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Narrowband ultraviolet B phototherapy for the treatment of steroid-refractory and steroid-dependent acute graft-versus-host disease of the skin

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Abstract

Background—Acute graft-versus-host disease (aGvHD) is a common complication of allogeneic stem cell transplantation. It is usually treated with high doses of corticosteroids and other immunosuppressive agents. When cutaneous features are predominant, narrowband ultraviolet B (NB-UVB) phototherapy may be an attractive option for its steroid-sparing effect.

Objective—We sought to examine the clinical efficacy of NB-UVB in the treatment of steroid-refractory and steroid-dependent cutaneous aGvHD.

Methods—We conducted a retrospective chart review of patients with steroid-refractory and steroid-dependent aGvHD, who received NB-UVB between 2005 and 2009 at our institution.

Results—We identified 14 patients with aGvHD treated with NB-UVB between 2005 and 2009. The median number of treatments was 15, administered over a median of 43 days. Eight of 14 patients (57%) achieved a complete response at the end of treatment; an additional 3 patients (21%) achieved a partial response; and 3 patients (21%) showed no improvement at the time when phototherapy was discontinued (nonre-sponders). Four patients developed chronic graft-versus-host disease (GvHD). Three of the 8 complete responders remained free of GvHD at 6 months' follow-up.

Limitations—The rarity of steroid-refractory aGvHD limited the study to a small number of participants. Because GvHD is variable in its presentation and course, and life-threatening in many cases, large controlled prospective trials for potential therapies are difficult.

Conclusions—NB-UVB is a viable option for the treatment of steroid-refractory and steroid-dependent aGvHD of the skin.

Keywords

graft-versus-host disease; narrowband ultraviolet B; phototherapy; skin; steroid-refractory; ultraviolet light

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Conflicts of interest: None declared.

Acute graft-versus-host disease (aGvHD) is a major complication in more than 50% of patients receiving allogeneic stem cell transplantation.[1] Graft-versus-host reactions occur when donor immunocompetent cells attack the tissue of the recipient. Graft-versus-host disease (GvHD) is classified as acute or chronic, with the development of aGvHD being a risk factor for the development of chronic GvHD. The skin is often the first organ affected by aGvHD, which initially manifests as a papular eruption but can progress to desquamation resembling toxic epidermal necrolysis. Typically, aGvHD develops between 2 and 6 weeks after transplantation. Chronic GvHD is characterized by lichenoid or sclerodermatous lesions of the skin. In many patients, GvHD is associated with significant morbidity and mortality.[2]

The currently accepted model for the pathophysiology of cutaneous aGvHD is described in 3 phases.[3] During the first phase, the conditioning regimen that the recipient patient undergoes before stem cell transplantation causes keratinocyte injury, resulting in a cytokine response that activates antigen-presenting epidermal dendritic (Langerhans) cells. In the second phase, antigen presentation results in activation of donor T cells and the production of cytokines within the Th1 pathway. The final step is host keratinocyte necrosis and apoptosis mediated by donor cytotoxic T cells. The established strategy for treatment of GvHD involves rapid control with high-dose systemic corticosteroids and long-term use of steroid-sparing immunosuppressive agents such as cyclosporine or tacrolimus.[4] If aGvHD does not respond to high-dose corticosteroids, it is designated “steroid-refractory” aGvHD. If aGvHD clears with high-dose systemic corticosteroids, but recrudesces on taper of steroids, it is designated “steroid-dependent” aGvHD. Unfortunately, in addition to toxicities associated with systemic steroid-sparing agents (eg, nephrotoxicity and neuropathy), the immunosuppression resulting from therapy (in addition to the immune dysregulation in the posttransplantation patient with GvHD) increases the risk of opportunistic infections, recurrent malignancy, secondary malignancies, and ultimately, death.[2,3,5]

When skin involvement is the predominant feature of aGvHD, there is an opportunity to administer skin-directed therapy with ultraviolet (UV) light. The immunomodulating effects of UV irradiation may allow for the reduction or replacement of standard systemic immunosuppressive therapy. After the Food and Drug Administration approval of psoralen plus UVA (PUVA) in 1982 for psoriasis and vitiligo, PUVA has been used to treat a variety of skin diseases, including GvHD.[6] More recent studies relate the efficacy of PUVA in treating GvHD,[7–9] and PUVA has become the standard of care with respect to skin-directed therapy for GvHD despite the theoretical risk of skin cancer and potential hepato-toxicity as a result of oral psoralen use.[10–12] Claims of greater long-term safety of narrowband UVB (NB-UVB) in comparison with PUVA has prompted the use of NB-UVB in treating cutaneous GvHD.[13] It is purported that NB-UVB therapy has a smaller risk of inducing skin cancer when compared with PUVA, even in patients requiring phototherapy for many years,[14] although this has not been substantiated in long-term studies, or in patients with allogeneic stem cell transplantation.

