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Antinuclear antibody positivity rate and clinical characteristics in prurigo chronica multiformis: a retrospective study of 26 patients

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Background: Prurigo chronica multiformis is a refractory chronic pruritic dermatosis that predominantly affects older adults, but the frequency and significance of antinuclear antibody (ANA) positivity remain unclear.
Methods: This retrospective single-center study included 26 patients with prurigo chronica multiformis treated between March 2016 and April 2025. Patients were divided into two groups according to ANA status, and baseline characteristics, allergic background, laboratory investigations, extent of skin involvement, and responsiveness to topical corticosteroids were compared.
Results: Five of 26 patients (19.2%) were positive for ANA. Among evaluable patients, all patients in the positive group showed sensitization to at least one allergen, which was more frequent than in the negative group (100% vs. 37.5%, p = 0.035). The median peripheral blood eosinophil percentage was significantly lower in the positive group (4.0% vs. 8.0%, p = 0.031). No significant differences were observed in the eosinophil count, total IgE levels, extent of skin involvement, or responsiveness to topical corticosteroids.
Conclusion: Approximately one-fifth of patients with prurigo chronica multiformis were positive for ANA. ANA-positive patients showed a high rate of allergen sensitization and a lower peripheral blood eosinophil percentage. These findings suggest that prurigo chronica multiformis may include an immunologic subtype in which autoimmunity and allergic inflammation intersect.

KEYWORDS
antinuclear antibody, chronic prurigo, eosinophil, IgE, prurigo chronica multiformis

Introduction

Prurigo chronica multiformis (PCM) is a chronic inflammatory dermatosis that predominantly affects older adults and typically develops on the lower abdomen, lumbosacral region, and thighs. Clinically, PCM initially presents as intensely pruritic urticarial papules, which gradually evolve into skin-colored to light-brown firm papules. Unlike prurigo nodularis, it is a refractory chronic inflammatory dermatosis characterized by the frequent clustering and coalescence of lesions, resulting in the formation of polygonal lichenified plaques. Mechanical scratching may induce wheals or urticaria-like erythema surrounding the lesions. Individual eruptions usually persist for several weeks to months and resolve with post-inflammatory hyperpigmentation; however, recurrence is common, and patients often experience repeated cycles of remission and exacerbation over several years [1, 2]. Histopathologically, PCM is characterized by mild superficial perivascular lymphocytic infiltration in the dermis, accompanied by variable eosinophilic infiltration in

perivascular and intercollagenous spaces [2, 3]. Because its clinical manifestations overlap with those of eczematous disorders including atopic dermatitis (AD), urticaria, insect bite reactions, scabies, drug eruptions, cutaneous lymphoma, and even prodromal or atypical bullous pemphigoid, establishing an accurate diagnosis is often challenging.

Although PCM has been proposed as a distinct disease entity primarily in Japan, its definition has not been fully standardized within the international classification system of chronic prurigo (CPG) because of its marked clinical heterogeneity. However, an expert consensus published in 2024 redefined PCM as an important independent disease entity characteristic of older individuals, accelerating discussions regarding its diagnostic criteria and pathophysiology [2].

PCM is thought to be predominantly driven by type 2 inflammation, and many patients exhibit elevated serum total IgE levels, sensitization to specific allergens, and increased levels of thymus and activation-regulated chemokine (TARC), which promotes eosinophil activation [1, 4]. Nevertheless, PCM is often highly refractory to conventional topical corticosteroids and antihistamines, suggesting that its pathogenesis involves complex immunological and neurological interactions beyond a simple allergic response. Recent studies have suggested that mediators such as β-endorphin and autotaxin may directly or indirectly stimulate peripheral sensory nerve endings and contribute to intractable pruritus in chronic prurigo, including PCM [5]. Furthermore, age-related physiological skin changes, including impaired barrier function and immunosenescence, may provide a background that amplifies these inflammatory processes [6].

We focused on the prevalence of antinuclear antibodies (ANA) as a potential pathophysiological difference between PCM and related disorders such as AD and prurigo nodularis. In AD, the ANA positivity rate has been reported to be 31.3%, and patients with positive ANA exhibited more severe facial eruptions and significantly lower leukocyte counts compared with patients without positive ANA [7]. Furthermore, most patients with positive ANA exhibit a homogeneous staining pattern, and human elongation factor-1α (hEF-1α) has been identified as a major target autoantigen of these antibodies [7]. The patients positive for anti-hEF-1α antibodies reportedly demonstrate clinical features resembling systemic lupus erythematosus (SLE), including severe facial involvement and lower white blood cell counts. Thus, a subset of adult AD may possess an autoimmune background [7]. In contrast, the ANA positivity rate in prurigo nodularis was reported to be 14.3%, and a recent report suggested that ANA-positive patients did not subsequently develop connective tissue diseases during follow-up [8].

