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ORIGINAL ARTICLE

Real-life experience from a tertiary phototherapy center on solar urticaria: clinical features, management, and long-term outcomes from a twenty-year cohort

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ABSTRACT

BACKGROUND: Solar urticaria (SU) is a rare, chronic photodermatosis characterized by immediate whealing upon exposure to ultraviolet (UV) or visible light. Tolerance-induction phototherapy (photohardening), delivered as action-spectrum desensitization or cross-spectrum photohardening, represents the mainstay of management in antihistamine-refractory cases, yet data on its long-term real-world efficacy and safety remain limited. The aim of the study was to evaluate the clinical and photobiological characteristics, therapeutic outcomes, and long-term efficacy of tolerance-induction phototherapy in patients with SU managed at a tertiary phototherapy center.

METHODS: A retrospective observational study was conducted on 53 patients with phototest-confirmed SU treated with UVA or narrowband UVB (NB-UVB) phototherapy between January 2005 and June 2025. Treatment regimens were tailored to individual action spectra and, in selected cases, applied as cross-spectrum photohardening according to clinical practice and safety considerations. Clinical responses, phototolerance (minimal urticarial dose, MUD), and adverse events were recorded. Long-term outcomes, including remission persistence, relapse, and patient satisfaction, were assessed through complete follow-up (median 4.7 years).

RESULTS: At the end of therapy, 33 patients (62%) achieved complete remission, 15 (28%) partial improvement, and 5 (9%) showed no change; overall, 91% experienced at least partial benefit. Remission was maintained in 31 patients (58%), while 22 (42%) relapsed after a mean of 4.2±1.6 years. In patients with available post-treatment phototesting, those maintaining remission exhibited a mean fourfold increase in phototolerance. The mean satisfaction score was 8.8±0.7 (median 9). Only mild transient erythema (15%) and pruritus (7%) were reported, with no serious adverse events.

CONCLUSIONS: Tolerance-induction phototherapy with UVA or NB-UVB is a safe and highly effective first-line treatment for solar urticaria, providing durable phototolerance and long-term remission in most patients. The extended follow-up confirms its sustained efficacy and favorable safety profile in real-world clinical practice.

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KEY WORDS: Urticaria, solar; Phototherapy; Dermatology.

Solar urticaria (SU) is an infrequent yet potentially debilitating photodermatosis characterized by the rapid development of pruritic erythematous wheals on sun-exposed areas within minutes following exposure to ultraviolet (UV) or visible light.[1, 2] Although it accounts for less than 0.5 percent of all photodermatoses, SU can

substantially impair quality of life, resulting in significant restrictions in social interaction, occupational activity, and outdoor leisure pursuits.[1-3] The disease most frequently affects adults in the third to fifth decades of life, with a slight female predominance, although pediatric and late-onset presentations have also been described.[2-5] Familial clustering is exceptional, and no clear genetic predisposition has been identified. Environmental cofactors such as cumulative ultraviolet exposure, hormonal changes, and concurrent autoimmune conditions have been proposed as potential modulators of disease onset and severity.[3-5] The condition often follows a chronic relapsing course, with spontaneous resolution being uncommon.[3-5]

From an immunopathological perspective, SU is considered a type I immediate hypersensitivity reaction whereby photo-induced antigens function as allergens capable of cross-linking IgE on dermal mast cells.[6, 7] The absorption of photons by an endogenous chromophore produces a transient photoallergen that triggers mast-cell degranulation and the subsequent release of histamine, leukotrienes, and other mediators.[6-8] The consequent vascular leakage leads to the formation of characteristic wheals, which typically resolve within hours after cessation of exposure. Despite extensive research, the precise nature of the responsible chromophore remains unidentified; multiple mechanisms, ranging from abnormal porphyrin metabolism to altered oxidative pathways, have been proposed.[6-8]

Diagnosis relies on a combination of clinical history and phototesting, which identifies the action spectrum responsible for urticarial reactivity (UVA, UVB, visible, or mixed) and determines the minimal urticarial dose (MUD), defined as the lowest energy dose capable of eliciting a reproducible wheal.[1, 3, 5] Accurate identification of the action spectrum is essential for diagnostic confirmation and therapeutic planning, as it guides the selection of tolerance-induction strategies, including traditional action-spectrum desensitization and, in selected cases, cross-spectrum photohardening protocols aimed at enhancing functional sunlight tolerance.

