



Not actual patients.

Diagnosing AD and PN in your patients

Get to know the signs and symptoms of atopic dermatitis (AD)
and prurigo nodularis (PN).

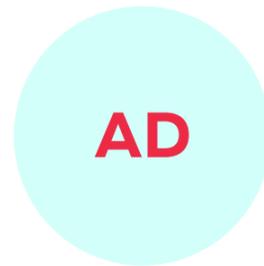
GALDERMA



How to diagnose AD

What is AD?

AD is a common, chronic, inflammatory skin disease that is characterized by persistent pruritus, embarrassing eczematous lesions, and frequent skin infections.^{1,2} Knowing how to diagnose AD can help you identify appropriate treatment options for your patients.



The signs and symptoms

AD is diagnosed clinically based on historical signs and symptoms, morphology and distribution of skin lesions, and associated clinical features. When examining patients, the signs and symptoms of AD to look out for are³:

Essential	<ul style="list-style-type: none"> • Pruritus • Acute, subacute, or chronic eczema*
Important	<ul style="list-style-type: none"> • Early age of onset • Atopy with personal and/or family history and immunoglobulin E reactivity • Xerosis
Associated	<ul style="list-style-type: none"> • Atypical vascular responses (eg, facial pallor, white dermographism, and delayed blanch response) • Keratosis pilaris/pityriasis alba/hyperlinear palms/ichthyosis • Ocular/periorbital changes • Other regional findings (eg, perioral changes/periauricular lesions) • Perifollicular accentuation/lichenification/prurigo lesions • Comorbidities and clinical associations such as allergies, asthma, allergic rhinitis/rhinoconjunctivitis, sleep disturbance, depression, cancer, and obesity

Differentiating AD from PN

AD presents with an often diffuse distribution of eczematous lesions while PN presents with an often symmetrical distribution of firm, nodular lesions.^{1,4}

*Essential AD features are typical morphology and age-specific patterns (facial, neck, and extensor involvement in infants and children, current or previous flexural lesions in any age group, and a sparing of the groin and axillary regions) and a chronic or relapsing history.³

Patients with AD may present with different phenotypes

AD is a heterogeneous disease associated with multiple phenotypes and variable intensity of itch and lesions⁵:

AD phenotypes^{5*}

<p>SI-ML Severe itch and mild-moderate lesions</p>	<p>SI-SL Severe itch and severe lesions</p>
<p>MI-ML Mild-moderate itch and mild-moderate lesions</p>	<p>MI-SL Mild-moderate itch and severe lesions</p>



Itch-dominant (SI-ML) AD is a common, burdensome, and distinct subtype of AD, occurring in up to 29% of patients. The proportion of patients with itch-dominant AD was higher in women and patients of African descent.⁵

*Results were based on a prospective, dermatology practice-based study of 592 adults with AD in the United States. This study used web-based questionnaires, physical exams, and combined itch and lesional severity to determine the characteristics, associations, burden, and disease course of patients with AD. Four AD subsets were defined using a verbal rating scale for average itch combined with EASI, objective SCORAD, or vIGA-AD.⁵
EASI=eczema area and severity index; SCORAD=SCORing Atopic Dermatitis; vIGA-AD=validated Investigator Global Assessment for AD.



Not an actual patient.

AD in patients of color

Recognizing the difference

The signs and symptoms of AD in skin of color may differ in visual appearance, predominantly due to differences in pigmentation and lesion distribution. It's important to recognize these differences in your patients with darker skin⁷:

<p>Pigmentation differences</p>	<ul style="list-style-type: none"> • Erythema that may appear violaceous* • Lichenification, hyperlinearity of the palms, Dennie-Morgan lines, and diffuse xerosis • Postinflammatory dyspigmentation (including hyperpigmentation and hypopigmentation)
<p>Location of lesions</p>	<ul style="list-style-type: none"> • Extensor involvement, which may be more common than flexural dermatitis • Perifollicular accentuation and scattered distinct papules on the extensor surfaces and trunk

Compared with White patients, those of Asian descent are more likely to have well-demarcated lesions and increased scaling and lichenification⁷



The damaging disease burden

Persistent pruritus is much more than just an irritating sensation—it is the most burdensome symptom for patients with AD and can significantly disrupt their daily activities, sleep, and psychological well-being.^{2,6}

It is recommended that you ask your patients with AD about³:

- Pruritus (itch)
- Sleep
- Impact on daily activity (including effects on work, school, and well-being)
- Disease persistence

*Look for the presence of edema, skin warmth, or scaling to discern underlying erythema in darker skin.⁷

How to diagnose PN

What is PN?

