DOI: 10.7860/JCDR/2026/84975.22569

Perforating Folliculitis: A Transepidermal Elimination Disorder

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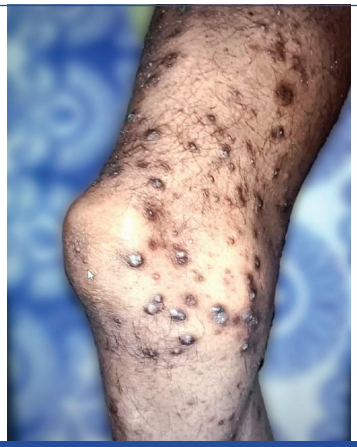
Keywords: Collagen extrusion, Follicular plugging, Perforating dermatoses, Pruritic papules

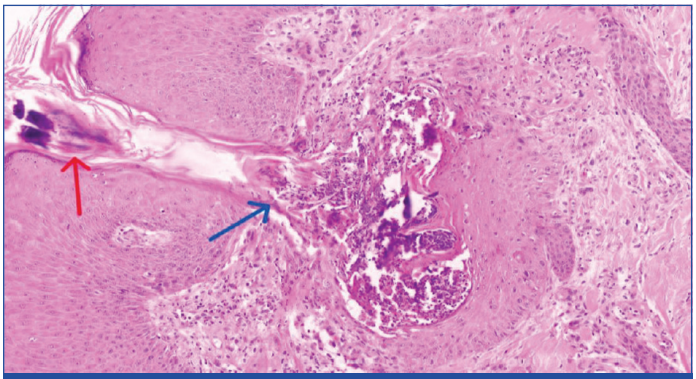
A 62-year-old male presented with complaints of dark, raised, pruritic lesions over bilateral upper and lower limbs for two months. The lesions initially started on the legs and were associated with pruritus and gradually progressed to involve the entire upper and lower limbs. The patient gave a 10-year history of uncontrolled diabetes, treated with oral hypoglycaemics and a 5-year history of chronic kidney disease. On examination, the patient exhibited multiple hyperpigmented papules and nodules over the bilateral upper and lower limbs [Table/Fig-1].

Based on the clinical presentation, the differential diagnoses considered were perforating folliculitis, Kyrle's disease, acquired perforating collagenosis, prurigo nodularis and hypertrophic lichen planus. Upon investigating, the patient had a fasting blood glucose level of 320 mg/dL and a creatinine value of 2.5 mg/dL.

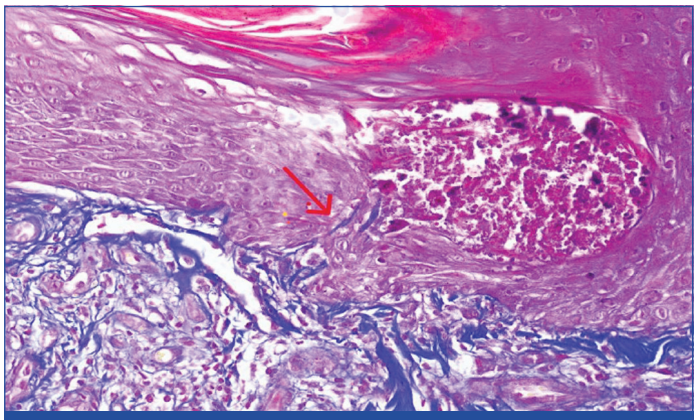
Histopathological examination revealed dilated hair follicles filled with basophilic debris, collagen, keratin and neutrophils. Transepithelial extrusion of collagen into hair follicles, along with perifollicular inflammatory infiltrate composed of lymphocytes and neutrophils, was observed [Table/Fig-2]. Masson's trichrome stain confirmed the presence of extruded collagen, supporting the diagnosis of perforating folliculitis [Table/Fig-3].



[Table/Fig-1]: Multiple hyperpigmented papules and nodules over the right lower limb.



