

Oral dehydroepiandrosterone might prevent frequent tears in atrophic skin: A case report



Harry W. Daniell, MD, FACP
Davis, California

INTRODUCTION

Large superficial tears frequently develop during minor trauma to the fragile skin of older men and women. These tears are more common in chronically ill or malnourished patients,^{1,2} can develop during attempted assisted transfer of weak patients, and were recently reported to have been treated annually in 1.5×10^6 MediCare patients during their care in an acute or long-term medical care facility. They also develop frequently in elderly out-patients and those using inhaled or systemic corticosteroids for extensively lengths of time.³

Postmenopausal hormonal supplementation⁴ and other systemic and local therapies have not been demonstrated to improve dermal thickening. Levels of dehydroepiandrosterone (DHEA) and of its sulfonated ester DHEAS are present in equilibrium. Their serum levels are most easily quantitated by measurement of DHEAS. Both decline rapidly with advancing age, and after age 60 years, average levels are only 20% of those of younger adults. Supplementation to increase DHEA and DHEAS levels is easily and inexpensively maintained by ingestion of oral DHEA, which is classified as a food additive in the United States and widely available without prescription; DHEA can be found in a variety of products that, however, contain unverified amounts, considering the product content advertised on the labels have not been verified. In the only available placebo-controlled trial of supplemental DHEA, Baulieu et al⁵ documented less progressive skin thinning, improved skin hydration, improved sebum production, and less bone mineral density loss in 140 postmenopausal women aged 60-79 years who ingested 50 mg DHEA daily for 12 months.

Frequent skin application of DHEA gel or lotion has improved skin collagen production in a dose-dependent manner in several investigations,⁶⁻⁸ and the addition of DHEA into the culture media of human skin fibroblasts has resulted in greater procollagen and collagen production in several studies.⁶⁻⁸ Multiple investigators have suggested the potential for DHEA as a skin antiaging agent,⁶⁻⁸ and Robinzon et al⁹ suggested that the low DHEA and DHEAS levels characteristic of long-term corticosteroid use might indicate a potential by DHEA replacement for the prevention of skin atrophy and other catabolic corticosteroid changes. We report details of serum levels of DHEA and DHEAS and the frequency of atrophic skin tears in an elderly man before and after his daily DHEA ingestion.

CASE REPORT

An 87-year-old retired internist in apparent good health had fragile skin that developed 10 triangular superficial tears on his arms or lower legs during an 8-month interval. He had no history of endocrine disease or corticosteroid use. Most tears had resulted from contact with wire garden fencing or another hard unyielding object, which had resulted in separation of his superficial epidermis from its deeper layers. Each of his skin tears measured 2-4 cm on each of 3 sides and healed rapidly after early epidermal application of a small amount of antibiotic cream and several days of protection with a nonstick gauze secured in place by an elastic bandage.

When the patient was 81 years of age, multiple serum DHEAS concentrations ranging 50-69 mcg/dL (reference range 30-200 mcg/dL) had been recorded

From the Department of Family Practice, University of California Medical School at Davis.

Funding sources: Supported by Medical Data Analysis without specific grant support. The opinions expressed are the private views of the author.

Conflicts of interest: None declared.

Correspondence to: Harry W. Daniell, MD, FACP, Medical Data Analysis, 1670 Market St #122, Redding, CA 96001. E-mail: hwdaniell@aol.com.

JAAD Case Reports 2017;3:534-5.
2352-5126

© 2017 by the American Academy of Dermatology, Inc. Published by Elsevier, Inc. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

<https://doi.org/10.1016/j.jidcr.2017.09.019>

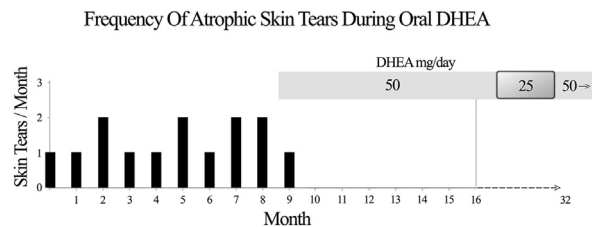


Fig 1. Frequency of atrophic skin tears in 87-year-old man before and after taking oral DHEA. DHEA, Dehydroepiandrosterone.

