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Photosensitivity in Mycosis Fungoides: From Photosensitive Mycosis Fungoides to Narrowband Ultraviolet B-Induced Abnormal Light Reactions

Tadamichi Shimizu [1]

1. Department of Dermatology, University of Toyama, Toyama, JPN

Corresponding author: Tadamichi Shimizu, shimizut@med.u-toyama.ac.jp

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Abstract

Photosensitivity in mycosis fungoides (MF) includes sunlight-induced or exacerbated lesions, photodistributed disease, eruptions resembling chronic actinic dermatitis (CAD), and abnormal light reactions (ALRs) during phototherapy. This review examines their clinical features, differential diagnosis, and management, with particular attention to narrowband ultraviolet B (NB-UVB)-induced ALRs. Actinic reticuloid, now considered part of the CAD spectrum, can be difficult to distinguish from MF because of overlapping clinical and histopathologic features. Photosensitive MF may show abnormal responses to ultraviolet A, ultraviolet B, or visible light. Some patients with MF also develop erythema, irritation, or pruritus after low-dose NB-UVB irradiation, although standardized diagnostic criteria for ALRs have not been established. Evaluation should consider irradiation parameters, clinical manifestations, medication history, phototesting, histopathology, and disease course. Management may include temporary interruption or dose reduction of NB-UVB, treatment of cutaneous inflammation, and targeted phototherapy for localized disease. Photosensitive MF and NB-UVB-induced ALRs may overlap clinically but are not necessarily identical. Further studies are needed to clarify their relationship, establish diagnostic criteria, and determine their mechanisms and clinical significance.

Categories: Oncology, Dermatology
Keywords: abnormal light reaction, chronic actinic dermatitis, mycosis fungoides (mf), narrowband ultraviolet b (nb-uvb), skin photosensitivity

Introduction And Background

Mycosis fungoides (MF) is a primary cutaneous T-cell lymphoma (CTCL) characterized by the proliferation of epidermotropic neoplastic T cells [1]. Treatment options for early-stage MF include topical corticosteroids, phototherapy, and radiotherapy. Narrowband ultraviolet B (NB-UVB) is widely used for patch-stage and selected plaque-stage disease because of its efficacy and practicality [2]. However, some patients develop erythema, irritation, or pruritus at relatively low NB-UVB doses [3].

Photosensitivity in MF has been reported since the 1970s, with abnormal responses to UVA, UVB, or visible light [4-6]. Photosensitive MF has been described in patients whose lesions are induced or exacerbated by sunlight or whose phototesting demonstrates abnormal responses. However, no uniform definition of photosensitive MF has been established.

The reported cases of photosensitive MF remain limited, and its frequency, clinical characteristics, and pathogenesis are incompletely understood. Erythema during phototherapy presents a diagnostic challenge because possible causes include an expected erythemal response, excessive irradiation, drug-induced photosensitivity, chronic actinic dermatitis (CAD), exacerbation of MF, and an abnormal light reaction (ALR). In our previous single-center study, we investigated ALR during NB-UVB therapy in patients with MF and used an operational definition based on erythema developing in clinically uninvolved skin after low-dose irradiation [3]. This definition has not been established as a general diagnostic criterion.

Accurate recognition of these reactions is clinically important because appropriate management may prevent unnecessary discontinuation of potentially effective phototherapy.

This review examines the relationship between photosensitive MF, CAD, and actinic reticuloid, with particular attention to the clinical features of NB-UVB-induced ALR, its differential diagnosis, possible pathogenesis, and management.

Review

Literature search and selection

This narrative review was based on relevant publications identified through PubMed searches updated through September 30, 2026. Search terms included "mycosis fungoides," "photosensitivity," "chronic actinic

dermatitis," "actinic reticuloid," "narrowband UVB," and "abnormal light reactions." The search terms were used in various combinations to identify publications relevant to photosensitivity, photodermatoses, and phototherapy in MF.

The PubMed search results were reviewed by the author, who selected publications based on their relevance to the scope of this review. In addition, key historical and contemporary publications previously known to the author and relevant references identified through citation tracking were considered. This approach also allowed the inclusion of relevant publications not indexed in PubMed. Publications unrelated to photosensitivity in MF, CAD, actinic reticuloid, and NB-UVB-induced abnormal light reactions were excluded.