NB-UVB’s efficacy in treating inflammatory disorders of the skin is likely derived from its antiproliferative and immunosuppressive effects. NB-UVB therapy suppresses the type 1 pathway (interleukin-12, interferon-γ, and interleukin-8), leads to apoptosis of skin-homing lymphocytes, increases the number of p53-positive epidermal cells, and reduces the number of Langerhans cells present in the epidermis and dermis.[15,16] These actions are likely critical to its effectiveness in treating cutaneous aGvHD, although studies on NB-UVB’s mechanism of action in this relatively rare disease are lacking to date.

METHODS

This report describes our center's experience with NB-UVB phototherapy in the treatment of steroid-refractory and steroid-dependent aGvHD of the skin between 2005 and 2009. This retrospective study was conducted under the approval of the Johns Hopkins Hospital Institutional Review Board.

Data collection

We retrospectively reviewed the records of patients with steroid-refractory and steroid-dependent aGvHD, who were treated with NB-UVB at the our hospital between January 2005 and August 2009. Patients were eligible for skin-directed therapy when they had skin-only disease or the extracutaneous aGvHD was either under good control or stable. Patients with chronic GvHD were excluded from this study. We collected data on patient demographics, malignancy diagnosis, donor source, conditioning regimen, GvHD prophylactic regimen, skin stage, GvHD grade, and aGvHD therapy given before administration of NB-UVB.

Definition of aGvHD, skin staging, and grading system

aGvHD was diagnosed according to typical clinical manifestations and supported by skin biopsy. Clinical staging for aGvHD, as defined by the Keystone criteria, takes into consideration the body surface area involved with rash, and the presence of bullae. Stage 1 involves less than 25% of skin surface area; stage 2 involves 25% to 50%; stage 3 involves more than 50%; and stage 4 denotes bullae formation.[17] The aGvHD grade is determined by comparing the stage of disease in each individual organ system: grade I involves skin only (stages 1 and 2); grade II involves the liver or gut, or has stage 3 skin involvement; grade III has stage 2 or 3 skin involvement, or stage 2 to 4 gut involvement; and grade IV has stage 4 skin involvement or stage 4 liver disease.

We defined steroid-refractory disease as advancement of aGvHD by at least one stage or systemic GvHD by one grade despite 48 hours of treatment with prednisone (2–2.5 mg/kg/d). Patients who showed no improvement with 4 days of prednisone treatment were also considered to have steroid-refractory disease. We defined steroid-dependent disease as the recrudescence of aGvHD of the skin upon taper of prednisone (with a similar response to a second taper of prednisone), despite adequate control of disease while on high-dose prednisone.

NB-UVB phototherapy regimen

NB-UVB phototherapy was administered in a full-body upright UV therapy cabinet (Daavlin 3 Series 311/350, Bryan, OH) fitted with 24 fluorescent bulbs emitting light with a 311-nm wavelength (Phillips 24 TL100W/01-FS72, Amsterdam, The Netherlands). The minimal erythema dose for each patient was determined by exposing different areas of a patient's upper back to a range of NB-UVB doses and observing the lowest dose producing pink erythema 24 hours later. The initial NB-UVB dose was set to 70% of the minimal erythema dose and increased by 10% every treatment. Patients with Fitzpatrick skin types I and II received treatment twice a week, whereas those with skin types III and VI received treatment 3 times a week. Eight patients were of skin type I, 5 were of skin type II, and one had type III skin. If erythema was observed, treatment was withheld until erythema had resolved and dose was reduced by 20% on resuming treatment. NB-UVB was discontinued if a patient was clinically clear of rash and/or systemic steroid was completely tapered and rash had cleared or reached a plateau, or at the discretion of the treatment team (worsening rash, hospitalization, development of significant aGvHD in other organ systems, or development of chronic GvHD, in some cases).