[Table/Fig-2]: Haematoxylin and Eosin (H&E) stained section of skin under low power (10x), showing parakeratotic follicular plugging (red arrow), degenerated keratin, inflammatory infiltrate and degenerated hair follicle (blue arrow).



[Table/Fig-3]: Masson's trichrome-stained section of skin under high power (40x), showing collagen extruding into the perforated and dilated hair follicle (red arrow).

Management included optimisation of elevated creatinine levels and improved glycaemic control, alongside a 7-day course of tablet Doxycycline, tablet Bilastine and twice daily application of keratolytic-based emollient containing urea and lactic acid. The patient was subsequently lost to follow-up and further clinical progression could not be assessed.

DISCUSSION

Perforating dermatoses are a group of disorders marked by the unusual process of trans-epidermal elimination, where the skin expels components normally found in the dermis. In 1989, Rapini RP et al., described multiple cases of perforating dermatosis in individuals with diabetes mellitus or renal disease and proposed the term Acquired Perforating Dermatosis (APD) [1]. APD encompasses four entities: Kyrle's disease, perforating folliculitis, elastosis perforans serpiginosa, and reactive perforating collagenosis. A comparative overview of the four entities is presented in [Table/Fig-4] [2,3].

Entity	Main clinical feature	Eliminated material
Kyrle's disease	Umbilicated papules with keratin plugs	Keratin and debris
Perforating folliculitis	Follicular, umbilicated papules with central keratotic plugs	Keratin and degenerated collagen via follicular canal
Reactive perforating collagenosis	Umbilicated papules with keratotic crust; Koebnerisation common	Collagen
Elastosis perforans serpiginosa	Hyperkeratotic papules arranged in a serpiginous pattern	Elastin

[Table/Fig-4]: Comparative overview of Acquired Perforating Dermatosis (APD) entities.

Perforating folliculitis is identified by extrusion of follicular contents with or without collagen fibres or elastin fibres [2]. This frequently occurs alongside conditions like diabetes, chronic renal failure, vitamin A deficiency and HIV infection. Proposed mechanisms include premature keratinisation, altered connective tissue or foreign material in the superficial dermis triggering transepidermal

elimination and involvement of fibronectin. A primary irritant, either chemical or physical, even chronic rubbing, could result in the mechanical disruption of the follicular wall by the hair shaft [3].

Histologically, a dilated follicular infundibulum packed with a compact ortho and parakeratotic plug containing a retained hair, degenerated collagen fibres, refractile eosinophilically altered elastic fibres, basophilic debris, inflammatory infiltrate, extracellular matrix and a perifollicular infiltrate consisting of lymphocytes, histiocytes and neutrophils is the characteristic feature [4]. Areas adjacent to perforation sites show altered collagen and refractile elastic fibres. Masson's trichrome stain aids in highlighting extruded collagen and in differentiating other disorders from perforating folliculitis, where collagen is eliminated through specific hair follicles [5].

Management of perforating disorders centres on correcting the underlying systemic disease. Reported treatments range from topical and intralesional corticosteroids, oral antihistamines, and topical retinoids [6]. Other therapies include Narrowband Ultraviolet B (NB-UVB), Psoralen + Ultraviolet A (PUVA), oral retinoids, allopurinol, doxycycline and methotrexate [7]. The main therapeutic goal is to address pruritus, as improvement in itching often parallels clinical resolution.

CONCLUSION(S)

This case spotlights transepidermal collagen elimination as a defining histopathological event in perforating folliculitis, reflecting a process in which damaged dermal collagen is channeled through a perforating epidermis and ultimately eliminated as keratinous debris at the skin surface.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Nov 21, 2025
- Manual Googling: Dec 13, 2025
- iThenticate Software: Dec 16, 2025 (3%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Oct 18, 2025**
Date of Peer Review: **Nov 24, 2025**
Date of Acceptance: **Dec 18, 2025**
Date of Publishing: **Mar 01, 2026**