PN, also described as chronic prurigo of the nodular type, is a distinct, underrecognized, and chronic inflammatory skin disease that is characterized by disfiguring, often excoriated nodules and intractable pruritus.^{4,8,9} PN is more common in older adults aged 50-55 years, women, and disproportionately people of African descent.^{4,9} Knowing how to diagnose PN can help you identify appropriate treatment options for your patients.



The signs and symptoms

PN has a characteristic appearance on morphology. When examining patients, the signs and symptoms of PN to look out for are^{4,10}:

Essential

- The presence of firm, nodular lesions*
- Pruritus lasting ≥6 weeks
- A history and/or signs of repeated scratching, picking, or rubbing (eg, excoriations and scars)

Important

- A symmetrical distribution of lesions on areas of skin that are accessible to scratching (rarely found on the face, palms, soles, scalp, or genitals)
- The presence of additional lesions induced by scratching, picking, or rubbing (eg, lichenified plaques, excoriations, ulcerations, and/or scars)
- Pruritus that is accompanied by burning, stinging, pain, and/or other sensations

Associated

- Burden of disease, such as impaired quality of life, sleep deprivation, missed work and/or school, emotional impact (eg, depression, anxiety, anger, shame, helplessness), and social isolation
- Systemic comorbidities, such as impaired liver, renal, or thyroid function, diabetes, HIV infection, hepatitis B/C, and malignancy

Differentiating PN from AD

PN presents with an often symmetrical distribution of firm, nodular lesions while AD presents with an often diffuse distribution of eczematous lesions.^{1,4}



Not an actual patient.

Laboratory assessment for PN

Initial laboratory assessment for all patients suspected of having PN should include¹⁰:

- A complete blood cell count with differential
- Hepatic and renal function tests