The available evidence was synthesized qualitatively. This article is a narrative review rather than a systematic review or meta-analysis. No meta-analysis, meta-regression, or quantitative evidence synthesis was performed. No formal study quality or risk of bias assessment was conducted.

In synthesizing the literature, we distinguished findings from individual case reports and case series, observational studies, and expert consensus recommendations, taking the nature and limitations of each type of evidence into consideration.

Chronic Actinic Dermatitis and Actinic Reticuloid

Chronic actinic dermatitis is a persistent photodermatosis characterized by eczematous lesions predominantly affecting sun-exposed skin. The CAD spectrum includes conditions historically described as photosensitive eczema, persistent light reaction, and actinic reticuloid [7,8]. CAD occurs mainly in older men and commonly affects the face, neck, upper chest, and dorsal hands. Clinical findings include intensely pruritic erythema, papules, infiltrated plaques, and lichenification. Clothing-covered areas are often relatively spared, although severe disease may extend to nonexposed skin and occasionally become erythrodermic.

The action spectrum of CAD is predominantly in the UVB range, although UVA and, in some patients, visible light may also provoke reactions. Phototesting may reveal a reduced minimal erythema dose (MED), persistent erythema, or eczematous lesions after repeated irradiation. Patch testing or photopatch testing may identify relevant contact allergens in some patients [8].

Actinic reticuloid is now regarded as a historical term within the CAD spectrum. It described severe disease with marked infiltration and lichenification and clinical and histopathologic features resembling cutaneous lymphoma. In 1969, Ive et al. reported 10 patients with severe persistent photosensitivity and lymphoma-like histopathologic findings [9]. Histopathology may show spongiosis, epidermal hyperplasia, and a dense dermal lymphocytic infiltrate, sometimes including atypical lymphocytes. These overlapping features have complicated the distinction between actinic reticuloid and MF.

Concept and Clinical Features of Photosensitive Mycosis Fungoides

Volden and Thune evaluated 12 patients with suspected photosensitivity associated with MF and confirmed abnormal phototest responses in 7 [4]. The provoking wavelengths were mainly in the UVA range, although reactions to UVB and visible light were also observed. In some patients, photosensitivity appeared after MF progression. In a subsequent report of 11 patients, Volden reported abnormal responses to UVB in 4 patients, UVA in 4, and visible light in 1 [5]. Of these 11 patients, 5 had also been described in the report by Volden [6]; therefore, the two reports partly overlap and should not be regarded as entirely independent patient cohorts. These reactions normalized after complete remission of the cutaneous MF lesions [5]. A further study of nine patients found abnormal UVA and UVB responses in approximately one-third of patients, with greater photosensitivity observed closer to MF lesions [6]. Together, these findings suggest that photosensitivity may be associated with MF activity or the cutaneous disease process, although a causal relationship has not been established.

De Silva et al. reported two patients initially diagnosed with actinic reticuloid whose early biopsies did not demonstrate clonal T-cell receptor gene rearrangement. The eruptions subsequently became more extensive, and later evaluation showed histopathologic findings consistent with MF and clonal T-cell receptor gene rearrangement [10]. The authors considered whether these cases represented photosensitive MF from the outset or whether MF developed over time in patients initially diagnosed with actinic reticuloid. These cases illustrate the importance of longitudinal assessment and repeat biopsy when the diagnosis remains uncertain.

In Japan, Seki et al. reported a patient with MF whose eruption and pruritus worsened after NB-UVB therapy was resumed. Phototesting demonstrated photosensitivity to UVA and UVB, and clonal T-cell receptor gene rearrangement was identified in the skin lesions [11]. Haber et al. described MF with a predominantly photodistributed eruption and photosensitivity, proposing that this presentation might represent a distinct clinical variant [12]. Sinha et al. subsequently reported papules and plaques aggravated by sunlight on the

forehead, dorsal hands, and forearms. Although CAD was considered clinically, histopathologic and immunohistochemical findings supported MF [13].

Additional reports have highlighted the diagnostic overlap between photosensitive MF and other photodermatoses. Agar et al. described four patients with erythrodermic cutaneous T-cell lymphoma and severe photosensitivity mimicking chronic actinic dermatitis (CAD), with reduced minimal erythema doses in both UVA and UVB ranges [14].