Therefore, in the present study, we retrospectively investigated, for the first time, the prevalence and clinical significance of ANA positivity in patients with PCM.

Materials and methods

Patients

This study was approved by the Wakayama Medical University Institutional Review Board (No. 2446), and written informed consent was obtained before patients were included in this study, in accordance with the Declaration of Helsinki.

Medical information was collected from 26 patients with PCM (20 males and 6 females) who were treated at our institute between March 2016 and April 2025. Their clinical features are shown in Table 1.

Clinical assessment

We diagnosed PCM according to the expert consensus of the Japanese Dermatological Association [2]. The diagnosis was based primarily on a persistent or recurrent course of intense pruritus and the presence of 2–3-mm solid papules, sometimes preceded by urticarial papules, that progressively aggregated or coalesced into polygonal lichenified plaques. Histopathological findings were used to support the diagnosis when skin biopsy was performed. Disease duration for several weeks to months was considered as "chronic", although no universally accepted definition has been established for PCM.

Medical records were reviewed to identify any history and clinical/laboratory features of internal malignancies as well as connective tissue diseases and other autoimmune diseases, including systemic lupus erythematosus, systemic sclerosis, Sjögren syndrome, dermatomyositis/polymyositis, and mixed connective tissue disease.

PCM patients were divided into ANA-positive and ANA-negative patients based on ANA status, and their clinical findings, laboratory data, and treatment responses were compared. ANA was measured by indirect immunofluorescence using HEp-2 cells. ANA positivity was defined as a titer of ≥1:40. The cutoffs for elevated total IgE and TARC were defined as > 358 IU/mL and >450 pg/mL, respectively. Specific IgE positivity was evaluated by MAST36 kit®, View Allergy 39® (View39) kit, or single-allergen assays, and Class ≥2 was considered positive for allergen sensitization.

Statistical analysis

Pearson's chi-square test (m × n-table) or Fisher's exact probability test was used to compare categorical variables between the two groups. The Mann–Whitney U test was used to compare medians between the two groups. $p < 0.05$ was considered statistically significant.

Results

Clinical characteristics of patients with PCM in the present study

Twenty-six patients were diagnosed with PCM based on a previously published report [2] (Table 1). Males were more frequently affected by PCM than females (male:female = 20:6), and median age at onset was 74.0 years (interquartile range [IQR]: 65.7–79.0). The median disease duration was 5.0 months (IQR: 2.7–15.0). Regarding allergic sensitization, a past history of allergic rhinitis, drug allergy, and food allergy was observed in five patients (19.2%) each, while one patient (3.8%) had bronchial

TABLE 1 Summary of clinical features in patients with prurigo chronica multiformis (n = 26).

Age at onset, years, median (IQR)	74.0 (65.7–79.0)
Sex	
Male	20
Female	6
Disease duration, months, median (IQR)	5.0 (2.7–15.0)
History of allergic diseases	
Bronchial asthma	1
Allergic rhinitis	5
Drug allergy	5
Food allergy	5
Family history of allergies	
+	1
–	25
Elevated total IgE levels*	
+	9
–	17
Sensitization to at least one allergen	
+	11
–	10
NA	5
Eosinophil %, median (IQR)	7.0 (3.7–13.1)
Eosinophil count, /μL, median (IQR)	524.0 (288.7–924.3)
Elevated TARC levels**	
+	17
–	6
NA	3
BSA, %, median (IQR)	13.0 (7.0–24.0)
Response to topical corticosteroids	
Good	5
Poor	21

Abbreviations: IQR, interquartile range; NA, not available; TARC, thymus and activation-regulated chemokine; BSA, body surface area. Unless otherwise indicated, values are numbers of patients.

*Elevated total IgE was defined as > 358 IU/mL.

**Elevated TARC was defined as > 450 pg/mL.

asthma. A family history of allergies was found in only one patient (3.8%).