Recognizing PN in patients of color

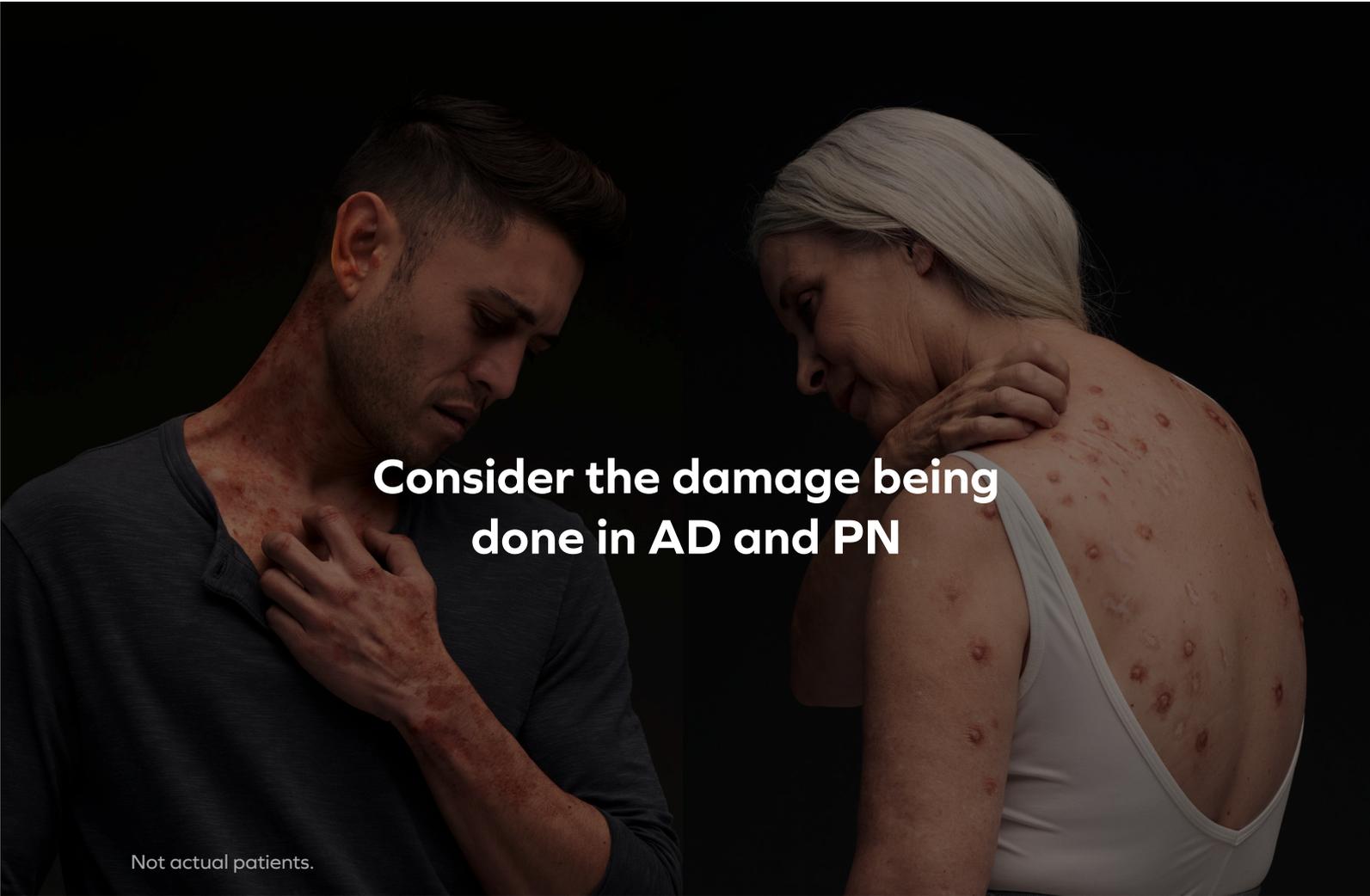
It's important to remember that the signs and symptoms of PN may look different in patients of color.^{4,10} Postinflammatory dyspigmentation, including hyperpigmentation and hypopigmentation, is more common in darker skin.¹¹

The burden of PN is oppressive

PN severely impacts many aspects of patients' lives.¹² Studies have shown that patients with PN experience a higher burden of disease than patients with several other chronic pruritic skin diseases. This may be due to the pruritus of PN being more severe than that of the other skin diseases.¹³

Although PN is sometimes associated with AD, most patients with PN do not have a history of AD¹⁴⁻¹⁶

*The presence of lesions may be localized or generalized. Lesions may also be clinically defined as pruriginous lesions, which are excoriated, scaling, and/or crusted papules and/or nodules and/or plaques, often with a whitish or pink center and a hyperpigmented border.¹⁰



Consider the damage being done in AD and PN

Not actual patients.

Interested in learning more?

Visit [IL31role.com](https://www.il31role.com) to learn more about AD, PN, and IL-31: a cytokine that plays a role in these chronic inflammatory skin diseases^{9,17,18}

The images in this material are not of actual patients with AD or PN. They were created based on informed Galderma insights. AD and PN can manifest in individuals differently.

References:

1. Langan SM, Irvine AD, Weidinger S. Atopic dermatitis. *Lancet*. 2020;396(10247):345-360. doi:10.1016/S0140-6736(20)31286-1
2. McCleary KK. More than skin deep: understanding the lived experience of eczema. March 18, 2020. Accessed February 16, 2022. <http://www.morethanskindeep-eczema.org/report.html>
3. Eichenfield LF, Tom WL, Chamlin SL, et al. Guidelines of care for the management of atopic dermatitis: section 1. Diagnosis and assessment of atopic dermatitis. *J Am Acad Dermatol*. 2014;70(2):338-351. doi:10.1016/j.jaad.2013.10.010
4. Elmariah S, Kim B, Berger T, et al. Practical approaches for diagnosis and management of prurigo nodularis: United States expert panel consensus. *J Am Acad Dermatol*. 2021;84(3):747-760. doi:10.1016/j.jaad.2020.07.025
5. Chovatiya R, Lei D, Ahmed A, Chavda R, Gabriel S, Silverberg JI. Clinical phenotyping of atopic dermatitis using combined itch and lesional severity: a prospective observational study. *Ann Allergy Asthma Immunol*. 2021;127(1):83-90.e2. doi:10.1016/j.anai.2021.03.019
6. Legat FJ. Itch in atopic dermatitis—what is new? *Front Med (Lausanne)*. 2021;8:644760. doi:10.3389/fmed.2021.644760
7. Kaufman BP, Guttman-Yassky E, Alexis AF. Atopic dermatitis in diverse racial and ethnic groups—variations in epidemiology, genetics, clinical presentation and treatment. *Exp Dermatol*. 2018;27(4):340-357. doi:10.1111/exd.13514
8. Whang KA, Mahadevan V, Bakhshi PR, et al. Prevalence of prurigo nodularis in the United States. *J Allergy Clin Immunol Pract*. 2020;8(9):3240-3241. doi:10.1016/j.jaip.2020.05.051
9. Williams KA, Roh YS, Brown I, et al. Pathophysiology, diagnosis, and pharmacological treatment of prurigo nodularis. *Expert Rev Clin Pharmacol*. 2021;14(1):67-77. doi:10.1080/17512433.2021.1852080
10. Ständer S, Pereira MP, Berger T, et al. IFSI-guideline on chronic prurigo including prurigo nodularis. *Itch*. 2020;5(4):e42. doi:10.1097/itx.000000000000042
11. Errichetti E, Lallas A, eds. *Dermoscopy in General Dermatology for Skin of Colour*. CRC Press; 2021.
12. Janmohamed SR, Gwillim EC, Yousaf M, Patel KR, Silverberg JI. The impact of prurigo nodularis on quality of life: a systematic review and meta-analysis. *Arch Dermatol Res*. 2021;313(8):669-677. doi:10.1007/s00403-020-02148-0
13. Steinke S, Zeidler C, Riepe C, et al. Humanistic burden of chronic pruritus in patients with inflammatory dermatoses: results of the European Academy of Dermatology and Venereology Network on Assessment of Severity and Burden of Pruritus (PruNet) cross-sectional trial. *J Am Acad Dermatol*. 2018;79(3):457-463.e5. doi:10.1016/j.jaad.2018.04.044
14. Huang AH, Canner JK, Khanna R, Kang S, Kwatra SG. Real-world prevalence of prurigo nodularis and burden of associated diseases. *J Invest Dermatol*. 2020;140(2):480-483. e4. doi:10.1016/j.jid.2019.07.697
15. Pereira MP, Hoffmann V, Weisshaar E, et al. Chronic nodular prurigo: clinical profile and burden. A European cross-sectional study. *J Eur Acad Dermatol Venereol*. 2020;34(10):2373-2383. doi:10.1111/jdv.16309
16. Kwon CD, Khanna R, Williams KA, Kwatra MM, Kwatra SG. Diagnostic workup and evaluation of patients with prurigo nodularis. *Medicines (Basel)*. 2019;6(4):97. doi:10.3390/medicines6040097
17. Nemmer JM, Kuchner M, Datsi A, et al. Interleukin-31 signaling bridges the gap between immune cells, the nervous system and epithelial tissues. *Front Med (Lausanne)*. 2021;8:639097. doi:10.3389/fmed.2021.639097
18. Datsi A, Steinhoff M, Ahmad F, Alam M, Buddenkotte J. Interleukin-31: the “itchy” cytokine in inflammation and therapy. *Allergy*. 2021;76(10):2982-2997. doi:10.1111/all.14791

GALDERMA

For US healthcare professionals only.

©2022 Galderma Laboratories, L.P. All Rights Reserved.
All trademarks are the property of their respective owners.

GL-NAD-2200008 February 2022