Nair et al. reported an aggressive form of photosensitive MF presenting with clinical features mimicking systemic lupus erythematosus. The patient had photodistributed erythematous lesions involving the face and forearms, illustrating the potential for photosensitive MF to resemble autoimmune disease [15]. Karadia et al. subsequently reported a patient with MF presenting with multiple atypical morphologies in a photoexacerbated distribution, further illustrating the clinical variability of photosensitive MF [16]. Oren-Shabtai et al. described eight patients with MF coexisting with photosensitive or autoimmune diseases, highlighting the therapeutic challenges associated with these comorbidities. Because the series included different types of comorbid conditions, its findings should not be interpreted as evidence that all patients had photosensitive MF [17].

CAD and photosensitive MF may both present with photodistributed lesions and abnormal phototest results. Consequently, lesion distribution and action spectrum alone may not distinguish them reliably [7,8,10-14]. Actinic reticuloid may also show lymphoma-like histopathologic features [9,14]. Immunohistochemistry and T-cell receptor gene analysis can provide additional diagnostic information, but neither should be interpreted in isolation. Diagnosis requires clinicopathologic correlation, including lesion morphology, phototest results, histopathology, molecular studies when appropriate, and changes over time [10,14,18].

Based on published reports, photosensitivity associated with MF can be considered in three overlapping clinical patterns: symptoms such as erythema, irritation, or pruritus triggered or exacerbated by sunlight; MF lesions predominantly affecting sun-exposed sites; and persistent eczematous or lichenified eruptions resembling CAD. These patterns are not mutually exclusive, and it remains uncertain whether abnormal phototest responses, photodistributed MF, and low-dose NB-UVB-induced erythema in clinically uninvolved skin share a common biological mechanism. Assessment should consider lesion distribution, provoking wavelengths, reproducibility, and the temporal relationship between photosensitivity and MF activity, while also evaluating alternative photodermatoses and drug-induced photosensitivity.

Although photosensitive MF and NB-UVB-induced abnormal light reactions both involve abnormal responses to light, they represent distinct clinical phenomena. Photosensitive MF is characterized by sunlight-associated induction or exacerbation of MF lesions, whereas NB-UVB-induced abnormal light reactions occur during therapeutic irradiation, often presenting as erythema in clinically uninvolved skin. Distinguishing these phenomena is important for evaluating photosensitivity and optimizing phototherapy in patients with MF.

Table *1* summarizes the clinical features of CAD, photosensitive MF, and NB-UVB-induced ALR.

Characteristic	CAD	Photosensitive mycosis fungoides	NB-UVB-induced ALR
Clinical context	An established chronic photodermatosis [7,8]	MF associated with sunlight-induced or phototest-confirmed photosensitivity; no uniform definition has been established [4-6,10-13,15,16]	An erythemal reaction occurring after low-dose NB-UVB irradiation in patients with MF [3]
Distribution	Persistent eczematous lesions predominantly affecting sun-exposed areas, with relative sparing of protected sites [7,8]	MF lesions may develop or worsen predominantly in sun-exposed areas; distribution varies among patients [10-13,15,16]	Erythema in clinically uninvolved skin exposed to NB-UVB, according to the definition used in our study [3]
Relevant wavelengths	Predominantly UVB; UVA and, in some patients, visible light may also be involved [7,8]	Abnormal responses to UVA, UVB, or visible light have been reported; the action spectrum varies among patients [4-6]	NB-UVB [3]
Main clinical manifestations	Eczematous erythema, papules, infiltrated plaques, lichenification, and pruritus [7,8]	Induction or exacerbation of MF lesions; erythema and pruritus may accompany the lesions [10-13,15,16]	Erythema, irritation, and pruritus [3]
Phototesting/irradiation response	Reduced MED, persistent erythema, and/or a positive repeated-irradiation test [8]	Abnormal responses to UVA, UVB, or visible light [4-6]	In our study, ALR was defined as erythema developing in clinically uninvolved skin at an NB-UVB dose of ≤0.5 J/cm² [3]
Histopathology	Chronic eczematous changes; severe cases may show pseudolymphomatous features [7-9]	Histopathologic findings consistent with MF [10,11,13]	No histopathologic findings specific to ALR have been established [3]
Clinical management	Photoprotection, avoidance of relevant allergens when identified, and anti-inflammatory treatment [8]	MF-directed treatment, photoprotection, and assessment of the provoking wavelengths [2,11-13,15]	Reduction or temporary discontinuation of NB-UVB; targeted phototherapy (e.g., 308-nm light/laser) may be considered [2,3,19]

TABLE 1: Clinical comparison of CAD, photosensitive mycosis fungoides, and NB-UVB-induced abnormal light reaction

Note: The NB-UVB dose threshold of ≤0.5 J/cm² represents the definition used in our study and is not an established diagnostic criterion for NB-UVB-induced ALR.

