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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 clinicopathologic 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,1 histologic evidence of lymphocytic scarring inflammation includes lupus erythematosus (LE), classic pseudopelade, central centrifugal cicatricial alopecia, alopecia mucinosa, keratosis follicularis spinulosa decalvans, and LPP.1

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,
ging, ill-defined areas of involvement, and no evidence of perilesional hyperpigmentation. Data revealed that 42 of the 45 patients were white (93%), 31 were female (69%), and average age at the time of diagnosis was 51 years. Interestingly, 3 of the 45 patients were diagnosed with a frontal fibrosing alopecia variant with eyebrow loss, and 1 of 45 had Graham-Little-Piccardi-Lasseur syndrome. These numbers substantiate previously reported data regarding prevalence and patient characteristics of LPP.

Twenty-nine patients with LPP met the criteria for analysis of treatment interventions. Those who did not return for follow-up, failed to obtain initial laboratory work, sought care elsewhere, or refused to undergo biopsy were not included in the analysis. Improvement was defined as follow-up as the absence of reported symptoms (pruritus, burning, and/or dysesthesia), lack of progression, reduction in erythema and follicular hyperkeratosis found on examination, and the ability to discontinue therapy. Most of the patients underwent treatment with supplemental topical steroids, topical minoxidil, 5%, and/or oral biotin with or without orthosilicic acid. A summary of instituted systemic immunosuppression approaches appears in the Table.

Results. For 15 patients, an initial tetracycline therapeutic choice (doxycycline hyclate) was given based its relatively low adverse effect profile and lack of laboratory monitoring required: 27% of patients showed improvement (n=4). Twenty-two of our patients took hydroxychloroquine sulfate during their therapy (some initially, others after doxycycline failure) with 9 of 22 showing improvement (41%). The Fisher exact test comparing these initial treatments did not find significant differences (P=.74). Of the patients for whom hydroxychloroquine treatment failed, 10 were treated with mycophenolate mofetil; none received this therapy without a failed trial of hydroxychloroquine. Three patients showed improvement under treatment with mycophenolate mofetil (30%). For those who did not improve, subsequent transition to methotrexate therapy in 1 patient was unsuccessful, and 2 of 3 patients who attempted acitretin treatment found success. The Fisher exact test comparing mycophenolate mofetil and acitretin found no significant difference (P=.51).

Comment. Many systemic agents have been used to treat LPP with limited success.1”5 Our retrospective analysis
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.

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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 IVIg 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 IVIg, 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 IVIg 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.

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