(during an unrelated study), and at 85 years of age, another DHEAS of 32 mcg/dL was recorded. After development of skin tears, he began to ingest DHEA (Finest Nutrition, Walgreens Corporation, Deerfield, IL) 50 mg daily. Subsequently, he made no effort for additional protection with gloves or long sleeves, and during the next 2 years, he developed only 1 subsequent skin tear 3 weeks after beginning DHEA (Fig 1). After 10 months of therapy, his DHEAS level was 706 mcg/dL. In response to this high reading, his DHEA intake was lowered to 25 mg daily for 2 months. His DHEAS level decreased to 169 mcg/dL, and he then resumed back to 50 mg daily. During the next 18 months, he remained in good health without the development of additional tears or apparent side effects from his new therapy.

DISCUSSION

Labrie et al investigated the mechanism by which oral DHEA might improve skin integrity.¹⁰ These investigators demonstrated increased systemic intracellular androgen activity during DHEA ingestion. Androgen activity potentially increased as a result of increased production of the androgen precursor, androstenedione, during first-pass DHEA metabolism through gastrointestinal and hepatic tissues. Increased androgen use by both skin and bones is consistent with improved collagen content, resulting in increased protection from injury.

DHEA therapy has not previously been suggested as a treatment for or a preventive measure for skin tears, even though DHEAS levels are depressed among patients taking long-term systemic or inhaled corticosteroid therapy and older persons who have

frequent skin tears.⁹ DHEAS concentrations have not been reported in patients utilizing topical corticosteroids, some of whom have skin atrophy. DHEA appears relatively safe, considering no significant side effects were reported after 2 years of daily ingestion of DHEA 50 mg.⁵

Our observations suggest a potential benefit for measuring DHEAS levels in patients with fragile skin, multiple skin tears, or documented skin atrophy. In these subjects, oral DHEA therapy might be a reasonable therapeutic intervention for those with low DHEA levels or frequent skin tears, pending the results of long-term placebo-controlled trials evaluating its effectiveness and safety.

I appreciate the assistance of Marcy Bates in the preparation of this article.

REFERENCES

1. Stephen-Haynes J, Carville K. Skin tears made easy. *Wounds Int.* 2011;2:1-6.
2. Baranoski S. Meeting the challenge of skin tears. *Adv Skin Wound Care.* 2005;18:74-75.
3. Capewell S, Reynolds S, Shuttleworth D, et al. Purpura and dermal thinning associated with high dose inhaled corticosteroids. *BMJ.* 1990;300:1548-1551.
4. Sator PD, Sator MO, Schmidt JB, et al. A prospective, randomized, double-blinded, placebo-controlled study on the influence of a hormone replacement therapy on skin aging in postmenopausal women. *Climacteric.* 2017;10:320-334.
5. Baulieu EE, Thomas G, Legrain S, et al. Dehydroepiandrosterone (DHEA), DHEA sulfate, and aging: contribution of the DHEAge Study to a sociobiomedical issue. *Proc Natl Acad Sci U S A.* 2000;97:4279-4284.
6. Calvo E, Luu-The V, Morissette J, et al. Pangenomic changes induced by DHEA in the skin of postmenopausal women. *J Steroid Biochem Mol Biol.* 2008;112:186-193.
7. El-Alfy M, Deloche C, Azzi L, et al. Skin responses to topical dehydroepiandrosterone: implications in antiaging treatment? *Br J Dermatol.* 2010;163:968-976.
8. Shin MH, Rhie G, Park CH, et al. Modulation of collagen metabolism by the topical application of dehydroepiandrosterone to human skin. *J Invest Dermatol.* 2005;124:315-323.
9. Robinzon B, Cutolo M. Should dehydroepiandrosterone replacement therapy be provided with glucocorticoids? *Rheumatology (Oxford).* 1999;38:488-495.
10. Labrie F, Belanger A, Labrie C, et al. Bioavailability and metabolism of oral and percutaneous dehydroepiandrosterone in postmenopausal women. *J Steroid Biochem Mol Biol.* 2007;107:57-69.