ALR: abnormal light reaction, CAD: chronic actinic dermatitis, MED: minimal erythema dose, MF: mycosis fungoides, NB-UVB: narrowband ultraviolet B

Table *2* summarizes representative published reports relevant to photosensitivity in MF, including related CTCL and comorbidity reports, as well as NB-UVB-induced ALR. The table presents patient numbers, phototesting findings, and major clinical features. These reports illustrate the clinical overlap between photosensitive MF and other photodermatoses, as well as the distinct clinical context of NB-UVB-induced ALR.

Study	Patients (number)	Photosensitivity/phototesting findings	Key clinical findings
Photosensitive MF and related reports			
De Silva et al. (2000) [10]	2	Reduced MEDs at 305-430 nm in patient 1; delayed photosensitivity to UV and visible light in patient 2	Two patients initially diagnosed with actinic reticuloid later developed widespread MF, including lesions on sun-protected skin; subsequent biopsies and T-cell receptor gene analysis supported MF
Agar et al. (2009)	4	Reduced MEDs throughout the UVA and UVB ranges	Four patients with erythrodermic CTCL had marked photosensitivity mimicking CAD; clonal T cells were identified in skin and blood or lymph nodes; all

[14]			subsequently died from systemic disease
Seki et al. (2013) [11]	1	UVB MED, 10 mJ/cm²; UVA irradiation induced erythema at 5 J/cm² but not at 1 J/cm²	After MF relapsed following chemotherapy, NB-UVB caused marked itching and irritation on irradiated skin even at 0.1 J/cm², leading to discontinuation; lesions improved with PUVA using lesion-limited psoralen application
Haber et al. (2017) [12]	2	Reduced MEDs to UVB, UVA, and visible light in both patients	Two men with photodistributed erythematous infiltrated lesions mimicking CAD; both had biopsy findings consistent with MF; one improved with romidepsin and the other with bexarotene
Sinha et al. (2021) [13]	1	Sunlight-associated exacerbation; UVA/UVB phototesting not reported; patch and photopatch tests negative	Photodistributed MF mimicking CAD, with lesions on the forehead, dorsal hands, and forearms
Nair et al. (2024) [15]	1	Photodistributed photosensitivity; phototesting findings not reported	Aggressive MF mimicking systemic lupus erythematosus, with erythematous macules, papules, and plaques on the face and forearms; the patient died before treatment
Karadia et al. (2025) [16]	1	Photoexacerbated distribution; phototesting findings not reported	MF presenting with multiple atypical morphologies in a photoexacerbated distribution
Oren-Shabtai et al. (2025) [17]	8	Coexisting photosensitive or autoimmune diseases; individual phototesting findings not reported	Retrospective series of patients with MF with heterogeneous photosensitive or autoimmune comorbidities; not all patients had photosensitive MF
Abnormal light sensitivity in MF			
Volden and Thune (1977) [4]	12	Abnormal responses in 7/12 patients, predominantly to UVA, with some responses to UVB and visible light	Sun-induced burning and erythema in 4 patients after progression to stage II
Volden (1980) [5]	11	Abnormal responses: UVB (4), UVA (4), and visible light (1)	Responses normalized after complete remission; 2 PUVA-treated patients had early burning and soreness on lesional skin
Volden (1980) [6]	9	Phototesting near lesions and at 3 and 10 cm; abnormal UVA/UVB responses in approximately one-third	Greater photosensitivity was demonstrated closer to MF lesions
NB-UVB-induced ALR			
Mizawa et al. (2025) [3]	40 (6 with ALR; 15%)	ALR was defined as erythema in clinically uninvolved skin after NB-UVB irradiation at ≤0.5 J/cm² (6/40, 15%); phototesting in one patient showed a broadband UVB MED of 50 mJ/cm² and an NB-UVB MED of 0.3 J/cm²; no abnormal UVA response was observed	Erythema with irritation and/or pruritus; onset within 1 month of starting NB-UVB

