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CASE REPORT

Perifollicular Elastolysis: A Case Report and Literature Review

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Abstract: Perifollicular elastolysis (PE) is a rare acquired elastolytic disorder, first described by Varadi and Saqueton in 1970, characterized by focal loss of elastic fibers around hair follicles. It is frequently underrecognized and mistaken for common conditions like acne vulgaris. This case report describes an 18-year-old female presenting with asymptomatic, skin-colored papules diffusely distributed on the chest and back persisting for over a year. Dermatological examination revealed millet-sized, noninflammatory folliculocentric papules with smooth surfaces. Histopathology demonstrated perifollicular elastic fiber fragmentation and reduction on Verhoeff-Van Gieson staining, accompanied by mild perivascular inflammation, confirming PE diagnosis. The discussion synthesizes current understanding of PE's clinical spectrum, highlighting its differentiation from mid-dermal elastolysis (MDE), papular elastorrhexis, and other elastolytic disorders through histopathological hallmarks. While the exact pathogenesis of PE remains unclear, potential triggers include mechanical trauma, bacterial elastase activity, and autoimmune dysregulation. Current management focuses on minimizing mechanical trauma and topical therapies, though no standardized treatment exists. This case underscores the need for greater awareness of PE and calls for further research to elucidate its molecular mechanisms and develop evidence-based treatments for this benign yet cosmetically concerning condition.

Keywords: perifollicular elastolysis, mid-dermal elastolysis, elastic fibers, elastolytic disorders

Case Presentation

An 18-year-old female patient presented to our hospital with multiple millet-sized, skin-colored papules on her trunk persisting for over a year (Figures 1–3). Approximately one year prior to admission, she had developed pinhead-sized, skin-colored, flat papules on her chest and back without identifiable triggers. The lesions were asymptomatic, with no associated pruritus or pain. Despite gradual increase in the number of lesions, the patient had not sought medical evaluation or treatment prior to this presentation. Dermatological examination revealed multiple well-demarcated, skin-colored, flat papules ranging from pinhead to millet size were observed diffusely distributed across the anterior chest and back. The lesions displayed smooth surfaces without coalescence. Each papule is perforated by a hair follicle at its center. Dermoscopy revealed uniformly distributed yellowish-white circular homogeneous structures lacking discernible vascular patterns with vellus hairs observed centrally within these structures. (Figure 4). Histopathological finding revealed superficial dermis with mild perivascular inflammation (Figures 5–6). Verhoeff-Van Gieson staining identified marked reduction and fragmentation of perifollicular elastic fibers (Figures 7–8). Based on the clinical presentation, histopathological features, and elastin staining results, a definitive diagnosis of perifollicular elastolysis (PE) was established.

Discussion

Perifollicular elastolysis (PE), first described by Varadi and Saqueton in 1970, is a rare acquired disorder characterized by focal loss of elastic fibers around hair follicles.[1] Clinically, it manifests as asymptomatic, noninflammatory, 1–4 mm folliculocentric papules that are skin-colored to yellowish-white, often featuring a central dell or wrinkled surface. These lesions have a predilection for the trunk, neck, upper extremities, and intertriginous areas (eg, inframammary folds,

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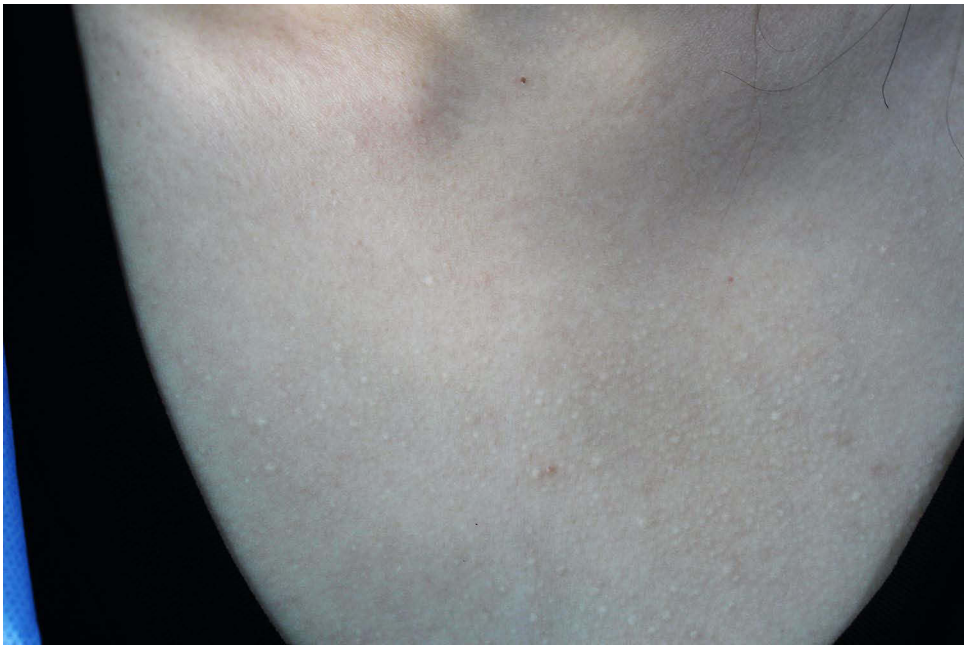