Pharmacological treatment with H1-antihistamines offers partial symptom control in numerous patients but seldom prevents recurrence following light exposure.[9] Consequently, tolerance-induction phototherapy (photohardening), delivered either as action-spectrum desensitization (using the provoking wavelengths) or, in selected cases, as cross-spectrum photohardening (employing an alternative UV spectrum), has become the principal management strategy in persistent or antihistamine-refractory SU.[10-13] This approach aims to elevate the threshold of mast-cell activation, potentially through depletion of mast-cell mediators, induction of regulatory cytokines such as interleukin 10 (IL-10), and modification of antigen presentation.[6-8] UVA and narrowband UVB (NB-UVB) are the most commonly employed modalities.

Although several small series have demonstrated short-term efficacy, data regarding long-term outcomes, durability of remission, and persistence of phototolerance remain limited.[2, 14, 15] Moreover, few studies have integrated photobiological, clinical, and patient-reported data within a comprehensive real-world framework.[2, 6, 14] To address this gap, we conducted a twenty-year retrospective study involving patients with SU managed within a tertiary phototherapy unit. Our objectives were to characterize the clinical and photobiological features of affected individuals, evaluate the efficacy and safety of tolerance-induction phototherapy using UVA or NB-UVB, and assess the longevity of remission and patient satisfaction through a structured long-term follow-up. The aim was to provide a thorough, practice-based overview of solar urticaria management in routine clinical care and to elucidate the enduring impact of phototherapy on disease control and quality of life.

Materials and methods

This retrospective observational study included all patients referred to a tertiary phototherapy unit between January 2005 and June 2025 for suspected solar urticaria (SU). Of 167 consecutively evaluated subjects, 53 met the eligibility criteria and were included in the analysis. The study protocol was approved by the local Ethics Committee (authorization N. 4277) and conducted in accordance with the Declaration of Helsinki. Data were extracted from medical records, and follow-up information was obtained through scheduled outpatient visits and/or structured telephone interviews. Written informed consent was obtained from all participants for data collection and processing.

All patients underwent a comprehensive dermatologic assessment and standardized history taking, including age at onset, disease duration, latency after sun exposure, lesion distribution, suspected action spectrum, and prior treatments. Inclusion criteria were: 1) a clinical history consistent with SU; 2) a positive phototest confirming UVA and/or UVB reactivity through determination of the MUD; 3) completion of at least one full tolerance-induction (desensitizing) phototherapy cycle; and 4) adequate clinical documentation with a minimum follow-up of three months. Exclusion criteria included negative or inconclu-

sive phototesting, concomitant chronic spontaneous or physical urticarias, and current exposure to photosensitizing drugs.

Visible-light provocation testing was performed when clinically indicated by exposing a single 2-3 cm^2 skin area to visible light from a slide projector positioned 20 cm from the skin for 20 minutes. However, study eligibility required confirmed UVA and/or UVB reactivity; therefore, patients with isolated visible-light sensitivity in the absence of UVA/UVB reactivity were excluded. Patients with concomitant visible-light reactivity were managed according to their documented UV action spectrum and received the same tolerance-induction phototherapy protocol, together with reinforced photoprotection counselling given the limited efficacy of sunscreens against visible wavelengths.

A standardized laboratory work-up was performed in all patients, including complete blood count, serum chemistry, urinalysis, erythrocyte sedimentation rate (ESR), complement components (C3, C4), total complement activity (CH50), C-reactive protein (CRP), antinuclear antibodies (ANA), antidouble-stranded DNA, extractable nuclear antigen (ENA) profile, anti-Ro/SS-A, antineutrophil cytoplasmic antibodies (ANCA), and total serum IgE. In selected cases, urinary and erythrocytic porphyrins were assessed to exclude porphyric disorders or other photosensitivity syndromes.

Phototesting was performed on previously unexposed, untanned skin (typically the upper back) using calibrated broadband UVB (BB-UVB), NB-UVB and UVA sources. Baseline MUD and minimal erythemal dose (MED) were assessed by irradiating two series of six adjacent test areas (2-3 cm^2 each) with progressively increasing doses. For BB-UVB and NB-UVB, initial doses ranged from 0.01 to 0.057 J/cm^2, with each subsequent dose increased by a factor of 1.414 ($\sqrt{2}$). UVA test doses ranged from 1.0 to 5.7 J/cm^2. If no MED or MUD was elicited after the first series, a second series was performed 2-7 days later using higher doses (0.08-0.45 J/cm^2 for BB-UVB and NB-UVB; 8.0-45.3 J/cm^2 for UVA). Test sites were assessed during irradiation, immediately after exposure, and at 10 and 30 minutes, and at 1, 2, 4, 8, 24, and 48 hours. A reproducible erythematous wheal was considered a positive urticarial response.