Measurement of response

The primary outcome was reduction in clinical staging of cutaneous aGvHD at the end of NB-UVB phototherapy. Body surface area and clinical stage assessments were recorded at onset of phototherapy and on discontinuation of phototherapy. Complete response was achieved when there was no evidence of cutaneous GvHD on discontinuation of therapy. Partial response was defined as reduction in clinical staging (by at least one stage) when phototherapy was discontinued. Nonresponse was defined as no reduction or worsening of clinical stage at the time that phototherapy was discontinued. Secondary outcome measures included mean change in cutaneous GvHD score, mean change in total body surface area, and mean change in daily steroid dose at the end of the treatment course of NB-UVB. Six-month and longer follow-up (presence of relapse of malignancy, the presence of aGvHD or chronic GvHD, and death) were included, when available. Adverse events were also reported. Differences between cutaneous GvHD scores, total body surface areas involved, and steroid doses before NB-UVB therapy and on discontinuation of therapy were summarized and compared using paired *t* test.

RESULTS

Patient characteristics

A total of 14 patients (10 male, 4 female; mean age 40 years) were treated for steroid-refractory and steroid-dependent aGvHD of the skin (3 refractory, 11 dependent) after allogeneic bone marrow, peripheral blood stem cell, or cord blood transplantation. Seven of the 14 patients showed additional organ manifestations (liver, gut, mouth, eyes). Conditioning and prophylactic regimens for each patient are shown in Table I.

On diagnosis of aGvHD of the skin, prednisone (2.0–2.5 mg/kg/d) was started for most patients. If not already part of the prophylactic regimen, tacrolimus was included for most patients if there was systemic involvement (ie, gut or liver). Secondary therapy other than NB-UVB included mycophenolate mofetil, sirolimus, extracorporeal photopheresis, and PUVA. One patient (7%) started therapy with stage 1 cutaneous GvHD, 7 patients (50%) began with stage 2 cutaneous GvHD, and 6 patients (43%) began with stage 3 cutaneous GvHD. No patients had stage 4 cutaneous GvHD. In all, 29% of patients had an overall GvHD grade of I and 71% were grade II. No patients had grade III or IV systemic GvHD.

Response rates: NB-UVB phototherapy doses, skin response, and steroid reduction

Mean initial dose of NB-UVB was 0.284 J/cm^2 (range: 0.100–0.840 J/cm^2). The mean cumulative NB-UVB dose was 8.607 J/cm^2 (range: 1.280–40.946 J/cm^2). The median number of treatments administered was 15 (range: 5–56). The median length of a therapy course was 43 days (range: 18–220 days) (Table II).

Eight of 14 patients (57%) showed no signs of cutaneous GvHD when therapy was discontinued (complete responders). Three patients (21%) achieved a partial response. Three patients (21%) showed no improvement at the time when phototherapy was discontinued (Table III).

Ten of the 13 patients taking steroids at the onset of therapy were able to reduce their steroid dose over the course of their therapy (Table IV). Two of the patients who showed a decrease in staging were not able to reduce their steroid dosage over the course of their phototherapy.

Four of the 6 patients who did not achieve complete responses discontinued therapy prematurely because of hospitalization for life-threatening infections (3 partial response and one nonresponse: patient 3, methicillin-resistant *Staphylococcus aureus* abscess; patient 5,

Pneumocystis pneumonia; patient 12, gram-negative sepsis; patient 14, fungal pneumonia). The other two patients were switched to PUVA regimens immediately after the physician deemed the patients to be nonresponders to NB-UVB (patients 4 and 13).

Of the 7 complete responders available for follow-up at 6 months after discontinuing NB-UVB, 3 patients (43%) had developed chronic GvHD. One complete responder had a recurrence of aGvHD that was then treated with PUVA.

Adverse events

All patients tolerated NB-UVB. One patient had a phototoxic reaction, requiring a 10-day discontinuation of NB-UVB. On resumption of NB-UVB, the patient was a complete responder. No patients completely discontinued therapy because of side effects from phototherapy itself. There was no incidence of skin cancer during the course of NB-UVB therapy in any patient. One patient was given the diagnosis of basal cell carcinoma 15 months after finishing therapy with a complete response, although he had a history of multiple skin cancers before undergoing phototherapy.

DISCUSSION

This retrospective review lends support that NB-UVB phototherapy may be an attractive steroid-sparing option for steroid-refractory and steroid-dependent aGvHD of the skin. Often, attempts to decrease immunosuppression in a patient are hindered by reoccurrences of cutaneous GvHD. If these flareups can be managed with phototherapy, reduction in immunosuppression may decrease the susceptibility to infection. It is still unclear whether control of aGvHD decreases the risk of chronic GvHD.