TABLE 2: Summary of published reports on photosensitive mycosis fungoides and ALR

Notes: Agar et al. reported four patients with erythrodermic CTCL and severe photosensitivity mimicking CAD; this related CTCL series should not be interpreted as four cases of photosensitive MF [14]. Of the 11 patients in Volden [5], 5 had also been reported in Volden [6]; these patient groups therefore overlap and should not be summed as independent cohorts. The Oren-Shabtai et al. series included heterogeneous photosensitive or autoimmune comorbidities and should not be interpreted as eight cases of photosensitive MF [17]. In Mizawa et al., the ≤0.5 J/cm² threshold was the study-specific operational definition of ALR, not an established diagnostic criterion [3].

ALR: abnormal light reaction, CAD: chronic actinic dermatitis, CTCL: cutaneous T-cell lymphoma, MED: minimal erythema dose, MF: mycosis fungoides, NB-UVB: narrowband ultraviolet B, PUVA: psoralen plus ultraviolet A, UVA: ultraviolet A, UVB: ultraviolet B

Clinical Features of Abnormal Light Reactions During Narrowband Ultraviolet B Therapy

During NB-UVB phototherapy, the starting dose is generally determined by the minimal erythema dose (MED) or a test exposure, and erythema, irritation, and pruritus are monitored during dose escalation [2,19]. Expert-based consensus recommendations have also addressed the indications and treatment schedules for NB-UVB and PUVA in MF [20]. More recently, the GEF-CILAD 2026 update reaffirmed NB-UVB as a treatment option for early-stage MF and provided updated recommendations regarding its clinical use and safety [21]. Because NB-UVB normally causes dose-dependent erythema, a reaction should not be classified as an ALR solely because it occurs after irradiation. Assessment should consider the dose, intensity and duration of erythema, associated symptoms, whether erythema occurs in lesional or clinically uninvolved skin, and reproducibility across treatment sessions.

In our single-center study, 40 patients with MF who received NB-UVB therapy were analyzed. Patients receiving bexarotene or etretinate were excluded. ALR was defined as erythema developing in clinically uninvolved skin after NB-UVB irradiation at a dose of 0.5 J/cm² or less [3]. Of 40 patients, 6 (15.0%; 95% CI: 7.1%-29.8%) met this definition, and all reactions occurred within the first month of treatment. Manifestations included erythema, irritation, and pruritus; some patients developed extensive erythema. None had a clear history of photosensitivity in daily life or an evident exposure to medications or environmental substances known to induce photosensitivity [3].

Phototesting was performed in one patient. The broadband UVB MED was 50 mJ/cm², whereas the NB-UVB MED was 0.3 J/cm²; no abnormal response to UVA was observed [3]. These findings are descriptive and should be interpreted cautiously because phototesting was performed in only one patient.

Regarding disease extent, the T classification was based on disease status at the time of MF diagnosis. ALR was observed in 1/19 patients (5.3%) with T1, 3/14 (21.4%) with T2, and 2/7 (28.6%) with T4 disease [3]. The two patients with T4 disease who developed ALR had been classified as T4 at diagnosis; however, neither had erythroderma at the initiation of NB-UVB therapy or at the time of ALR development. In both patients, ALR was identified by the appearance of erythema on clinically uninvolved skin. The small number of affected patients precludes firm conclusions about this pattern. Age, disease duration, serum soluble interleukin-2 receptor (sIL-2R) levels, and lactate dehydrogenase (LDH) levels did not differ significantly between patients with and without ALR (Mann-Whitney U test; $P = 0.987$, $P = 0.368$, $P = 0.289$, and $P = 0.426$, respectively) [3]. However, these comparisons were based on a small cohort, including only six patients with ALR, and should therefore be interpreted cautiously.

These findings suggest that routine clinical and laboratory variables may not reliably identify patients at risk, underscoring the importance of monitoring for reactions early in treatment.

Importantly, the current understanding of NB-UVB-induced ALR in MF is based largely on a single-center study, and independent validation in other patient populations is lacking.