Figure 1 Numerous skin-colored flat papules distributed across the anterior chest. The lesions exhibit well-defined borders, possess a smooth texture, and do not merge with one another.

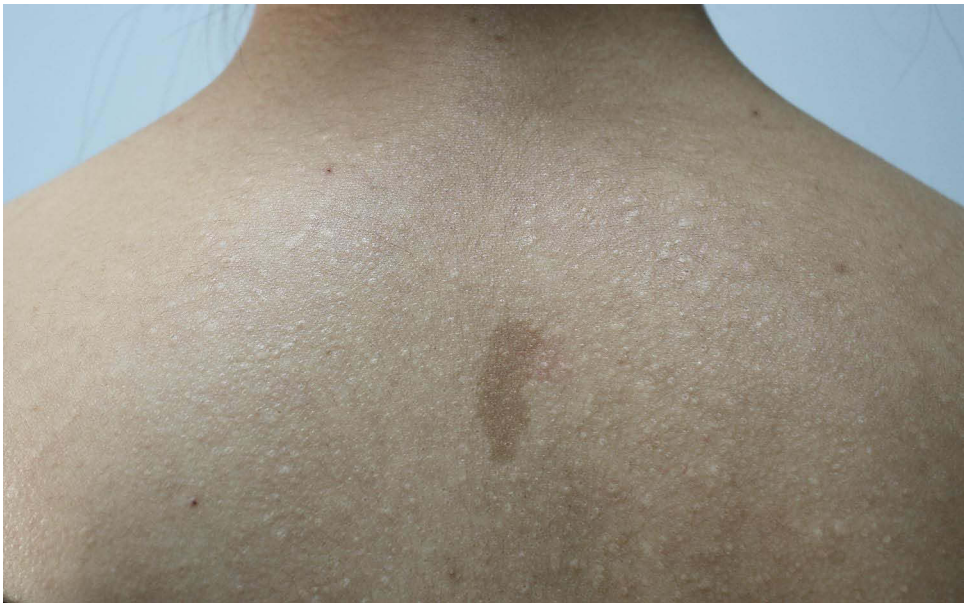

Figure 2 Numerous skin-colored flat papules distributed across the upper back. The lesions exhibit well-defined borders, possess a smooth texture, and do not merge with one another.

axillae), and may coalesce into plaques. While historically linked to acne vulgaris, recent reports associate PE with diverse triggers, including mechanical trauma, autoimmune diseases, and bacterial elastase activity.[2,3] Unique cases report PE in atypical locations, such as the armpits due to repeated shaving trauma or in association with Behçet's disease.[4,5]

Diagnostically, PE must be distinguished from other acquired elastolytic disorders. Unlike mid-dermal elastolysis (MDE), it lacks mid-dermal band-like elastolysis but shares overlapping clinical features with anetoderma and papular elastorrhexis.[6] Confirmation typically requires histopathological examination with special elastic stains to demonstrate

