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# High-Dose Oral Vitamin D for Toxic Effects of the Skin Associated With Chemotherapy and Radiation

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**IMPORTANCE** Severe cutaneous toxic effects from chemotherapy and radiation often require treatment interruptions. High-dose oral vitamin D (hdVD) has shown potential as a rapid immunomodulator in experimental models and small human studies, but robust clinical data in oncology populations remain limited.

**OBJECTIVE** To evaluate the clinical response, safety profile, and time to improvement for hdVD in patients with toxic erythema of chemotherapy (TEC) and acute radiation dermatitis (ARD).

**DESIGN, SETTING, AND PARTICIPANTS** This retrospective multicenter case series included 33 patients treated across 3 academic medical centers between December 2021 and January 2024. Eligible participants were those receiving hdVD (100 000 international units) for TEC or ARD with at least 10 days of follow-up.

**EXPOSURES** One or 2 oral doses of 100 000 international units of cholecalciferol or ergocalciferol.

**MAIN OUTCOMES AND MEASURES** The primary outcomes were time to patient-reported symptomatic relief and clinician-assessed objective improvement in erythema (using a 5-point Likert scale). Secondary outcomes included changes in serum calcium and the ability to continue anticancer therapy.

**RESULTS** Among 33 patients (mean [SD] age, 60.9 [14.6] years; 19 [58%] female), 28 (85%) had TEC and 5 (15%) had ARD. Subjective symptom relief was reported by 26 of 30 patients (87%) within 10 days of treatment. The median (range) time to improvement was 5 (1-28) days overall and 3 (1-16) days for the inpatient subgroup. The mean (SD) Likert erythema score decreased from 4.36 (0.60) at baseline to 2.21 (1.36) by day 10. Patients with neutrophilic eccrine hidradenitis and Stevens-Johnson syndrome/toxic epidermal necrolysis–like subtypes showed the most rapid responses. No meaningful changes in serum calcium levels were observed, and 24 of the 33 patients (73%) continued anticancer therapy without interruption. No treatment-related adverse events were reported during the study.

**CONCLUSIONS AND RELEVANCE** In this case series, hdVD was associated with rapid symptomatic and objective improvement in chemotherapy- and radiation-induced toxic effects of the skin without treatment-related adverse events. These findings support further study of hdVD as a supportive care approach in oncodermatology.

Supplemental content

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Severe cutaneous toxic effects from chemotherapy and radiation pose substantial challenges due to limited treatment options and the need to continue oncologic therapy. Vitamin D, a fat-soluble hormone involved in calcium homeostasis and bone metabolism, has shown therapeutic potential for these toxic effects.[1-4] Vitamin D is primarily produced endogenously through UV-induced synthesis in skin, with dietary intake contributing less.[5-7]

High-dose oral vitamin D (hdVD) has been investigated as a rapid modulator of acute skin inflammation.[1,2] In a randomized, placebo-controlled study of UV-induced dermatitis, a single oral cholecalciferol dose (50 000-200 000 international units [IU]) within 1 hour of exposure reduced erythema and edema at 48 hours, accompanied by decreased vesiculation and dermal edema on lesional biopsy.[2] Participants receiving 200 000 IU showed considerably lower cutaneous tumor necrosis factor α and inducible nitric oxide synthase transcripts compared with placebo, supporting molecular attenuation of inflammation.[2]

In a randomized, double-blind, placebo-controlled human model of topical mechlorethamine (nitrogen mustard)-induced dermatitis, healthy adults underwent untreated exposure, then were rechallenged on the contralateral arm and randomized to a single dose of 200 000 IU of oral cholecalciferol or placebo.[1] The hdVD attenuated acute inflammation by prespecified clinical and histologic end points, showing less erythema, reduced skin thickness, and improved blinded dermatopathology scores.[1] Effects persisted up to 6 weeks after 1 dose, and multi-omic profiling demonstrated suppression of CCL20, CCL2, and CXCL9.[1] These are upregulated during acute inflammation, supporting hdVD-mediated attenuation of inflammatory and interleukin 17 signaling.[1,4]