In patients in whom no MUD could be identified despite repeated testing, MED was determined using the same exposure scheme with extended dose ranges (UVA 6-64 J/cm^2; UVB 0.06-2.0 J/cm^2). UVA irradiation for phototesting was delivered via a Medisun 250 phototherapy unit (Dr Hönle AG, Martinsried, Germany) equipped with 340-400 nm filters, while NB-UVB phototesting was performed using a Cosmedico TL-01 system (Philips, Eindhoven, the Netherlands) fitted with 20-W fluorescent tubes emitting at 311 nm. Visible-light provocation testing was performed with a slide projector carousel equipped with a 300-W bulb (Kodak AG, Stuttgart, Germany). UVA phototherapy was delivered using a PUVA Combi Light cabinet (model 8048; DERMAT bvba, Heverlee, Belgium) equipped with high-output 100-W UVA fluorescent tubes (total system power 5.2 kVA), and NB-UVB phototherapy was delivered with a W7001k cabinet (Waldmann Lichttechnik GmbH, Schwenningen, Germany) equipped with 48 Waldmann TL01/100-W fluorescent tubes.The tolerance-induction (photohardening) phototherapy regimen was tailored to each patient's photosensitivity spectrum and safety considerations. Patients reactive to UVA received NB-UVB phototherapy, while those reactive to UVB underwent UVA phototherapy (cross-spectrum photohardening).[11] Both protocols followed an intensive desensitization regimen comprising three daily sessions at four-hour intervals for five consecutive days (Monday to Friday) during the initial treatment week. The starting dose was set at approximately 50% of the individual threshold for the administered modality (MUD when measurable for that spectrum; otherwise MED), with incremental increases of 10–20% per session, provided no urticarial or phototoxic reactions occurred. Irradiation initially targeted the upper trunk, face, and upper limbs, and was progressively extended to the lower trunk and legs if tolerated. For safety and tolerance reasons, phototherapy was administered as sequential half-body exposures (anterior and posterior), performed at 20-30-minute intervals to reduce thermal load and minimize urticarial flares. Patients tolerating the initial intensive phase continued with once-daily exposures, three times weekly, for an additional three to six weeks. All subjects received oral cetirizine 10 mg/day during the first week to mitigate histamine-mediated reactions. If treatment was interrupted due to erythema or urticarial recurrence, therapy was paused until complete resolution and then resumed at 50% of the last tolerated dose. For patients where MUD determination was not feasible, phototherapy followed a prophylactic protocol modeled after polymorphous light eruption, beginning at 70% of MED with incremental adjustments based on erythemal response. Following the phototherapy cycle, patients were advised to gradually expose themselves to natural sunlight at midday (12:00-14:00) for 20-30 minutes, two or three times per week during spring and summer, to consolidate phototolerance. All patients received standardized pho-

toprotection counselling, including behavioral avoidance strategies and daily use of broad-spectrum, high-SPF sunscreens with high UVA protection.

Clinical assessments were conducted at one- and three-months post-treatment to evaluate symptom recurrence and phototolerance. Whenever feasible, phototesting (including MUD reassessment) was repeated at the end of the photohardening cycle and/or at early follow-up visits; however, systematic post-treatment MUD reassessment was not available for all patients due to the retrospective design and logistical constraints. Long-term outcomes were systematically obtained for all patients through periodic outpatient evaluations and/or structured telephone interviews, ensuring complete follow-up. Data recorded included remission status, time to relapse, duration of phototolerance, and patient satisfaction on a 0-10 Visual Analogue Scale (VAS; 0 = no satisfaction, 10 = highest satisfaction), assessed at the last available follow-up visit or structured telephone interview for each patient.

Statistical analysis

Continuous variables are reported as mean ± standard deviation (SD) or median (interquartile range, IQR), categorical variables as counts and percentages. All analyses were performed using IBM SPSS Statistics version 29 (IBM Corp., Armonk, NY, USA).

