunta has served as speaker/consultant for Abbvie, Boehringer-Ingelheim, Eli Lilly, Novartis, Sandoz and UCB. Maria Concetta Fargnoli has served on advisory boards, received honoraria for lectures and/or research grants from AMGEN, Almirall, Abbvie, Boehringer-Ingelheim, BMS, Galderma, Kyowa Kyrin, Leo Pharma, Pierre Fabre, UCB, Lilly, Pfizer, Janssen, MSD, Novartis, Sanofi-Regeneron and Sunpharma. Cataldo Patruno has acted as speaker, consultant and/or advisory board member AbbVie, Almirall, Amgen, Galderma, Leo Pharma, Lilly, Novartis, Pfizer, Pierre Fabre and Sanofi. Maddalena Napolitano has acted as

speaker, consultant and/or advisory board member for Abbvie, Eli Lilly, Leo Pharma, Novartis, Incyte and Sanofi. Andrea De Berardinis, Luca Potestio and Lina Maria Magnanimi have nothing to disclose.

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