EA DV SYMPOSIUM 2026
ATHENS 7 - 9 MAY



Abstract N°: ID-297

Topic: Inflammatory skin diseases

Graham-Little-Piccardi-Lassueur syndrome and its relation to Antigen mimicry

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Introduction

Graham-Little-Piccardi-Lasseur syndrome (GLPLS) is a rare lichenoid dermatosis classified as a variant of follicular lichen planus, also known as classic lichen planopilaris. The condition is characterized by the triad of cicatricial scalp alopecia, noncicatricial alopecia in the axillary and groin regions, and numerous follicular papules distributed across the body (Fig.1a-d).



Fig.1a-d: Dermatological findings: 1a: Capillitium: diffuse cicatricial alopecia with plaque-like areas of desquamation; 1b: Numerous livid hypertrophic papules and nodules with fine desquamation on the the trunk 1c: Reduced body hair, in the right axilla (noncicatricial alopecia). 1d: Numerous livid hypertrophic papules and nodules with fine desquamation on the right and left hands

Materials and Methods

We present a 64-year-old female with clinically and histologically confirmed GLPLS. Two biopsies were conducted, resulting in lichen planopilaris/ pseudopelade Brocq and lichen planus hypertrophicus (Fig.2a-c).

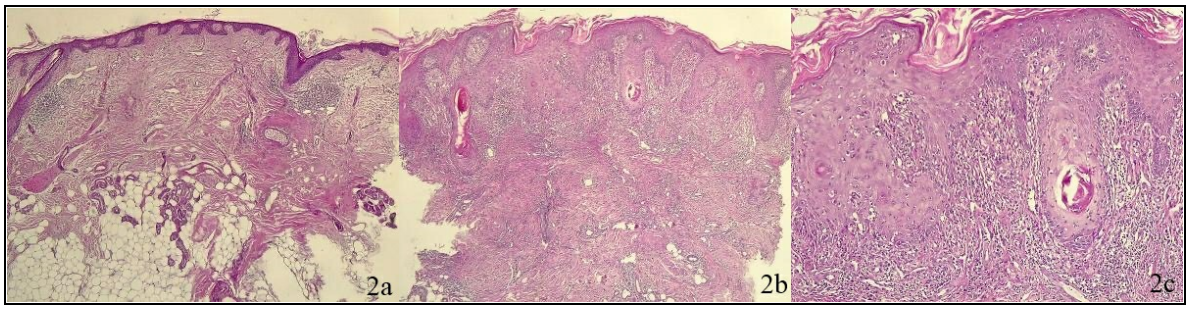


Fig.2a-c: Histology findings: A - pronounced ortho- and follicular hyperkeratosis, uneven acanthosis, and a reduced number of sebaceous glands throughout the dermis, replaced by diffuse fibrous tracts. A retained hair follicle fragment was covered by a lichenoid inflammatory reaction, with round cells destroying the external epithelial sheath. These findings were consistent with lichen planopilaris/ pseudopelade Brocq; B,C - marked ortho- and follicular hyperkeratosis, irregular acanthosis alternating with pseudoepitheliomatous hyperplasia, vacuolar degeneration of basal keratinocytes, and a lichenoid lympho-plasmacytic inflammatory infiltrate obscuring the dermo-epidermal junction and lining the papillary dermis. These findings were consistent with lichen planus hypertrophicus 2a: cicatricial alopecia HE x 40 2b: Lichen planus hypertrophicus HE x 40 2c: Lichen hypertrophicus HE x 100

Results

Initial therapy included loratadine and topical clobetasol propionate. Due to the suspicion of possible drug-induced autoimmunity, the antihypertensive therapy, which consisted of valsartan, bisoprolol, spironolactone, and chlorthalidone, was discontinued and replaced with moxonidine. Urinary infection caused by Escherichia coli and dental infection were noted. Prescribed outpatient treatment included acitretin, bilastine, and topical prednisolone for the scalp. Improvement was observed in the lesions located on the trunk and upper and lower extremities following betamethasone/salicylic acid ointment was prescribed, and methylprednisolone aceponate cream. Re-application or return of the old systemic medication on an outpatient basis resulted in a worsening/exacerbation of the clinical picture and a re-need to change medication. It is this fact that suggests that polymedication could also be considered as a trigger of lichen planus and its subforms such as GLPLS.

Conclusions

The hypothesis about antigen / molecular mimicry, based on the surrounding alterations in tissue homeostasis as a potential trigger factor for autoimmunity is being discussed, with a specific focus on infectious and drug-induced forms of lichen planus, as well as Graham-Little-Lasseur syndrome.