Possible Pathogenesis of Narrowband Ultraviolet B-Induced Abnormal Light Reactions

UVB-induced erythema is mediated in part by prostaglandin E2 and nitric oxide, which contribute to the vascular response following irradiation [22]. In MF, malignant T cells interact with immune and stromal cells within the tumor microenvironment, influencing local inflammation and malignant cell behavior [23]. Histopathologic studies have also identified mild mononuclear cell infiltration and occasional epidermal infiltration in clinically uninvolved skin of patients with MF [24]. Single-cell RNA sequencing has identified malignant T cells with distinct gene expression profiles in clinically uninvolved skin of patients with MF [25]. However, these findings should be interpreted cautiously because the study involved a relatively small cohort that included patients with advanced-stage MF. Their generalizability to patients with early-stage disease remains uncertain.

These findings raise the possibility that altered UV responsiveness may extend beyond visible MF lesions. NB-UVB irradiation could affect subclinical malignant T-cell populations, reactive immune cells, or other components of the cutaneous microenvironment, resulting in inflammation that becomes clinically apparent as ALR. The numerically higher frequency of ALR in patients with more extensive skin involvement is compatible with this hypothesis [3] but does not establish a causal relationship. The original study did not directly examine malignant T cells in clinically uninvolved skin or inflammatory responses following irradiation, and the number of affected patients was small. Thus, the proposed involvement of tumor-associated immune alterations in ALR remains hypothetical.

More broadly, the available evidence on NB-UVB-induced ALR in MF remains limited and is based largely on a single-center study conducted at the author's institution. Together with the predominance of case reports and small case series in the literature, this limits the generalizability of the current findings. Independent studies in other patient populations are needed to validate these observations and further clarify the underlying mechanisms.

Clinical assessment and management

Clinical Evaluation

During the early stages of NB-UVB therapy for MF, clinicians should monitor for erythema, irritation, and pruritus [19]. When a reaction occurs, the irradiation dose, onset and duration, associated symptoms, distribution, and reproducibility should be assessed. Technical factors, including equipment output, treatment settings, patient positioning, and shielding, should also be reviewed, along with recent sunlight exposure and medications, topical products, supplements, or other substances that may cause or aggravate photosensitivity.

When no apparent technical, medication-related, or environmental cause is identified, ALR should be considered in patients who develop erythema in clinically uninvolved skin after low-dose NB-UVB. The dose threshold used in our study is an operational definition and is not an established diagnostic criterion [3].

Differential Diagnosis

The differential diagnosis includes an expected post-irradiation erythemal response, excessive irradiation, drug-induced photosensitivity, and exacerbation of MF lesions. Reactions also triggered or aggravated by natural sunlight warrant consideration of photosensitive MF. Phototesting with broadband UVB and UVA, and visible light when indicated, may help characterize the response. Persistent photodistributed eczematous or lichenified lesions occurring independently of NB-UVB treatment should prompt consideration of CAD or another photodermatosis. Skin biopsy may be warranted if lesion morphology or distribution changes, while patch testing or photopatch testing may be useful when photoallergic contact dermatitis is suspected.

Management

If ALR is suspected, NB-UVB should be withheld until the erythema resolves. Topical anti-inflammatory treatment may be used according to symptom severity. If phototherapy is resumed, a reduced dose followed by cautious dose escalation is appropriate [19]. For patients who cannot continue whole-body irradiation and have localized MF lesions, targeted phototherapy with a 308-nm excimer light or laser may be considered [2].

In our single-center study, topical corticosteroids were used to manage ALR. In many patients, phototherapy could be continued after NB-UVB dose reduction or a change to, or combination with, 308-nm excimer laser treatment [3]. MF lesions also improved during low-dose NB-UVB therapy in some patients who developed ALR. Thus, ALR does not necessarily require permanent discontinuation of phototherapy; individualized adjustment of the dose or irradiation modality may allow treatment to continue. However, treatment responses have not been quantitatively compared between patients with and without ALR [3]. Documenting baseline skin findings, irradiation parameters, reaction onset and duration, distribution, and associated symptoms can help distinguish ALR from expected erythema, excessive irradiation, and MF exacerbation.

Figure *1* summarizes the clinical assessment and management of suspected ALR during NB-UVB therapy for MF.

