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Amelioration of Dominant Dystrophic Epidermolysis Bullosa Ulceration by Combination Gentian Violet and Trichloroacetic Acid Therapy

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ABSTRACT

Introduction: Dominant dystrophic epidermolysis bullosa (DDEB) is a hereditary genetic disorder with a mutation of the type VII collagen gene (COL7A1), leading to a destabilized dermal-epidermal junction. Current treatments for DDEB are supportive, and new gene therapies are being developed to target DDEB. However, gene therapy can be expensive.

Case Report: A 59-year-old woman presented with eroded blisters on her right lower extremity. Genetic testing identified a pathogenic COL7A1 mutation, confirming the diagnosis of DDEB. She was treated with 6 weeks of gentian violet and trichloroacetic acid peel, resulting in significant improvement of her lesions.

Discussion: DDEB is characterized by dysregulated inflammation of chronic wounds and aberrant fibroblast activity. Gentian violet and trichloroacetic acid may address inflammation while reducing fibroblast activity and preventing infection. The treatment worked well for the patient, and there was minimal pain with the application of these topical therapies.

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INTRODUCTION

Dominant dystrophic epidermolysis bullosa (DDEB) is a hereditary genetic disorder with a mutation of the type VII collagen gene (COL7A1), resulting in a defect of anchoring collagen VII (Col VII), which stabilizes dermal-epidermal adhesion.[1] Consequently, patients with DDEB experience skin blisters and fragility. Most clinical consequences of DDEB result from delayed wound healing, aberrant skin remodeling, and chronic inflammation.[2]

Currently, clinical management for DDEB focuses on supportive treatment and trauma avoidance.[1] Patients may undergo skin transplant or surgical repair of complications that develop from scarring following epidermal injury, such as mitten hand deformities.[1] Biological treatments such as dupilumab have been studied for controlling itch in patients with pruritic subtypes of DDEB.[3] Emerging treatments in gene and stem cell therapy show promise in clinical application, while new advancements in bioengineered skin substitutes promote local wound healing.[1]

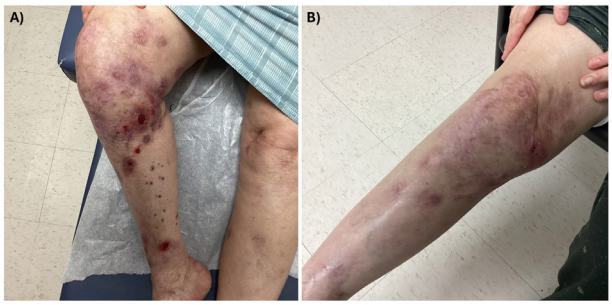
We report a case of a patient with DDEB lesions of the lower extremity treated with gentian violet (GV), a small molecule with both anti-inflammatory and antibacterial properties, with trichloroacetic acid (TCA) to enhance GV penetration into cutaneous tissue.[4] Over the course of 6 weeks, the patient's wounds demonstrated substantial healing, reepithelialization, and recovery.

CASE REPORT

Our patient, a 59-year-old woman, presented with eroded blisters on her right lower extremity. She was told she had a genetic blistering disease with lifelong effects, especially on the lower extremities. She had no family history of blistering disease. Prior to her office visit, she had treated her blisters and wounds with supportive measures, including dressings to reduce friction. Based upon the presence of scarring, location, and morphology of the blisters, and absence of family history, we felt that a diagnosis of a dominant dystrophic epidermolysis was likely. Genetic testing was performed to determine whether she would be eligible for gene therapy with Vyjuvek (beremagene geperpavec).

Genetic testing showed that the patient was heterozygous in the COL7A1 gene for a pathogenic c.6235G.A substitution, which has been reported in patients with DDEB, confirms the diagnosis.

FIGURE 1. Lesion site of patient with DDEB (A) before treatment with eroded and flaccid blistering and (B) after 6 weeks of treatment with 1% gentian violet and 50% TCA



However, Vyjuvek was denied by her insurance. Because of this, we opted to treat her for 6 weeks with one coat of 50% TCA on affected skin immediately followed by application of 1% gentian violet.[4] The patient did not notice pain upon application of TCA directly to the ulcers. The treatment was applied every two weeks in the office, and she also applied gentian violet twice weekly at home. After 6 weeks of treatment, the lesional skin showed significant improvement (Figure 1).