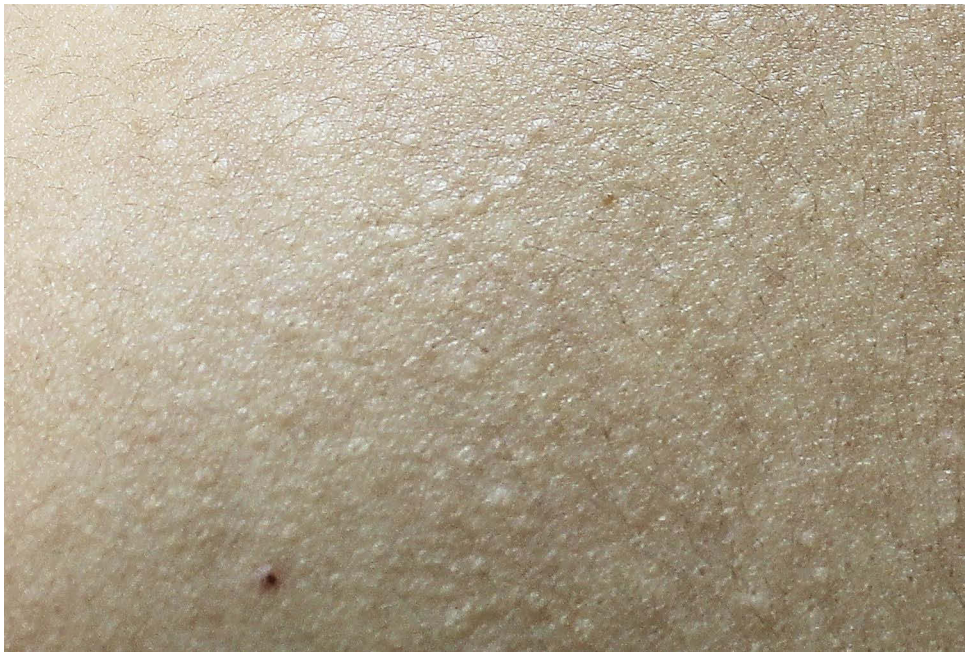

Figure 3 Each papule is perforated by a hair follicle at its center. (close-up view).

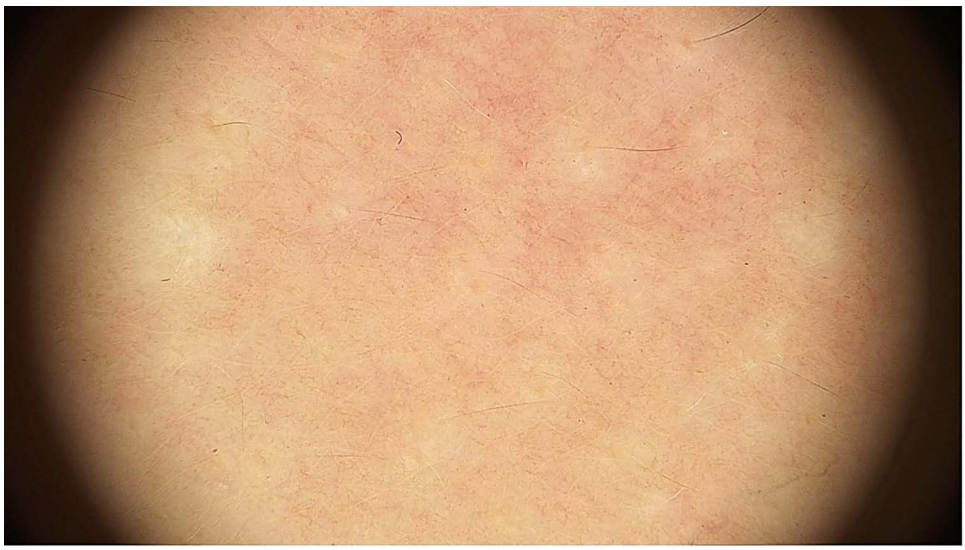

Figure 4 Under dermoscopy, one can observe uniformly distributed yellowish-white round homogeneous structures, devoid of distinct vascular structures with vellus hairs observed centrally within these structures. (fotofinder x 20).

elastolysis around follicles. This review synthesizes current knowledge on PE, emphasizing its clinical, histopathological, and etiological spectrum.

The histopathological diagnosis of PE is based on distinctive features best visualized with specialized staining techniques. Routine H&E staining often shows unremarkable skin, leading to underdiagnosis. The key indicator of PE is the selective loss of elastic fibers around hair follicles and sebaceous glands, while collagen remains intact. Elastic stains, such as Verhoeff-Van Gieson, reveal fragmented or absent fibers around these structures, with preserved elastin in adjacent dermis.[1] This staining technique highlights elastic fibers in blue-black and collagen in bright red, marking focal elastolysis. A characteristic halo of absent or fragmented elastic fibers is seen around upper hair follicles, typically extending 50–100 μm into the perifollicular dermis, while inflammation is usually absent, and collagen architecture is