Results

A total of 53 patients (30 males and 23 females; mean age 45±13 years, range 21–74) were included in the analysis. The patients exhibited Fitzpatrick skin phototypes II (32%), III (45%), and IV (23%). The mean latency between sun exposure and wheal onset was 9 minutes (range 3-25). The action spectrum was attributed to UVA in 47%, UVB in 43%, and mixed UVA/UVB in 10% of cases. The visible-light provocation test (slide projector) was positive in 23% of patients. Phototherapy allocation did not necessarily mirror action-spectrum categories, as treatment was frequently delivered as cross-spectrum photohardening in routine practice; accordingly, the distribution of administered modalities (NB-UVB N.=26; UVA N.=22; mixed N.=5) differs from the provoking action spectrum (UVA N.=25; UVB N.=23; mixed N.=5) (Figure 1; Table I).

Photobiological parameters indicated a mean MUD of 7.6±2.3 J/cm² (range 3.2-13.0) for UVA and 0.22±0.07 J/cm² (range 0.09-0.37) for UVB. Additionally, the mean MED in instances lacking measurable MUD was 23.8±6.1 J/cm² for UVA and 1.2±0.4 J/cm² for UVB.

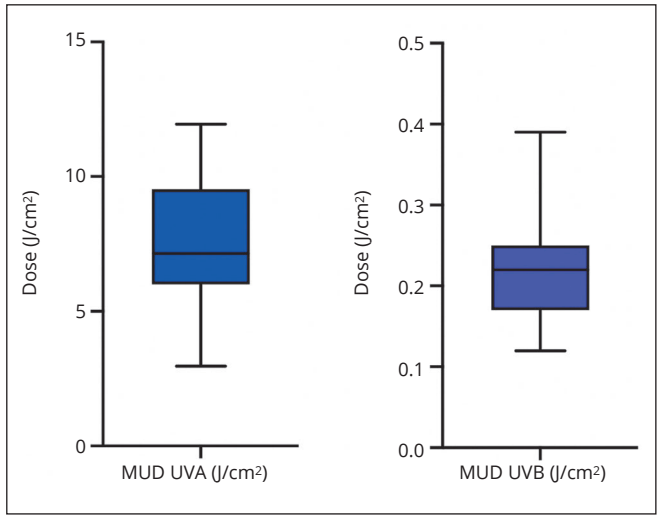

Figure 1.—The box-and-whisker plots illustrate the distribution of the MUD for UVA and UVB spectra within the patient cohort. The boxes denote the IQR, with horizontal lines representing medians, and the whiskers extending to 1.5 times the IQR. The UVA and UVB measurements are expressed in joules per square centimeter (J/cm²). Blue boxes indicate UVA (315-400 nm), while purple boxes represent UVB (280-315 nm).
IQR: interquartile range; MUD: minimal urticarial dose.

All patients completed at least one full tolerance-induction (photohardening) cycle. The median number of phototherapy sessions was 17 (IQR 6) overall, with a cumulative dose approximately 2.0±0.3 times the individual MUD. Twenty-six patients (49%) underwent NB-UVB phototherapy and 22 (42%) received UVA, while the remaining 5 (9%) received mixed regimens based on dual-spectrum sensitivity. Extended treatment cycles were needed in 10 (19%) of patients, and 6 (11%) underwent a second cycle within the same year due to symptom recurrence. When stratified by treatment modality, the median number of sessions was 18 (IQR 5) for NB-UVB and 19 (IQR 5) for UVA phototherapy. The number of patients treated with mixed regimens was limited (N.=5), precluding a meaningful stratified analysis. Overall, treatment was well tolerated. Mild erythema occurred in 8 (15%) of patients, transient pruritus in 4 (7%), and short-lived urticarial flares in 3 (5%). No severe adverse events were reported, and only one patient (2%) temporarily discontinued phototherapy due to moderate erythema.

By the end of the desensitization protocol, a complete clinical response, defined as the absence of whealing after sunlight exposure, was achieved in 33 (62%) patients, while 15 (28%) experienced partial improvement and 5 (9%) showed no significant change in photosensitivity. Overall, 48 (91%) of patients demonstrated at least partial remission.