Because GvHD is rare, variable in its presentation and course, and life-threatening in many cases, large controlled trials for potential therapies are difficult. Almost a third of this study's population was forced to discontinue treatment after developing high-risk infections leading to hospitalization, often in the critical care setting. Because there is no established protocol for the treatment of steroid-refractory and steroid-dependent aGvHD, there will always be a physician bias with respect to the specific algorithm used in treating a population of patients.

At the time of submission, we believe this is the largest series of patients treated with NB-UVB for steroid-refractory and steroid-dependent aGvHD. Based on these findings of complete responses in 8 of 14 patients and an overall response rate of 78%, we believe that NB-UVB is a viable option for the treatment of steroid-refractory and steroid-dependent aGvHD of the skin. These results align with the data from the Grundmann-Kollmann et al[13] study in which 7 of 10 patients showed complete clearance of steroid-refractory GvHD after NB-UVB therapy. In our study, two of the 3 patients with steroid-refractory disease showed a reduction in clinical staging in addition to the 9 patients with steroid-dependant disease who also showed a reduction. In the largest study on PUVA therapy for steroid-dependent aGvHD of the skin, Furlong et al[8] described complete response in 24 of 65 patients. Remarkably, 92% of the patients treated with PUVA in that study developed chronic GvHD.

We chose to evaluate the efficacy of NB-UVB in aGvHD because we believe it is an attractive alternative to PUVA. NB-UVB does not require administration of an oral photosensitizer. Psoralen causes nausea in many patients, ultimately decreasing compliance with photochemotherapy. There is also a small risk of hepatotoxicity with psoralen, and in patients with GvHD, elevated liver enzymes can be a result of multiple causes, including GvHD involvement of the liver, viral diseases, and the hepatotoxicity of other medications, making interpretation of abnormal laboratory study results difficult. Furthermore, if NB-

UVB treatment is shown in long-term studies to have a lower carcinogenic risk than PUVA at irradiation levels used for GvHD therapy, it may become the preferred method of skin-directed therapy for aGvHD.

The findings of this study make it clear that we need prospective randomized trials comparing PUVA and NB-UVB phototherapy to evaluate for equivalence or superiority of NB-UVB over PUVA. Nonetheless, our findings demonstrate that NB-UVB has activity in aGvHD. Long-term follow-up is needed to address concerns about GvHD reoccurrence, progression to chronic GvHD, and carcinogenicity.

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Abbreviations used

aGvHD	acute graft-versus-host disease
GvHD	graft-versus-host disease
NB-UVB	narrowband ultraviolet B
PUVA	psoralen plus ultraviolet A
UV	ultraviolet

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CAPSULE SUMMARY

- We retrospectively reviewed our institution's experience in treating 14 patients with steroid-refractory and steroid-dependent acute graft-versus-host disease using narrowband ultraviolet (UV) B.
- Eleven of 14 patients had a reduction in clinical staging of acute graft-versus-host disease with narrowband UVB therapy.
- No patients experienced side effects causing them to discontinue phototherapy.
- Narrowband UVB may be an attractive alternative to psoralen plus UVA in the treatment of cutaneous acute graft-versus-host disease, especially in those patients intolerant of oral psoralen.

Table I

Patient characteristics

Patient No.	Age, y	Sex	Diagnosis	Donor/source	Conditioning regimen	GvHD prophylaxis	Skin stage	GvHD grade	Other organs	GvHD therapy
1	18	F	GDL	MRD/BM	Cy, TBI	CsA, MTX	1	I		Pred + rapamycin
2	18	M	HL	H/BM	Fludarabine, Cy, TBI	Cy, FK506, MMF	2	I		Pred
3	63	F	CML	MRD/BM	Bu, Cy	Cy	2	II	Liver	Pred
4	50	M	AML	H/BM	Fludarabine, Cy, TBI	Cy, FK506, MMF	2	I		Pred
5	1	M	AML	H/BM	Bu, Cy	CsA, MTX	2	II	Liver + eyes	Pred
6	16	F	HL	MUD/PBSC	Bu, Cy	CsA, MTX	2	II	Gut + eyes	Pred
7	52	F	AML	MRD/BM	Bu, Cy	Cy	2	I	Eyes	Pred
8	14	M	AML	H/BM	Bu, Cy	CsA, MTX	2	II	Liver	Pred
9	65	M	AML	MUD/BM	Bu, Cy	Cy	3	II		None
10	69	M	DLCL	H/BM	Fludarabine, Cy	Cy, FK506, MMF	3	II		Pred
11	51	M	CLL	MRD/BM	Bu, Cy	Cy	3	II		Pred + FK506
12	64	M	AIL	H/BM	Fludarabine, Cy, TBI	Cy, FK506, MMF	3	II	Oral	Pred
13	49	M	CML	MUD/BM	Bu, Cy	Cy	3	II		Pred + FK506
14	29	M	AML	Cord blood	Fludarabine, Cy, TBI	CsA, MMF	3	II	Gut	Pred + FK506