Laboratory investigations showed that elevated total IgE levels were present in 9 patients (34.6%). Of the 21 patients whose specific IgE levels were evaluated, 11 (52.4%) were positive for at least one allergen. Elevated serum TARC levels were identified in 17 of 23 patients (73.9%) for whom data were available. The median blood eosinophil percentage and absolute count were 7.0% (IQR: 3.7–13.1) and 524.0/μL (IQR: 288.7–924.3), respectively.

In terms of treatment history, 21 patients (80.8%) exhibited a poor response to topical corticosteroids, whereas only five patients (19.2%) showed a good response. Apart from slightly lower proportions of elevated IgE levels (34.6% vs. 57.0%) [2] and the higher proportion of patients showing a poor response to topical corticosteroids (80.8% vs. 47.7%), these findings were generally consistent with those reported in previous studies of patients with PCM [3]. No patients had a documented history, clinical features, or laboratory findings of connective tissue diseases or other autoimmune diseases associated with ANA positivity. On

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TABLE 2 Association between clinical features and ANA positivity in patients with prurigo chronica multiformis (n = 26).

Variable	Patients with positive ANA (n = 5)	Patients with negative ANA (n = 21)	p value
Age at onset, years, median (IQR)	74.0 (69.0–80.5)	73.0 (65.5–79.0)	0.744
Sex			1.000
Male	4	16	
Female	1	5	
Disease duration, months, median (IQR)	8.0 (4.5–36.0)	5.0 (2.0–16.5)	0.266
History of allergic diseases			
Bronchial asthma	0	1	1.000
Allergic rhinitis	0	5	0.545
Drug allergy	0	5	0.545
Food allergy	1	4	1.000
Family history of allergies			0.192
+	1	0	
–	4	21	
Elevated total IgE levels*			1.000
+	2	7	
–	3	14	
Sensitization to at least one allergen			0.035
+	5	6	
–	0	10	
Eosinophil %, median (IQR)	4.0 (2.3–5.6)	8.0 (5.5–13.7)	0.031
Eosinophil count, /μL, median (IQR)	399.0 (150.0–582.0)	798.0 (315.0–1,091.2)	0.152
Elevated TARC levels**			1.000
+	2	15	
–	1	5	
BSA, %, median (IQR)	22.0 (4.0–30.5)	12.0 (7.2–16.5)	0.708
Response to topical corticosteroids			0.545
Good	0	5	
Poor	5	16	

Abbreviations: ANA, antinuclear antibody; IQR, interquartile range; TARC, thymus and activation-regulated chemokine; BSA, body surface area. Data on specific IgE levels, TARC levels, or BSA were not available for 0, 2, or 0 patients with positive ANA and 5, 1, and 1 patient in the ANA-negative group, respectively.
*Elevated total IgE was defined as > 358 IU/mL.
**Elevated TARC was defined as > 450 pg/mL.

the other hand, two patients had concomitant malignancies. However, the clinical course of the skin lesions was not associated with the treatment response or progression of the underlying malignancies. Accordingly, none of the cases were considered to represent paraneoplastic PCM.

Correlation between ANA positivity and blood eosinophil levels

To clarify the potential influence of autoimmune dysregulation on the inflammatory profile of PCM, patients were subgrouped based on their ANA status. ANA positivity was identified in 5 of 26 patients (19.2%): two patients showed mixed homogeneous and speckled staining patterns at a titer of 1:40, one showed a homogeneous staining pattern at 1:40, one showed a speckled staining pattern at 1:40, and one showed a homogeneous staining pattern at 1:80.

Clinical and laboratory parameters were compared between ANA-positive and ANA-negative patients. As shown in Table 2, no significant differences were observed between the ANA-positive and ANA-negative groups in median age at onset (74.0 vs. 73.0 years, p = 0.744) or sex distribution (male:female = 4:1 vs.

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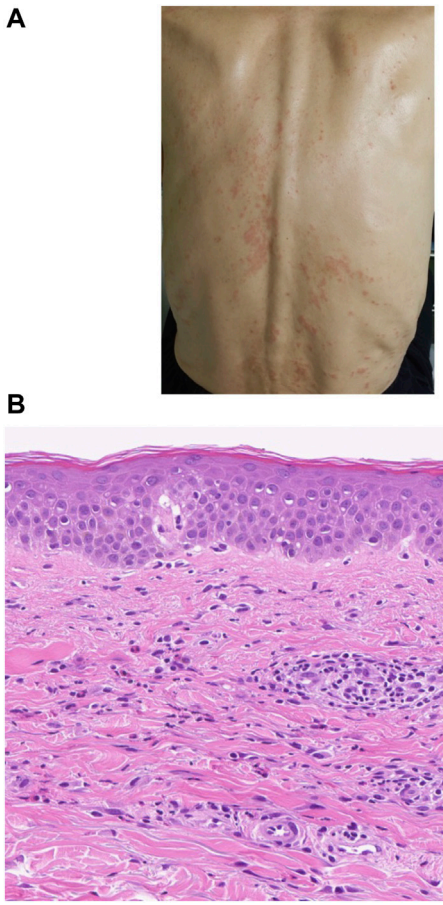