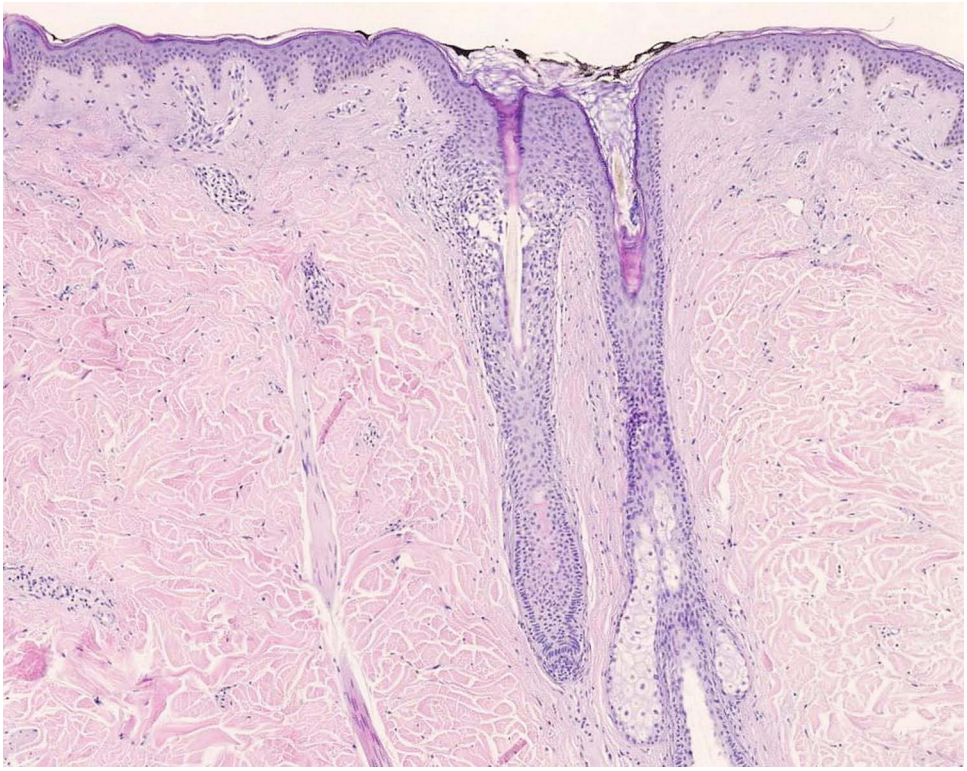

Figure 5 The epidermis demonstrates atrophy and thinning, accompanied by increased pigmentation in the basal layer. (hematoxylin-eosin stain, 50×original magnification).

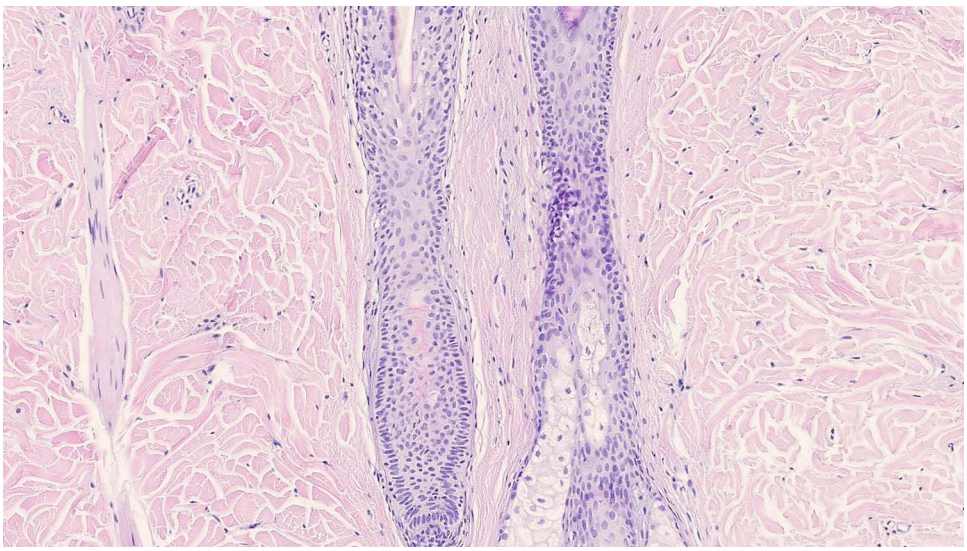

Figure 6 Histopathological examination revealed sparse perifollicular fibrosis accompanied by a modest infiltration of lymphocytes surrounding the blood vessels. (hematoxylin-eosin stain, 100×original magnification).

preserved.[3] This pattern differentiates PE from other elastolytic disorders, making elastic staining crucial for accurate diagnosis since key features are not visible with standard H&E stains.