DISCUSSION

Recently, gene therapy has been utilized in the treatment of epidermolysis bullosa,[1] and overexpression of wild-type Col VII could potentially ameliorate the phenotype of DDEB by outcompeting mutant Col VII. However, permanent correction has not been definitively accomplished and may require multiple expensive treatments. Of interest, while each keratinocyte contains a pathogenic mutation causing DDEB, the phenotype in this patient manifested itself on the extensor surface of the leg. This implies that additional factors are required for full-fledged expression of the ulceration phenotype. The additional factors that promote the full phenotype present an opportunity for therapeutics. Localized inflammatory stromal changes in DDEB likely contribute to ulcer formation, and it is possible that the combination of gentian violet and TCA may promote reversion to a nonulcerative stroma. Advantages of this treatment include ease of administration and expense. The patient did not notice pain upon application of TCA directly to the ulcers. GV and TCA may also promote wound healing through advantages in antimicrobial action, as well as controlling inflammation and fibrosis.

Controlling the inflammatory stage of chronic DDEB wounds is crucial in facilitating wound healing. Increased activity of elastase and matrix metalloproteinase in chronic wounds acts as a counterforce to growth factors and extracellular matrix that try to remodel epidermis in the wake of disrupted dermal-epidermal junction.[5] Inflammation also disrupts keratinocyte differentiation needed to restore the skin barrier at wound sites, and extended inflammation can lead to fibrosis or hypergranulation instead, leading to complications of DDEB such as mitten hand.[2,6] Aberrant activated fibroblast activity may also contribute to dysregulated stroma in lesional DDEB.[7]

Treatment with GV and TCA may synergize to ameliorate ulceration in DDEB through reducing inflammation and fibroblast activity.

GV has the effect of reducing inflammatory expression that is present in lesional DDEB skin. Other cutaneous disorders characterized by aberrant inflammation, such as palmoplantar psoriasis, have shown robust response to GV therapy, and previous case reports have documented GV suppressing granulation tissue in patients with non-Herlitz junctional epidermolysis bullosa with subsequent wound reduction.[4,8]

In one particular mechanism, increased inflammation in DDEB may be mediated by activation of the T helper (Th) 1, 2, and 17 pathways. Transcriptomes of DDEB skin versus healthy skin have shown increased transcription of GATA3+ Th2 cells in affected skin.[3] Nuclear factor kappa B (NF-kB) is one key mediator in the activation of GATA3+ Th2, and GV inhibits NF-kB via decreasing oxidation of inhibitor of nuclear factor kappa B (IkB).[9,10] Thus, GV may contribute to dampening GATA3+ Th2 inflammation expressed in lesional DDEB skin. Dupilumab treatment reduced GATA3+ Th2 expression in DDEB, reducing pruritus and the itch-scratch-blister cycle of DDEB, and GV may have a similar effect, targeting the same inflammatory culprits.[2,3]

Additionally, studies investigating GV use on chronic wounds have highlighted other advantages of GV as a wound dressing. Aside from low cost, GV effectively decreases superficial bacterial burden as a wound dressing, especially against gram-negative bacteria.[11]

TCA has been shown to decrease collagen and matrix metalloproteinase synthesis in fibroblasts, and has been used clinically to reduce scarring in conditions such as acne.[12,13] Lesional DDEB has a stroma that is over-fibrotic, leading to a disorganized skin barrier, and as such, intermittent application of TCA in the patient may have reduced this fibrotic effect.

Overall, the regional application of GV and TCA to wounds of patients with DDEB could help modulate the aberrant inflammatory profile found in lesional skin. In addition, it creates a supportive environment for healing and acts as an antimicrobial to prevent infection and inflammatory responses to bacteria. This case highlights the topical application of this inexpensive dressing as an effective method for promoting healing in DDEB lesions.

One limitation of this study is the difficulty in assessing progress on DDEB wounds, given their tendency to heal, recur, and then

re-heal.[5] Therefore, evaluating healing at one time point does not necessarily mean the success of an intervention. However, there was no ulcer recurrence at her site at her follow-up appointment a month following the end of treatment, whereas re-ulceration is typical. In addition, previous conservative treatment had not yielded improvement in this patient, providing evidence that this intervention helped ameliorate her ulcers.

DISCLOSURES

The authors have no conflicts of interest to disclose. Patient Consent: The authors obtained written consent from patients for their photographs and medical information to be published in print and online, and with the understanding that this information may be publicly available.

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