FIGURE 1
Representative ANA-positive PCM case. **(A)** Clinical presentation of prurigo chronica multiformis with ANA positivity. Skin lesions started as intensely pruritic urticarial papules on the trunk and aggregated into polygonal plaques. **(B)** Histopathological features of prurigo chronica multiformis with ANA positivity. Slight vacuolar degeneration at the dermo-epidermal junction as well as eosinophilic infiltration in perivascular and intercollagenous spaces were observed.

16:5, p = 1.000). The median disease duration was slightly longer in patients with positive ANA compared with those without (8.0 vs. 5.0 months), although the difference was not statistically significant (p = 0.266).

On the other hand, of the 21 patients whose specific IgE levels were evaluated, the prevalence of sensitization to at least one allergen was significantly higher in patients with positive ANA (100%) compared with patients with negative ANA (37.5%, p = 0.035). In contrast, no significant differences were found in the frequency of elevated total IgE levels between the two groups (40.0% vs. 33.3%, p = 1.000).

Analysis of peripheral blood markers showed an inverse relationship between ANA status and eosinophilia; the median percentage of blood eosinophils was significantly lower in patients with positive ANA (4.0%; IQR: 2.3–5.6) compared with patients with negative ANA (8.0%; IQR: 5.5–13.7; p = 0.031). Correspondingly, the median absolute eosinophil count was also numerically lower in patients with positive ANA (399.0/μL) than in patients with negative ANA (798.0/μL), although this did not achieve statistical significance (p = 0.152). Serum TARC levels showed a high frequency of elevation in both groups, with no significant difference in the prevalence of elevated levels (66.7% vs. 75.0%, p = 1.000).

Regarding clinical severity and therapeutic outcomes, the median body surface area (BSA) affected by skin lesions was 22.0% in patients with positive ANA and 12.0% in patients with negative ANA, a difference that was not statistically significant (p = 0.708). Finally, the clinical response to topical corticosteroids was consistently poor in both subgroups; all five patients with positive ANA (100%) and 16 of 21 patients with negative ANA (76.2%) showed a poor response (p = 0.545).

A representative case of an ANA-positive PCM patient

A 74-year-old male presented with pruritic papules primarily on his trunk that had appeared 3 months prior to his initial visit to our department. The condition did not respond to treatment with oral dexchlorpheniramine maleate and topical difluprednate. The skin lesions started as intensely pruritic urticarial papules and aggregated into polygonal plaques (Figure 1A). The histopathological findings of the urticarial erythema on his back showed slight vacuolar degeneration at the dermo-epidermal junction as well as eosinophilic infiltration in perivascular and intercollagenous spaces (Figure 1B).

Laboratory testing revealed a low eosinophil count of 147/μL (2.6%) and an elevated serum IgE level of 2,812 IU/mL. Specific IgE antibodies were positive for Timothy grass, orchard grass, mugwort, Japanese cedar, Japanese cypress, Dermatophagoides farinae, wheat, soybean, rice, shrimp, crab, sesame, and peach by MAST36. ANA was positive at a titer of 1:40, showing both homogeneous and speckled patterns.

Based on the clinical and histopathological findings, a diagnosis of PCM was made. Despite treatment with topical clobetasol propionate ointment and oral olopatadine, the patient's eruption showed minimal improvement. Subsequently, the administration of oral prednisolone (15 mg/day) led to an adequate clinical response.