A combined murine UV-injury model and paired human biopsy analysis subsequently demonstrated that vitamin D protected against UV-induced cutaneous injury by enhancing autophagy in M2-polarized macrophages and upregulating M2-associated genes (eg, *ARG1*).[8] In human sunburn biopsies, 200 000 IU of oral vitamin D3 increased LC3 signal within $CD163^+$ macrophages relative to placebo, consistent with macrophage-specific autophagy and accelerated resolution of UV-mediated epidermal injury.[8] Collectively, these data support hdVD as a strategy to dampen early cutaneous inflammation in injury paradigms relevant to toxic erythema of chemotherapy (TEC) and acute radiation dermatitis (ARD).[3,4] Although mechanistically distinct, TEC and ARD share similar paradigms of cytotoxic cutaneous injury, as evidenced by overlapping histologic findings (vacuolar interface changes, epidermal necrosis, necrotic keratinocytes, and subepidermal blistering).

Small case reports and series have highlighted rapid symptom improvement after hdVD in TEC and ARD.[3,4,9,10] In a 2023 inpatient series, 6 patients received 50 000 to 100 000 IU of vitamin D on days 0 and 7; all experienced improved pain, pruritus, and swelling by day 1 and visible erythema reduction within 1 to 4 days.[3] In a small follow-up ARD series, 2 doses of 100 000 IU of oral ergocalciferol given 1 week apart produced symptomatic relief within 3 to 7 days.[4] Erythema improved more modestly, likely reflecting deeper dermal injury from radiation dermatitis.[4]

### Key Points

**Question** Is high-dose oral vitamin D associated with rapid clinical improvement and safety in patients with chemotherapy- or radiation-related toxic effects of the skin?

**Findings** In this case series of 33 patients with toxic erythema of chemotherapy or acute radiation dermatitis, 26 patients reported symptomatic relief within 10 days of high-dose oral vitamin D administration (100 000 international units). Objective erythema scores considerably improved, with a median time to improvement of 5 days, and no cases of hypercalcemia or treatment-related adverse events.

**Meaning** Patients who received high-dose oral vitamin D therapy were subsequently noted to have rapid improvement in radiation- and chemotherapy-related toxic effects of the skin without any substantial safety concerns.

In this case series, we sought to build on these preliminary observations via a larger cohort of patients with TEC and ARD who were then treated with hdVD. We characterized clinical course, safety, time to response, quantitative erythema change, and continuation of anticancer therapy after hdVD. We hypothesized that 1 to 2 hdVD doses (100 000 IU) would produce clinically meaningful, rapid improvement in chemotherapy- and radiation-associated cutaneous toxic effects, thereby facilitating continuation of anticancer therapy without adverse effects.

## Methods

We conducted a retrospective study of patients with TEC or ARD who were treated with hdVD by oncodermatologists across 3 academic institutions between December 2021 and January 2024. The study was deemed exempt from review by the institutional review boards of Mass General Brigham and Beth Israel Deaconess Medical Center due to the retrospective nature of the study. Need for patient informed consent was also waived owing to use of deidentified data. Case patients were identified through the Mass General Brigham Research Patient Data Registry and dermatology prescription records at Beth Israel Deaconess Medical Center and included if 10-day follow-up data after the initial hdVD dose were available.

Demographic, oncologic, treatment, and safety laboratory data were extracted from medical records. Symptomatic improvement and objective erythema improvement were assessed over the 10-day follow-up period. Objective erythema was graded retrospectively using a standardized 5-point Likert scale (eFigure in Supplement 1) applied to clinical photographs or examination descriptions when photographs were unavailable. Additional details regarding outcome definitions, laboratory monitoring, and statistical analyses are provided in the eMethods in Supplement 1.

