Author Contributions: Study concept and design: Robinson. Acquisition of data: Robinson and Ortiz. Drafting of the manuscript: Robinson. Critical revision of the manuscript for important intellectual content: Ortiz. Obtained funding: Robinson. Administrative, technical, and material support: Ortiz. Study supervision: Robinson.

Financial Disclosure: None reported.

Funding/Support: This study was supported by grant 5R21 CA-103833-02 from the National Cancer Institute (Dr Robinson).

Additional Contributions: Rob Turrisi, PhD, performed the statistical analysis for this study.

Disclaimer: Dr Robinson was not involved in the editorial evaluation or editorial decision to accept this work for publication.


Lichen Planopilaris: Retrospective Study and Stepwise Therapeutic Approach

Lichen planopilaris (LPP) is a primary lymphocytic scarring alopecia that causes inflammation, erythema, pruritus, dysesthesia, and alopecia that can be treatment resistant. After approval from the institutional review board, we performed a retrospective case analysis of alopecia due to LPP to assess possible therapeutic effectiveness.

Methods. All medical charts with International Classification of Diseases, Ninth Revision (ICD-9) diagnoses of alopecia (2004-2007) were analyzed: 674 cases were non-scarring (81%), and 159 were scarring alopecia (19%).

Based on clinical-pathologic correlation, LPP was diagnosed in 45 cases (28% scarring alopecia, 5% overall), and all patients were diagnosed by the same observer (J.C.E.). According to the criteria of the North American Hair Research Society, histologic evidence of lymphocytic scarring inflammation includes lupus erythematosus (LE), classic pseudopelade, central centrifugal cicatricial alopecia, alopecia mucinosa, keratosis follicularis spinulosa decalvans, and LPP.

Lichen planopilaris and LE require close scrutiny for distinction. Histologic criteria used to distinguish LPP from LE were superficial infiltrate, lack of basement membrane thickening, excess mucin, epidermal thinning, or telangiectasia. Clinically, a diagnosis of LPP was favored over LE if the alopecia had perifollicular hyperkeratosis and erythema, lack of follicular plug-
Corroborates that a consistent and successful therapy for refractory disease remains elusive.

Limitations to this study include its retrospective nature and end points that did not include percentage of scalp hair loss or hair counts. Clinically, we suggest offering patients stepwise therapy with doxycycline or hydroxychloroquine as first-line systemic agents. Our data do not suggest that one is superior. If needed, transition to mycophenolate mofetil or acitretin therapy can be made for relief of symptoms, but potential for adverse effects must be considered. In our experience, the risk-benefit ratios of other agents (e.g., cyclosporine and thalidomide) preclude their use for this non–life-threatening condition. Evidence-based therapy with a prospective, multicenter, randomized, placebo-controlled study with set end points is needed.

Lori A. Spencer, MD, PhD
Elena Balestreire Hawryluk, PhD
Joseph C. English III, MD

Correspondence: Dr English, Department of Dermatology, University of Pittsburgh, 190 Lothrop St, Ste 145, Lothrop Hall, Pittsburgh, PA 15213 (englishjc@upmc.edu).

Author Contributions: Study concept and design: Spencer and Hawryluk. Acquisition of data: Spencer, Hawryluk, and English. Analysis and interpretation of data: Spencer, Hawryluk, and English. Drafting of the manuscript: Spencer and Hawryluk. Critical revision of the manuscript for important intellectual content: Spencer, Hawryluk, and English. Administrative, technical, and material support: English. Study supervision: English.

Financial Disclosure: None reported.


COMMENTS AND OPINIONS

Intravenous Immunoglobulin Therapy for Dystrophic Calcification Cutis: Unreliable in Our Hands

We read with interest the recent report, “Response of Dystrophic Calcification to Intravenous Immunoglobulin,” by Schanz et al1 detailing the response to intravenous immunoglobulin (IVIg) of 1 patient with CREST syndrome–associated dystrophic calcification (CREST syndrome includes calcinosis cutis, Raynaud phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia). Because the more traditional medical therapies, including warfarin, diltiazem hydrochloride, probenecid, colchicine, and bisphosphonates, are seldom reliable, newer safe and effective therapies are sought.2 Unfortunately, while the success reported by Schanz et al is encouraging, we have experienced less success with IVlg therapy for dermatomyositis-associated dystrophic calcification.

Report of Cases. We describe 2 patients with dermatomyositis, both experiencing chronic, extensive, and progressive dystrophic calcification (Figure) despite nearly 5 years of therapy with IVlg, 2g/kg/mo, in divided doses. Concurrent therapies included diltiazem hydrochloride, 240 mg, and hydroxychloroquine sulfate, 200 mg, orally twice daily as well as alendronate sodium, 70 mg orally once per week. Both patients have normal renal function and calcium-phosphorus parameters.

Comment. While IVlg is an excellent treatment option for dystrophic calcification associated with collagen vascular disease, it does not appear to be universally successful. For these reasons, the search for more reliable therapies for dystrophic calcification must continue.

Andrew H. Kalajian, MD
Jennifer H. Perryman, MD
Jeffrey P. Callen, MD

Correspondence: Dr Kalajian, 310 E Broadway, Ste 200, Louisville, KY 40202 (akalajian@yahoo.com).

Financial Disclosure: Dr Callen has received honoraria from Amgen, Abbott Immunology, Genentech, Centocor, Electrical Optical Sciences, Medicis, and Steifel. He serves on a safety monitoring committee for Genmab.

Disclaimer: Dr Callen is Associate Editor of the Archives of Dermatology, but he was not involved in any of the decisions regarding review or acceptance of this article.