Discussion

To clarify the potential impact of dysregulated autoimmunity on the inflammatory profile of PCM, we divided 26 patients with PCM into ANA-positive and ANA-negative groups, and compared their clinical features, laboratory findings, and treatment responses. The ANA positivity rate in PCM patients was 19.2%. We found that specific IgE positivity to at least one allergen was significantly higher in patients with positive ANA (100%) compared with patients with negative ANA (37.5%, p = 0.035). In contrast, there was no significant difference in the frequency of elevated total IgE levels between the two groups (40.0% vs. 33.3%, p = 1.000). In addition, the median percentage of blood eosinophils was significantly lower in patients with positive ANA compared with patients with negative ANA (4.0% vs. 8.0%, p = 0.031).

Tan et al. reported an ANA positivity rate (≥1:40) of 31.7% in healthy individuals [9]. Furthermore, ANA positivity is known to increase with aging, and some ANA-positive cases in the present study may simply reflect nonspecific age-related autoantibodies. Nevertheless, the ANA-positive group demonstrated distinct immunological characteristics in PCM patients, suggesting that these findings cannot be explained solely by age-related changes.

In the future, we will need to be vigilant for complications associated with connective tissue diseases.

The ANA positivity rate (≥1:40) in AD has been reported to be 31.3% [7]. The rate in prurigo nodularis was 14.3%, although cutoff value was not indicated [8]. Thus, the ANA positivity rate in patients with PCM was lower than that in patients with AD and may be comparable to that in patients with prurigo nodularis. PCM shares several pathophysiological characteristics with these diseases, including Th2-dominant inflammation and chronic pruritus, and the ANA positivity in PCM observed in the present study may similarly reflect autoimmune dysregulation associated with chronic neuroimmune inflammation.

One of the most intriguing findings of the present study was the paradoxical hematological profile observed in the ANA-positive group of PCM. In general, strong allergic sensitization, as reflected by specific IgE positivity in all tested ANA-positive patients, is expected to be accompanied by increased peripheral eosinophil counts as part of a typical Th2 inflammatory response. However, recent studies have demonstrated that different biomarkers of Th2 inflammation may indicate distinct immunological axes, with eosinophils primarily reflecting IL-5-related inflammation and IgE more closely reflecting IL-4/IL-13-related inflammation [10]. Therefore, discordance between eosinophils and IgE may occur even within Th2-dominant inflammation. Our findings suggest that PCM may not be immunologically homogeneous, but may instead encompass multiple endotypes.

One possible explanation for the reduced eosinophil percentage in PCM patients with positive ANA is not depletion of eosinophils themselves, but rather enhanced recruitment of circulating eosinophils into lesional skin tissue, which represents the primary site of inflammation. Serum TARC levels were elevated in a high proportion of patients with PCM (73.9%), suggesting active migration of Th2 cells and eosinophils into the dermis. In patients with positive ANA, certain immunological triggers may induce eosinophil recruitment to the skin at a level exceeding the circulating eosinophil pool, thereby relatively reducing peripheral blood eosinophil percentages. The marked eosinophilic infiltration observed in perivascular and intercollagenous spaces in the representative histopathological specimen further supports this hypothesis.

Similar to PCM, chronic spontaneous urticaria (CSU), another type 2 inflammatory disorder, has been reported to involve both "type I autoallergy," mediated by IgE autoantibodies against self-antigens, and "type IIb autoimmunity," mediated by IgG autoantibodies against FcεRI or IgE [10, 11]. Patients with type IIb autoimmunity are clinically characterized by ANA positivity, low eosinophil and basophil counts, and treatment resistance. Although total IgE levels are often low in typical type IIb cases, overlap phenotypes between type I and type IIb autoimmunity have also been reported, and these patients do not necessarily exhibit low total IgE levels. A similar overlap-type immune dysregulation may also underlie ANA-positive PCM cases in the present study. Importantly, type IIb autoimmunity in CSU refers to mast cell activation mediated by autoantibodies against FcεRI or IgE and does not itself indicate ANA positivity. Nevertheless, the immunological features observed in ANA-positive PCM patients may suggest overlapping immune dysregulation involving both autoimmunity and autoallergy.

This study has several limitations. First, this was a single-center study with a relatively small sample size, which may limit the generalizability of our findings. Second, because this was a retrospective study, the assay and allergen panel were selected according to routine clinical practice and were therefore not identical among all patients. In the ANA-negative group, of the 16 patients tested, one underwent the View39 test and the remaining 15 underwent the MAST36 test. In the ANA-positive group, of the five patients tested, three underwent the MAST36 test, one underwent the View39 test and one underwent a single-allergen test, thus indicating that the assay and allergen panel did not affect the result. Lastly, we did not directly evaluate the relationship between peripheral blood eosinophil counts and actual eosinophilic infiltration in skin lesions. Therefore, the precise biological mechanisms of lower blood eosinophil percentage in patients with positive ANA are speculative. Alternative explanations, including aging, treatment intervention, and fluctuations in inflammatory status at the time of blood sampling, cannot be excluded. Larger multicenter studies incorporating quantitative histopathological and immunological analyses are required to validate our findings.