## Results

Thirty-three patients (mean [SD] age, 60.9 [14.6] years; 19 [58%] female) received hdVD (100 000 IU in 1 or 2 doses) for chemo-

Table. Patient Demographics, Clinical Characteristics, Culprit Chemotherapies, and Treatment Response

| Characteristic | No. (%) | |
|---|---|---|
| | Total (N = 33) | Inpatient (n = 12) |
| Age, mean (range), y | 60.85 (27-88) | 62.75 (43-82) |
| Sex | | |
| Female | 19 (58) | 5 (42) |
| Male | 14 (42) | 7 (58) |
| Self-reported race and ethnicity | | |
| Asian | 3 (9) | 1 (8) |
| Black | 1 (3) | 1 (8) |
| Hispanic | 2 (6) | 1 (8) |
| Persian | 1 (3) | 1 (8) |
| Portuguese | 2 (6) | 2 (17) |
| White | 26 (79) | 7 (58) |
| Clinical setting | | |
| Inpatient | 12 (36) | 12 (100) |
| Outpatient | 21 (64) | NA |
| Cancer type | | |
| Solid organ malignant neoplasm | 25 (76) | 7 (58) |
| Breast cancer | 6 (18) | 2 (17) |
| Bladder cancer | 4 (12) | 3 (25) |
| Lung cancer | 4 (12) | 1 (8) |
| Desmoid tumor | 3 (9) | 0 |
| Brain cancer | 2 (6) | 0 |
| Gynecologic cancer (including ovarian and cervical carcinoma) | 2 (6) | 1 (8) |
| Gastrointestinal cancer (including esophageal adenocarcinoma and anal squamous cell carcinoma) | 2 (6) | 0 |
| Skin cancer (including squamous cell carcinoma and spindle cell carcinoma) | 2 (3) | 0 |
| Hematologic cancer | 8 (24) | 5 (42) |
| Acute myeloid leukemia | 4 (12) | 4 (33) |
| Central nervous system lymphoma | 1 (3) | 1 (8) |
| Cutaneous T-cell lymphoma | 1 (3) | 0 |
| Hodgkin lymphoma | 1 (3) | 0 |
| Multiple myeloma | 1 (3) | 0 |
| Symptoms reported | | |
| Pain | 15 (45) | 9 (75) |
| Pruritus | 27 (82) | 7 (58) |
| TEC by subtype[a] | 28 (85) | 12 (100) |
| Hand-foot syndrome | 11 (33) | 2 (17) |
| Flexural/intertriginous | 8 (24) | 3 (25) |
| SJS/TEN-like | 4 (12) | 3 (25) |
| Neutrophilic eccrine hidradenitis | 2 (6) | 2 (17) |
| Not classified | 3 (9) | 2 (17) |

(continued)

therapy- or radiation-associated cutaneous eruptions (**Table**). Twelve patients (36%) were treated in the inpatient setting.

TEC accounted for 28 cases (85%), with subtypes including hand-foot syndrome in 11 (33%), flexural and intertriginous eruptions in 8 (24%), Stevens-Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN)-like eruptions in 4 (12%), and neutrophilic eccrine hidradenitis in 2 (6%). TEC subtype responses are summarized in eTable 1 in Supplement 1. Five patients (15%) had ARD. Liposomal doxorubicin (present in 9 patients [27%]) was most frequently implicated. Prior supportive dermatologic therapy was documented in 22 patients (67%); 30 patients (91%) received concurrent supportive therapy, most commonly topical corticosteroids in 28 (85%).

Twenty-eight patients (85%) received a second dose of hdVD within 7 days, while 5 patients (15%) received only 1 dose due to resolution before day 7. During the 10-day follow-up interval, 26 of 30 patients (87%) reported subjective symptom relief, while 23 (77%) had clinician-confirmed clinical improvement. Overall, median (range) time to improvement was 5 (1-28) days and 3 (1-16) days among inpatients. The mean (SD) Likert score de-

Table. Patient Demographics, Clinical Characteristics, Culprit Chemotherapies, and Treatment Response (continued)

