

'Butterfly' Plaque of Antecubital Fossa: A Diagnostic Dilemma

PRIYADARSHAN ANAND JATEGAONKAR¹, SUDEEP PRADEEP YADAV²



Keywords: Lichen planus, Pseudoepitheliomatous hyperplasia, Skin

Dear Editor,

The 20-year-old female, a farmer with no medical history or current medications, presented with a large discoid soft-tissue mass arising from the left antecubital fossa since childhood. It started as a pea-nut-sized verrucous swelling and slowly enlarged over the last one-and-a-half decades. She experienced occasional local itching but never had pain. There were no ulcerations, discharge, or similar lesions elsewhere. Her past and family histories were non contributory, and she could not recall any local trauma in the past. Primarily, inadequate elbow flexion and cosmetic disfigurement compelled her to seek medical consultation.

While her general and systemic examinations were unremarkable, the local examination revealed an $8\times6\times2$ cm, non tender, well-demarcated, thick, firm, encrusted plaque at the left antecubital fossa. With near-symmetrical morphology, it had a flat, dry, pigmented, pinkish-brown surface abundant with whitish striae, resembling a colourful butterfly [Table/Fig-1]. It was not fixed to the underlying structures, and the surrounding skin showed no inflammation or satellite nodules. Notably, elbow flexion was restricted by about 25°, and she had no loco-regional lymphadenopathy.



[Table/Fig-1]: Pseudoepitheliomatous Hyperplasia (PEH) at the antecubital fossa. Note- the flat-topped, polygonal, shiny, pinkish-brown, thick soft-tissue mass with sharp borders simulating a butterfly sitting on a flower.

Her routine haemogram was normal. Subsequently, a deep punch biopsy revealed florid epidermal hyperplasia with brisk dermal extensions, hyperkeratosis, and a dense inflammatory infiltrate; however, there was no band-like lymphocytic infiltrate. There were no hyalinising collagen bundles, granulomas, keratinocyte atypia, mitosis, or pleomorphism.

The probable differential diagnosis were Spontaneous Flexural Keloid (SFK), Cutaneous Hypertrophic Lichen Planus (CHLP), verrucous type Cutaneous Squamous Cell Carcinoma (CSCC), and Pseudoepitheliomatous Hyperplasia (PEH) [Table/Fig-2] [1-5]. However, the clinicopathological findings were consistent with PEH. Complete excision of the lesion followed by detailed histological analysis confirmed the diagnosis. During regular follow-ups over the last six months, she has had no recurrence.

Feature	SFK [4]	CHLP [5]	CSCC [1,3]	PEH [1,2]		
History						
Age (years)	10-30	30-50	50-70	50-80		
Ethnicity	Africans	Any	Whites	Any		
Duration	++++	+++	++	++++		
Spontaneity	++++	++	+++	+++		
Growth	+	+++	++++	++		
Intractable pruritus	_	++++ (classical)	_	_		
Pain	_	+++	_	_		
Discharge	_	_	+/	_		
Past trauma	++	_	_	+/		
Examination						
Common site	Upper trunk	Shin	Sun-exposed	Flexural		
Size	Varies	Centimeters	Centimeters	Giant		
Shape	Oval	Polygonal (classical)	Warty	Varies		
Margins	Well-defined	Sharply-defined	Irregular	Varies		
Beyond margins	++++	_	_	_		
Symmetry	+	++++ (classical)	_	-		
Surface	Flat	Flat (classical)	Uneven	Flat		
Colour	Brown-black	Purplish (classical)	Reddish	Pinkish		
Shininess	_	++++	_	+/		
Encrustation	_	+/	+++	+++		
Wickam's striae	_	++++	_	_		
Ulcerations	_	++	++++	+		
Satellite nodules	_	_	++++	_		
Regional lymph nodes	_	_	++++	-		
Oral lesions	_	+++ (50%)	_	_		
Histology						
Inflammatory cells	Mixed	Mainly lymphocytes	+/	Mixed		
Inflammatory density	++	++++	++	++++		
Dermal pseudo- invasion	_	_	_	++++		
Band-like lymphocytes	_	++++ (classical)	_	_		
Basement membrane	Intact	Vacuolar degeneration	Infiltration	Distorted		
Epidermal hyperplasia	-	+++	++	++++		
Hyperkeratosis	++	++++	++	++		
Immature collagen	++++	-	-	-		
Hyalinising collagen	++++	-	-	_		