Because of its clinical heterogeneity and treatment resistance, PCM is a disorder that requires both high diagnostic accuracy and flexible therapeutic strategies in dermatological practice. In conclusion, the present study identified a unique endotype characterized by ANA positivity, reduced peripheral eosinophil percentages, and high allergen sensitization among patients with PCM. Rather than considering PCM a uniform inflammatory disorder, future studies should validate these findings and investigate the interplay between autoimmunity and allergic inflammation.

Data availability statement

The original contributions presented in the study are included in the article/supplementary material, further inquiries can be directed to the corresponding author.

Ethics statement

The studies involving humans were approved by Research Ethics Committee of Wakayama Medical University. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

KM and MJ contributed to the study design. KM, YI, and KK enrolled patients. KM, YI, and MJ contributed to the data analysis, and all authors contributed to interpretation of the data, critically revised the manuscript for intellectual content, and approved the final version of the manuscript for publication. All authors contributed to the article and approved the submitted version.

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Conflict of interest

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References

1. Satoh T, Yokozeki H, Murota H, Tokura Y, Kabashima K, Takamori K, et al. 2020 guidelines for the diagnosis and treatment of prurigo. *J Dermatol* (2021) 48(9): e414–31. doi: 10.1111/1346-8138.16067

2. Satoh T, Murota H, Aoyama Y, Hashimoto T, Ishiuji Y, Hatano Y, et al. Prurigo chronica multiformis: expert consensus of the Japanese dermatological association. *J Dermatol* (2024) 51(11):e376–83. doi: 10.1111/1346-8138.17487

3. Inui K, Ugajin T, Namiki T, Yokozeki H. Chronic prurigo: a retrospective study of 168 cases. *J Dermatol* (2020) 47(3):283–9. doi: 10.1111/1346-8138.15212

4. Ugajin T, Yokozeki H, Namiki T, Okiyama N. Dupilumab for prurigo chronica multiformis: a pilot case study. *J Cutan Immunol Allergy* (2024) 7:e12518. doi: 10.3389/jcia.2024.12518

5. Ishikawa T, Hashimoto T, Munetsugu T, Yokozeki H, Satoh T. Increased β-endorphin and autotaxin in patients with prurigo. *J Cutan Immunol Allergy* (2019) 2(4):94–101. doi: 10.1002/cia2.12062

6. Berger TG, Steinhoff M. Pruritus in elderly patients—eruptions of senescence. *Semin Cutan Med Surg* (2011) 30(2):113–7. doi: 10.1016/j.sder.2011.04.002

7. Ohkouchi K, Mizutani H, Tanaka M, Takahashi M, Nakashima K, Shimizu M. Anti-elongation factor-1α autoantibody in adult atopic dermatitis patients. *Int Immunol* (1999) 11(10):1635–40. doi: 10.1093/intimm/11.10.1635

8. Acer E, Ağaoğlu E, Kaya Erdoğan H, Çavuş HL, Öztürk BN, BīLgīN M, et al. Narrowband ultraviolet B phototherapy for dermatoses seen in elderly: a real-life experience. *Lasers Med Sci* (2026) 41(1):52. doi: 10.1007/s10103-026-04850-3

9. Tan EM, Feltkamp TEW, Smolen JS, Butcher B, Dawkins R, Fritzler MJ, et al. Range of antinuclear antibodies in "healthy" individuals. *Arthritis Rheum* (1997) 40(9):1601–11. doi: 10.1002/art.1780400909

10. Chovatiya R, Hawkes JE, DiRuggiero D, Pansch LA, Simcox E, Gonzalez T. Type 2 inflammation and its role in dermatologic diseases. *Int J Dermatol* (2025) 64(6): 978–91. doi: 10.1111/ijd.17707

11. Küçük ÖS, Yücel MB. Clinical and molecular aspects of managing chronic spontaneous urticaria: identifying endotypes, phenotypes, and determinants of treatment response and resistance. *Front Allergy* (2026) 6:1706705. doi: 10.3389/falgy.2025.1706705