| Characteristic | No. (%) | |
|---|---|---|
| | Total (N = 33) | Inpatient (n = 12) |
| ARD | 5 (15) | 0 |
| Culprit chemotherapies[b] | | |
| Liposomal doxorubicin | 9 (27) | 1 (8) |
| Enfortumab vedotin | 4 (12) | 3 (25) |
| Cytarabine | 3 (9) | 3 (25) |
| Carboplatin | 3 (9) | 0 |
| Paclitaxel | 3 (9) | 1 (8) |
| Capecitabine | 2 (6) | 1 (8) |
| Pemetrexed | 2 (6) | 0 |
| 5-Fluorouracil | 2 (6) | 1 (8) |
| Fludarabine | 1 (3) | 1 (8) |
| Busulfan | 1 (3) | 1 (8) |
| Elranatamab | 1 (3) | 0 |
| Thiotepa | 1 (3) | 1 (8) |
| Supportive dermatologic therapies for TEC/ARD[c] | | |
| Prior therapy before hdVD | 22 (67) | 5 (42) |
| Concurrent therapy with hdVD | 30 (91) | 10 (83) |
| Concurrent topical corticosteroids | 28 (85) | 10 (83) |
| Concurrent emollients/barrier agents | 8 (24) | 0 |
| Concurrent systemic corticosteroids | 5 (15) | 3 (25) |
| Treatment response | | |
| Patient-reported subjective improvement, mean (range), d | 6.27 (1-28) | 3.41 (1-16) |
| Clinician-reported subjective improvement, mean (range), d | 8.17 (1-37) | 3.67 (1-16) |
| Score on Likert scale, mean (SD) | | |
| Day 1 | 4.36 (0.60) | 4.42 (0.51) |
| Day 5 | 3.17 (1.03) | 3.00 (0.89) |
| Day 10 | 2.21 (1.36) | 1.82 (1.08) |
| Δ of Likert scores | 1.92 (1.32) | 2.42 (1.24) |
| Continuation of chemotherapy/radiation | 24 (73) | 8 (67) |

Abbreviations: ARD, acute radiation dermatitis; hdVD, high-dose oral vitamin D; NA, not applicable; SJS/TEN, Stevens-Johnson syndrome/toxic epidermal necrolysis; TEC, toxic erythema of chemotherapy.

[a] Some overlap exists between the TEC subtypes, so percentages may not sum to 100%.

[b] In cases where the culprit chemotherapy was busulfan or fludarabine, or carboplatin or pemetrexed, each are counted, so percentages may not sum to 100%.

[c] Patients could receive more than 1 supportive therapy, so percentages do not sum to 100%. Concurrent supportive therapies most commonly included topical corticosteroids, urea cream, and emollients; less common therapies included topical silver sulfadiazine, mupirocin, diclofenac gel, and compounded lidocaine-amitriptyline-ketamine ointment.

creased from 4.36 (0.60) at baseline (day 1; the first day of hdVD administration) to 3.17 (1.03) by day 5 and 2.21 (1.36) by day 10, reflecting a reduction of 1.92 (1.32) points. Clinically, this corresponded to improvement from baseline inflammatory erythema with or without bullae/desquamation to muted erythema by day 5 and little to no erythema by day 10. Inpatients receiving hdVD demonstrated a larger reduction in erythema Likert score (mean [SD], 2.42 [1.24]) than the overall cohort (**Figure 1**). Representative clinical images are shown in **Figure 2**.

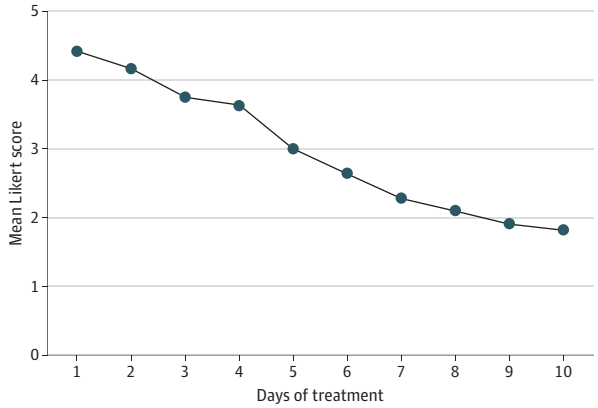
Chemotherapy or radiation was continued without interruption in 24 of the 33 patients (73%). Four patients (12%) discontinued due to toxic effects of the skin: 3 with SJS/TEN-like eruptions and 1 with severe hand-foot syndrome. Among the remaining 5 patients, therapy was discontinued in 3 (9%) for reasons unrelated to skin toxic effects, reduced in 1 (3%) due to rash, and temporarily held in 1 (3%) due to rash. Three patients developed recurrent TEC with subsequent chemotherapy, of whom 1 received repeat hdVD and rapidly improved. Pre-hdVD and post-hdVD laboratory test results were routinely monitored and showed no meaningful changes in white blood cell count or calcium levels. Mean (SD) baseline estimated glomerular filtration rate (eGFR) was 84.64 (22.40) mL/min/1.73 $m^2$; posttreatment data were inconsistently available. Among 13 patients with posttreatment 25-hydroxyvitamin D levels measured, all values remained within normal limits (range, 21-57 ng/mL [to convert to nmol/L, multiply by 2.496]), without evidence of vitamin D toxic effects (eTable 2 in Supplement 1).