The pathogenesis of PE remains incompletely understood, with several proposed mechanistic pathways emerging from current research. First, bacterial elastase production has been postulated as a potential contributor, particularly from cutaneous flora such as *Staphylococcus epidermidis* and *Cutibacterium acnes*, which may degrade perifollicular elastic fibers through enzymatic activity.[1] However, this hypothesis remains controversial as contradictory evidence from

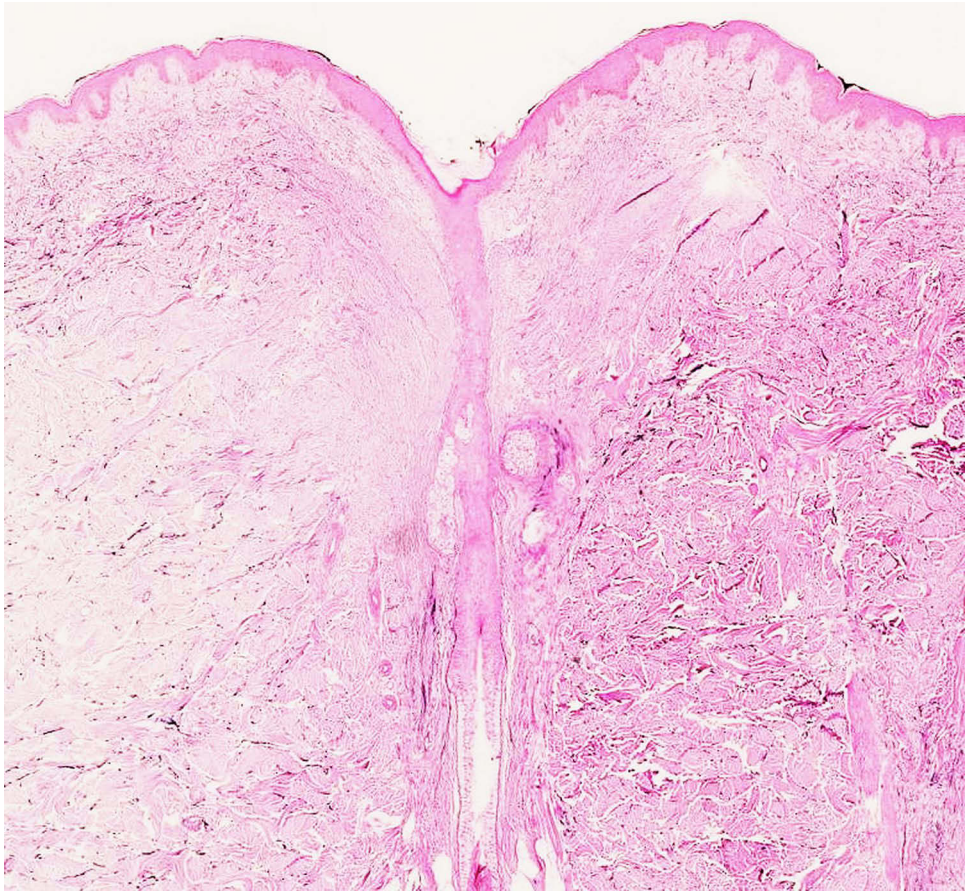

Figure 7 Elastic fiber staining demonstrates a significant reduction and fragmentation of elastic fibers within the superficial and middle layers of the dermis surrounding hair follicles. (Verhoeff-Van Gieson staining, 25×original magnification).