TABLE I.—*Clinical and photobiological characteristics of 53 patients with solar urticaria treated with desensitizing phototherapy.*

Total patients, N.	53
Sex, N. (%)	Males 30 (56.6%), Females 23 (43.4%)
Age, years (mean±SD, range)	45±13 (21-74)
Fitzpatrick skin phototype, N. (%)	II: 17 (32%), III: 24 (45%), IV: 12 (23%)
Latency after sun exposure, minutes (mean, range)	9 (3-25)
Action spectrum, N. (%)	UVA: 25 (47%), UVB: 23 (43%), Mixed: 5 (10%)
Visible-light provocation test (slide projector), positive N. (%)	12 (23%)
MUD	
UVA (J/cm^2, mean±SD, range)	7.6±2.3 (3.2-13.0)
UVB (J/cm^2, mean±SD, range)	0.22±0.07 (0.09-0.37)
MED	
UVA (J/cm^2, mean±SD)	23.8±6.1 (14-35.1)
UVB (J/cm^2, mean±SD)	1.2±0.4 (0.6-2.0)
Median number of phototherapy sessions (IQR)	17 (6)
Cumulative dose (× MUD, mean±SD)	2.0±0.3
Phototherapy type, N. (%)	NB-UVB: 26 (49%), UVA: 22 (42%), Mixed: 5 (9%)
Extended cycle required, N. (%)	10 (19%)
Second cycle within 12 months, N. (%)	6 (11%)
Adverse events, N. (%)	Mild erythema: 8 (15.1%); pruritus: 4 (7.6%); urticarial flare: 3 (5.7%); severe AE: 0 (0%)
Complete clinical response, N. (%)	33 (62%)
Partial improvement, N. (%)	15 (28%)
No response, N. (%)	5 (9%)
At least partial remission, N. (%)	48 (91%)
Follow-up duration, months (median, IQR)	56 (25-94)
Remission maintained, N. (%)	31 (58%)
Relapse, N. (%)	22 (42%)
Time to relapse, months (mean±SD)	50±19
Increase in phototolerance (fold), when post-treatment MUD reassessment was available	4×
Patient satisfaction (VAS, mean±SD, median, range)	8.8±0.7; 9 (7-10)
Improved sunlight tolerance at final follow-up, N. (%)	29 (55%)

Continuous variables are expressed as mean±SD or median (IQR), and categorical variables as frequencies and percentages.
IQR: interquartile range; SD: standard deviation; MUD: minimal urticarial dose; MED: minimal erythemal dose; NB-UVB: narrowband ultraviolet B; AE: adverse event; VAS: Visual Analogue Scale.

The median follow-up duration was 4.7 years (IQR 2.1-7.8 years). During this period, remission was maintained in 31 (58%) while 22 (42%) experienced relapse after a mean of 4.2±1.6 years. Post-treatment MUD reassessment was available in a subset of patients; within this subgroup, those maintaining long-term remission exhibited a mean fourfold increase in phototolerance (based on MUD). Patients maintaining remission exhibited a mean fourfold increase in phototolerance (based on the MUD). Subjective satisfaction remained high, with a mean VAS of 8.8±0.7 (median = 9, range = 7-10). At the last follow-up, 55% reported sustained improvement in tolerance to natural sunlight.

Discussion

This 20-year experience provides one of the most extensive real-world datasets on desensitizing phototherapy for SU. The high overall response rate – over 90% of patients achieving at least partial improvement – confirms the central role of controlled phototherapy in the management of this rare and often disabling photodermatosis.[2, 10-12, 14, 15] The complete clinical remission observed in 62% of patients and the persistence of disease control in nearly two-thirds at long-term follow-up are consistent with previously published series, reinforcing the reproducibility of these results in daily clinical practice.[2, 10-12, 14, 15] Comparable response rates have been reported by Imamura *et al*.[14] for UVA and by Snast *et al*.[15] for NB-UVB, supporting the effectiveness of both modalities regardless of the initiating spectrum. These findings collectively indicate that the key determinant of efficacy lies in the immunophysiological adaptation induced by repeated subthreshold irradiation rather than in the specific wavelength employed.[10, 13]

The mechanisms underlying the induction and maintenance of phototolerance are complex and likely multifactorial. Controlled irradiation below the urticarial threshold depletes mast-cell granules, modifies cytokine profiles,

and downregulates the expression of IgE and FcεRI on target cells.[6-8] UVA, with its deeper dermal penetration, may primarily affect dermal mast cells and dendritic cells, whereas NB-UVB exerts more superficial effects on epidermal and papillary dermal structures, enhancing the release of immunoregulatory mediators such as prostaglandin E_2, IL-10, and TGF-β.[6-8] Emerging evidence also suggests that phototherapy modulates neuroimmune signaling pathways, raising the cutaneous activation threshold and contributing to durable desensitization.[6-8] Together, these mechanisms explain the capacity of both UVA and NB-UVB to induce long-lasting tolerance irrespective of the provoking wavelength. Similar tolerance-induction strategies, including rush hardening and NB-UVB-based protocols, have been reported in real-life series, supporting the feasibility of achieving clinical phototolerance even when the administered spectrum does not strictly mirror the provoking wavelength.[10-13, 15]