## Discussion

This multicenter retrospective analysis supports hdVD as a safe therapeutic with potential to rapidly alleviate symptoms and erythema associated with TEC and ARD while enabling continuation of anticancer therapy. This improvement may reflect hdVD-mediated attenuation of proinflammatory pathways and enhanced skin repair.[1] This improved timeline provides context against expected supportive-care courses: TEC often takes up to 2 to 4 weeks to resolve, with acral erythema desquamating over 7 to 15 days and intertriginous eruptions requiring several weeks; ARD also resolves over several weeks.[11-13]

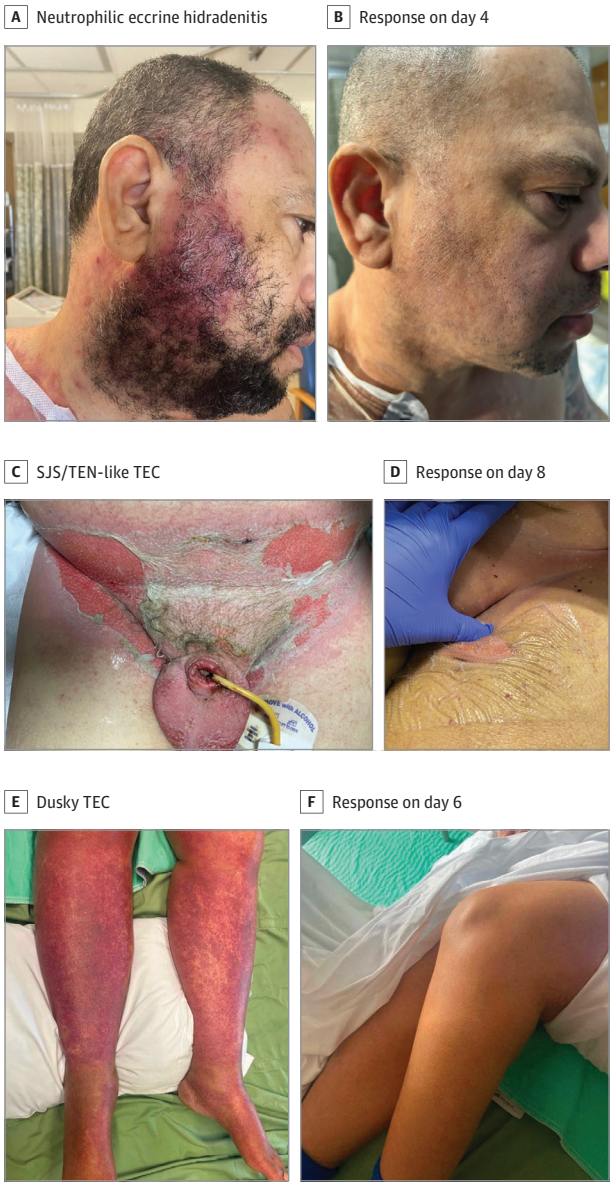
This analysis also provides initial insight into hdVD response by TEC subtype. NEH and SJS/TEN-like eruptions had the largest 10-day Likert reductions (3.0 and 2.5, respec-

Figure 1. Line Graph of Objective Erythema Assessment




Objective erythema Likert scores over time among the inpatient cohort treated with high-dose oral vitamin D.