AIL, Angioimmunoblastic T-cell lymphoma; *AML*, acute myeloid leukemia; *BM*, bone marrow; *Bu*, busulfan; *CLL*, chronic lymphoid leukemia; *CML*, chronic myeloid leukemia; *CsA*, cyclosporine; *Cy*, cyclophosphamide; *DLCL*, diffuse large B-cell lymphoma; *F*, female; *FK506*, tacrolimus; *GDL*, gamma delta T-cell lymphoma/leukemia; *GvHD*, graft-versus-host disease; *H*, haploidentical donor; *HL*, Hodgkin lymphoma; *M*, male; *MMF*, mycophenolate mofetil; *MRD*, matched related donor; *MTX*, methotrexate; *MUD*, matched unrelated donor; *PBSC*, peripheral blood stem cell; *Pred*, prednisone; *TBI*, total body irradiation.

Table II

NB-UVB phototherapy details and outcomes

Patient No.	Skin type	Course length, d	NB-UVB treatments	Start dose, J/cm^2	Maximum dose, J/cm^2	Cumulative dose, J/cm^2	Initial steroid, mg/kg/d	Final steroid, mg/kg/d	BSA before, % (stage)	BSA after, % (stage)	Response	6-mo Follow-up	Last known status
1	I	220	56	0.210	0.540	26.001	1.00	0.00	18 (1)	0 (0)	CR	Remission	Alive
2	II	18	5	0.210	0.306	1.280	2.00	1.00	27 (2)	0 (0)	CR	Chronic GvHD (skin)	Alive
3	II	19	7	0.560	0.642	4.716	0.69	0.69	27 (2)	18 (1)	PR	Died (liver failure)	-
4	III	21	9	0.140	0.297	2.835	0.78	0.56	36 (2)	63 (3)	NR	aGvHD (skin)	Alive
5	I	23	9	0.100	0.221	1.394	0.75	0.75	36 (2)	9 (1)	PR	Died (*Pneumocystis* pneumonia)	-
6	I	77	17	0.200	0.826	13.204	1.50	0.50	36 (2)	0 (0)	CR	Remission	Died (relapsed leukemia)
7	I	31	11	0.840	0.840	4.847	0.47	0.08	45 (2)	0 (0)	CR	Chronic GvHD (skin)	Died (liver failure not caused by GvHD)
8	II	112	27	0.100	0.339	7.047	2.00	0.00	45 (2)	0 (0)	CR	Chronic GvHD (lung, liver)	Died (bronchiolitis obliterans)
9	II	201	40	0.280	0.804	28.229	0	0	54 (3)	0 (0)	CR	Lost to follow-up	Died (relapsed leukemia)
10	I	90	19	0.200	0.565	5.814	0.91	0.00	54 (3)	0 (0)	CR	aGvHD (skin)	Alive (basal cell carcinoma)
11	I	38	13	0.280	0.541	7.992	0.71	0.09	63 (3)	0 (0)	CR	Remission	Alive
12	I	30	9	0.350	0.679	4.672	1.04	0.63	63 (3)	18 (1)	PR	Died (septic shock)	-
13	I	48	18	0.210	0.590	5.519	1.88	0.50	72 (3)	54 (3)	NR	Chronic GvHD (skin)	Alive (chronic GvHD)
14	II	60	17	0.300	0.530	6.941	2.00	0.00	100 (3)	72 (3)	NR	Remission	Died (progressive leukemia)

aGvHD, Acute graft-versus-host disease; *BSA*, body surface area; *CR*, complete response; *GvHD*, graft-versus-host disease; *NB-UVB*, narrowband ultraviolet B; *NR*, nonresponse; *PR*, partial response.

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Table III

Response rates

	Percent	SE
Complete response	57	0.13
Partial response	21	0.11
No response	21	0.11
Overall response rate	78	0.11

Table IV

Changes in outcome measures

	Change, mean (SD)	*P**
Cutaneous GvHD stage	−1.5 (1.2)	.000514
Body surface area (%)	−31.6 (23.1)	.000196
Prednisone dose, mg/kg/d	−0.781 (0.676)	.000823

GvHD, Graft-versus-host disease.

* Based on paired *t* test comparing values at onset and discontinuation of therapy.