From a patient-centered standpoint, the present study demonstrates that desensitizing phototherapy not only suppresses acute urticarial reactivity but also restores functional phototolerance and psychosocial well-being. More than half of the patients reported lasting improvement in sunlight tolerance and regained confidence in outdoor and occupational activities. The high mean satisfaction score (VAS 8.8±0.7) underscores the strong perceived benefit and real-world acceptability of this approach. Importantly, all patients were followed for a median of 4.7 years, one of the longest observational periods reported to date. During this extended follow-up, 58% maintained long-term remission, whereas 42% experienced relapse after a mean of 4.2 years. These data confirm that phototherapy-induced tolerance can persist for several years and suggest that periodic low-dose UV exposure or maintenance desensitization cycles may help prevent late relapse.[10-12, 14, 15]

The safety profile observed was excellent. Only mild, transient erythema (15%) and pruritus (7%) were recorded, with no systemic or delayed adverse effects. This confirms the well-established tolerability of UVA and NB-UVB, consistent with their use in other chronic photodermatoses such as polymorphous light eruption and chronic actinic dermatitis, where repeated cycles are administered safely without cumulative toxicity.[10-13] The absence of long-term adverse effects in our cohort supports the feasibility of repeated seasonal or maintenance treatments in patients with recurrent diseases.

Pharmacological options for SU remain limited. H1-antihistamines provide partial symptomatic relief but rarely prevent light-induced whealing.[1, 9] Omalizumab has shown variable efficacy in small case series and a phase II trial,[16-19] and intravenous immunoglobulins have produced benefit in selected severe cases.[20, 21] Cyclosporine A has demonstrated only modest results,[22] while broad-spectrum sunscreens combined with antihistamines may offer additional protection in some patients.[23] In this therapeutic context, desensitizing phototherapy stands out as a non-pharmacologic, pathogenetically oriented intervention that addresses the underlying hypersensitivity rather than merely alleviating symptoms.[10-13]

Limitations of the study

The strengths of this study include its complete and prolonged follow-up, the uniform photobiological characterization of all cases, and the standardized therapeutic protocol applied in a single tertiary center. Limitations derive from its retrospective nature, the absence of randomization between UVA and NB-UVB, and the reliance on patient-reported outcomes for long-term assessment. Nonetheless, the extended duration of observation and the consistency of results across different spectral sensitivities provide robust real-world evidence supporting the long-term efficacy and safety of desensitizing phototherapy.

Future research should aim to elucidate the molecular correlates of phototolerance induction, integrate standardized patient-reported outcome measures (*e.g.*, DLQI, UV-QOL), and evaluate predictors of sustained remission in prospective multicenter studies.[6, 7, 24, 25] Combining suberythemal phototherapy with targeted biologics such as omalizumab or ligelizumab may represent a promising synergistic approach to enhance and prolong tolerance while minimizing cumulative UV exposure.[16-19]

Conclusions

In conclusion, desensitizing phototherapy remains the primary treatment modality for confirmed solar urticaria, combining efficacy, safety, and long-term durability unmatched by alternative therapies. Given its reproducibility and favorable safety profile, it should be maintained as the first-line approach and incorporated into forthcoming evidence-based management guidelines.

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Conflicts of interest

The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' contributions

Stefano Bighetti, Piergiacomo Calzavara-Pinton, Marina Venturini, and Mariateresa Rossi have given substantial contributions to the study conception and design; Luca Bettolini, Marina Venturini, and Benedetta Galli contributed to the data acquisition, to the clinical management of patients, and to the database curation; Isacco Cattaneo and Alessia Loda contributed to the data analysis and interpretation; Andrea Carugno, Nicola Zerbinati, and Mariateresa Rossi contributed to the manuscript critical revision for important intellectual content and to the contextualization of findings within the current literature; Stefano Bighetti contributed to the manuscript draft, and Marina Venturini and Mariateresa Rossi revised it critically. All authors read and approved the final version of the manuscript.

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