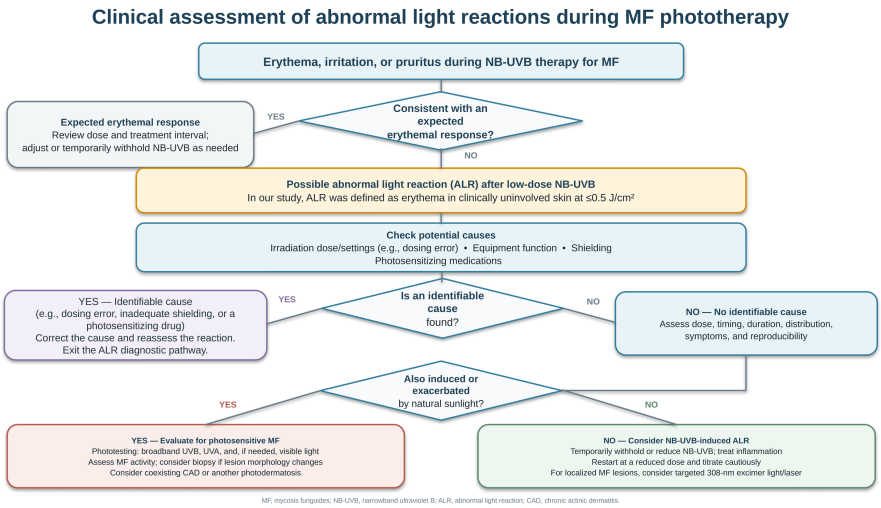


FIGURE 1: Clinical assessment of abnormal light reactions during MF phototherapy

The flowchart outlines the evaluation of erythema, irritation, or pruritus during NB-UVB therapy, including differentiation between an expected erythemal response, photosensitive MF, and NB-UVB-induced ALRs. The assessment includes checking for identifiable technical or treatment-related causes, such as dosing errors, equipment problems, inadequate shielding, and photosensitizing medications. When an identifiable cause is found, it should be corrected, and the reaction reassessed before further diagnostic evaluation. If the reaction persists despite correction of the identified cause, alternative explanations, including ALR, should be reconsidered. The assessment also considers whether reactions are induced or exacerbated by natural sunlight, which may warrant evaluation for photosensitive MF. In our study, ALR was defined as erythema in clinically uninvolved skin following NB-UVB irradiation at a dose of ≤0.5 J/cm². This study-specific definition is not an established diagnostic criterion.

ALR: abnormal light reaction, CAD: chronic actinic dermatitis, MF: mycosis fungoides, NB-UVB: narrowband ultraviolet B, UVA: ultraviolet A, UVB: ultraviolet B

Future directions

Future research should establish standardized criteria for defining and assessing ALR. In addition to the irradiation dose, these criteria should consider the patient's minimal erythema dose (MED), the intensity and duration of erythema, associated irritation or pruritus, and reproducibility across exposures. Standardized phototesting with UVA, broadband UVB, NB-UVB, and, when indicated, visible light could help characterize the action spectrum and clarify the relationship between photosensitive MF and NB-UVB-induced ALR.

Multicenter prospective studies are needed to determine the incidence and clinical correlates of ALR. Comparisons between patients with and without ALR should assess treatment response, the number of irradiations required for remission, cumulative UVB dose, and recurrence. Mechanistic studies of lesional and clinically uninvolved skin before and after irradiation could investigate DNA damage responses, inflammatory mediators, malignant T-cell distribution and clonality, reactive immune cell populations, and gene expression changes. These studies may help establish the biological basis of ALR and guide safer, more individualized phototherapy for MF.

Conclusions

Photosensitivity in MF includes sunlight-induced or photodistributed lesions, CAD-like eruptions, and abnormal reactions during NB-UVB therapy. These presentations require careful differentiation based on clinical findings, phototesting, histopathology, and disease course.

NB-UVB-induced ALR is not yet clearly defined and may not represent the same process as photosensitive MF. Recognition of atypical reactions and appropriate adjustment of phototherapy may allow treatment to continue. The available evidence remains limited by the small number of published reports and generally small sample sizes. Further studies are needed to establish diagnostic criteria and clarify the clinical significance and underlying mechanisms of ALR.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the

work.

Concept and design: Tadamichi Shimizu

Acquisition, analysis, or interpretation of data: Tadamichi Shimizu

Drafting of the manuscript: Tadamichi Shimizu

Critical review of the manuscript for important intellectual content: Tadamichi Shimizu

Supervision: Tadamichi Shimizu

Disclosures

Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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