Figure 2. Clinical Images Before and After High-Dose Oral Vitamin D Administration in Patients With Toxic Erythema of Chemotherapy (TEC)




A and B, Biopsy-proven neutrophilic eccrine hidradenitis due to high-dose cytarabine treatment, with a Likert erythema score of 4 on day 1 (A) and a reduction in the Likert erythema score to 1 on day 4 (B). C and D, Stevens-Johnson syndrome/toxic epidermal necrolysis (SJS/TEN)-like TEC secondary to fluorouracil and oxaliplatin (FOLFOX), with a Likert erythema score of 5 on day 1 (C) and a reduction in the Likert erythema score to 2 on day 8 with re-epithelialization (D). E and F, Dusky erythema of chemotherapy secondary to combination cytarabine and daunorubicin therapy, with a Likert erythema score of 4 on day 1 (E) and a reduction in the Likert erythema score to 1 on day 6 (F).

tively). By promoting M2 macrophage polarization and dampening neutrophil-driven inflammation, vitamin D accelerates neutrophilic debris clearance and tissue repair, providing a plausible basis for NEH improvement after hdVD.[14] In contrast, slower improvement in flexural/intertriginous eruptions may reflect occlusion-related delayed barrier recovery.

The inpatient cohort reported improvement sooner than outpatients, with symptom relief at a mean of 3.4 and 6.3 days, respectively, indicating potentially more rapid relief among those with severe cutaneous toxic effects. This may also reflect earlier hdVD administration for abrupt, severe inpatient presentations. More consistent inpatient assessments may also have yielded more reliable response estimates (outpatient responses may be underestimated due to less frequent follow-up during the 10-day window).

Vitamin D insufficiency is common among patients with cancer and may worsen during cytotoxic chemotherapy.[15-19] Consistent with prior reports, 14 of 23 patients in the present cohort with available pretreatment 25-hydroxyvitamin D levels had deficient or insufficient levels.[20] HdVD, hence, may promote acute immunomodulation in cutaneous injury and address vitamin D insufficiency.[1,2,8]

Furthermore, the safety profile of hdVD in oncodermatology and critical care supports its broader clinical application.[21] Single oral doses up to 600 000 IU have been administered safely in critically ill and oncology populations without increased calcium levels, systemic toxic effects, or interference with cancer therapy response.[22] In small cutaneous oncology series, single 100 000-IU regimens (repeated at 7 days) were not associated with hypercalcemia, laboratory toxic effects, or interruption of anticancer therapy.[3,4] In a multicenter trial of critically ill, vitamin D-deficient adults, a single enteral dose of 540 000 IU of cholecalciferol, compared with placebo, rapidly corrected deficiency and showed no serious adverse events, including hypercalcemia (2.7% vs 2.1%), nephrolithiasis (0% vs 0.6%), or fall-related fractures (0.8% vs 0.4%).[22] A meta-analysis of randomized trials among intensive care unit cohorts likewise found no adverse events with hdVD.[23]

Outpatient data have similarly shown no meaningful changes in calcium or eGFR with hdVD (50 000-100 000 IU/wk) for 12 months.[24] In this series, white blood cell count and calcium levels did not meaningfully change after hdVD, and no adverse events were attributed to hdVD. Post-hdVD eGFR was inconsistently available, although prior studies have not reported substantial kidney dysfunction.[3,4,21-23]

## Limitations

Limitations include the retrospective design, no placebo or untreated control group, variable follow-up, potential selection bias, incomplete laboratory data, and the use of a novel, nonvalidated Likert scale. ARD-specific conclusions are limited by small sample size. Prospective controlled trials using standardized grading of toxic effects, protocolized laboratory monitoring, and longer follow-up are needed to assess efficacy, durability, long-term safety, and optimal hdVD dosing. Prophylactic hdVD for prevention of toxic effects of the skin also warrants investigation.

## Conclusions

This case series shows that as cancer treatments grow increasingly complex, incorporating hdVD into supportive care protocols may minimize treatment interruptions, mitigate cutaneous toxic effects, and improve patient outcomes. HdVD warrants further exploration to define its role in the evolving oncodermatology landscape, where rapid, effective, and safe interventions are urgently needed, particularly for toxic effects from chemotherapy and radiation.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**Author Contributions:** Mr Patil and Dr Iriarte had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis. Mr Patil and Mr Sakunchotpanit contributed equally to this work and should be considered co-first authors. Drs Guggina and Nambudiri contributed equally to this work and should be considered co-second senior authors.
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