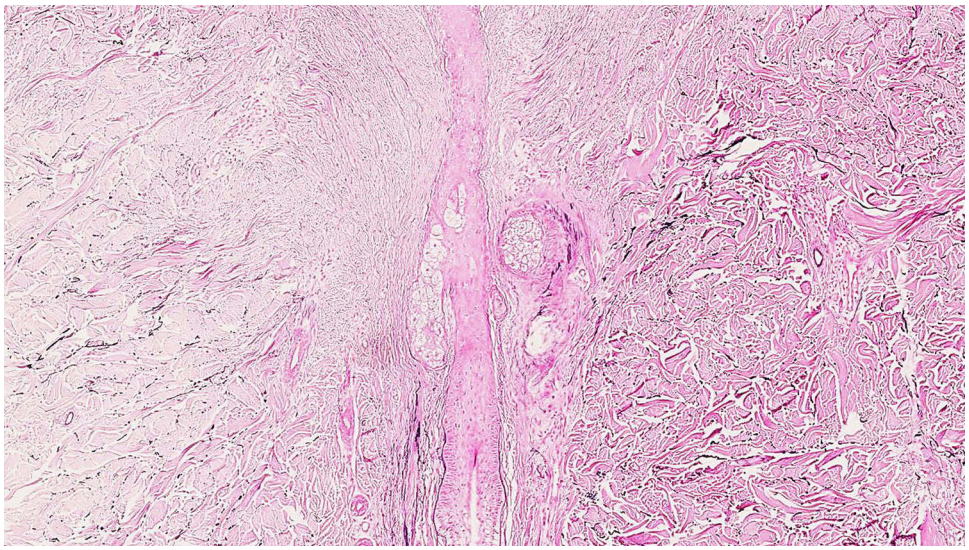

Figure 8 Elastic fiber staining demonstrates a significant reduction and fragmentation of elastic fibers within the superficial and middle layers of the dermis surrounding hair follicles. (Verhoeff-Van Gieson staining, 50×original magnification).

bacterial culture studies by Dick et al demonstrated no detectable elastolytic capacity in clinical isolates.[7] Second, mechanical trauma induced by repetitive physical manipulation including shaving practices and friction from axillary hair removal has been implicated in causing follicular microtrauma that may initiate elastolytic processes.[4] Third, clinical

associations with inflammatory conditions such as Behçet's disease, atopic dermatitis, and acne vulgaris suggest potential involvement of sterile inflammatory pathways or cytokine-mediated elastin degradation mechanisms.[5,8,9] Finally, emerging evidence points to potential autoimmune components, particularly through dysregulation of extracellular matrix maintenance via abnormal matrix metalloproteinase (MMP) activity or aberrant immune responses mediated by CD68+ histiocytes.[10] These multifactorial hypotheses underscore the complex interplay of microbial, mechanical, inflammatory, and immunological factors in PE pathogenesis.

PE requires careful clinicopathological differentiation from other elastolytic disorders with overlapping features. MDE, classically categorized into Type I (wrinkled plaques) and Type II (perifollicular protrusions), demonstrates selective mid-dermal elastin loss while preserving perifollicular elastic fibers, which is a key histological distinction from PE.[10] Papular elastorrhexis presents as nonfollicular, firm papules characterized by reticular dermal elastin fragmentation without follicular involvement, contrasting with PE's peri-infundibular elastolysis.[11] White fibrous papulosis manifests as neck-localized, nonfollicular papules accompanied by papillary dermal fibrosis, differing both anatomically and histologically from PE.[12,13] Clear cell papulosis, though rare, should be considered in the differential spectrum due to its distinct histiocytic infiltration pattern.[14] Lastly, pseudoxanthoma elasticum-like dermal papillary elastolysis (PXE-like elastolysis) shares phenotypic similarities with PXE but exhibits selective papillary dermal elastolysis without systemic involvement, requiring immunohistochemical analysis for definitive distinction.[6,15] Accurate differentiation hinges on correlating lesional morphology, anatomical distribution, and histopathological elastin staining patterns.

No established therapeutic protocol currently exists for PE, necessitating a tailored approach centered on symptom mitigation and preventive strategies. Therapeutic strategies emphasize mechanical trauma mitigation through cessation of predisposing practices such as shaving and elimination of exacerbating agents like topical corticosteroids. Pharmacological interventions primarily involve topical retinoids (eg, tretinoin) to modulate epidermal turnover and antibiotics (eg, clindamycin), targeting both textural improvement and potential secondary infection prevention, though evidence remains limited to small observational studies.[9] Critical to management is patient education regarding PE's benign, non-progressive course, which effectively addresses psychological morbidity associated with chronic dermatoses while reducing unnecessary interventions. The natural history of PE typically involves persistent lesions with minimal progression, though rare cases of spontaneous resolution have been documented, suggesting variable biological behavior.[8]

PE represents a distinct entity within the spectrum of elastolytic disorders. While its pathogenesis remains elusive, emerging associations with autoimmune diseases and mechanical trauma highlight its multifactorial nature. Histopathology remains diagnostic gold standard, differentiating PE from mimics like MDE or anetoderma. Future research should explore genetic predispositions and targeted therapies to address this cosmetically concerning yet benign condition.

Ethics and Consent Statement

The written informed consent was obtained from the patient for the publication of the case details and images. No further institutional approval was required.

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Disclosure

The authors report no conflicts of interest in this work.

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