Keratin pearls	_	_	++++	_		
Cellular atypia / mitosis	_	_	++++	_		
Perineural infiltration	_	_	++++	_		
General						
Spontaneous regression	_	++++ (2 years)	_	+		
Recurrence	++++	+/	++	+/		
Subcutaneous infiltration	_	_	+++	_		
Close differentials	+	+++	+++	++++		
Premalignant	_	+++ (1%)	++++	_		
Autoimmune aetiology	_	++++	_	_		

[Table/Fig-2]: Differential diagnosis of pseudoepithelial hyperplasia.

(— to ++++: Grades of association; SFK: Spontaneous flexural keloid; CHLP: Cutaneous hypertrophic lichen planus; CSCC: Cutaneous squamous-cell carcinoma; PEH: Pseudoepitheliomatous hyperplasia)

PEH is a rare benign reactive dermatosis characterised by exuberant hyperplasia of the epidermis and adnexal epithelium, extreme-degree acanthosis, along with a plethora of inflammatory cells in the reticular dermis [1,2]. Though the exact aetiology is unclear, trauma, infective, or inflammatory processes seem to play vital roles [1,2]. Because of its characteristic verrucous growth pattern and marked clinicopathological semblance, it is difficult to differentiate PEH from major ailments like CSCC, CHLP, and SFK [1-5]. CSCC may even arise within a long-standing PEH [1]. However, a detailed history, a keen eye for morphology as well as histology, and healthy collaboration with the pathologist are pillars for an accurate diagnosis [1,2]. Distinguishing PEH from CSCC is of utmost importance [1,2]. For this purpose, deep punch biopsy including portions of the dermis is the gold standard [1,2]. Moreover, immunohistochemistry demonstrating less p53 immunostaining and lower expression of the C15orf48 gene than the KRT9 gene robustly distinguishes PEH from CSCC [2].

Treating PEH satisfactorily is always a major challenge [2]. Conservative modalities like topical photodynamic therapy and intralesional corticosteroids are the front-line therapies with acceptable results; however, complete surgical excision with plastic reconstruction remains the treatment of choice, particularly for large-sized PEH like this case [2].

In summary, PEH should be considered as one of the most probable differentials while managing a large flat-topped antecubital fossa growth. Judicious use of histologic assessment avoids overdiagnosing it as CSCC or underdiagnosing it as CHLP, saving the patient from potentially life-threatening treatment modalities.

Acknowledgement

Authors would like to acknowledge and express their gratitude to Dr. Gayatri Khatri Yadav; MD (Dermatology, Venereology & Leprology), DNB (Dermatology), FRGUHS (Cosmetology), MNAMS, MBBS; Consultant Dermatologist and Cosmetologist; Khatri Clinic Pune, for providing valuable suggestions and guidance during the preparation of the manuscript.

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PARTICULARS OF CONTRIBUTORS:

- 1. Professor, Department of General Surgery, Jawaharlal Nehru Medical College, Sawangi (Meghe), Wardha, Maharashtra, India.
- 2. Consultant, Department of Plastic and Reconstructive Surgeon, Khatri Clinic, Pune, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Priyadarshan Anand Jategaonkar,

403, Royal Heritage, Shrinivas Colony, Ramnagar, Wardha-442001, Maharashtra, India. E-mail: jategaonkarpa@gmail.com

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

PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Jan 05, 2024
- Manual Googling: Feb 21, 2024
- iThenticate Software: Feb 26, 2024 (3%)

ETYMOLOGY: Author Origin

EMENDATIONS: 5

Date of Submission: Jan 04, 2024
Date of Peer Review: Feb 19, 2024
Date of Acceptance: Feb 27, 2024
Date of Publishing: May 01, 2024