

EDITORIAL

Annular Keloid Mimicking Granuloma Annulare: A Diagnostic Challenge

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Keloid, a dermal tumour, is a benign fibroproliferative disorder characterized by abnormal deposition of collagen within a wound, predominantly type 1 collagen fibers. It presents as firm to rubbery nodules, which may be pruritic or painful. It usually spreads beyond the margin of original wound and commonly recurs following excision. Common sites includes chest, shoulders, upper back, nape of neck, and posterior aspect of the ear lobule.¹

A 55 year old female presented to the outpatient department of dermatology at tertiary care centre with chief complaints of single elevated lesion over right breast associated with itching and pain. Patient had history of unknown surgical intervention before development of lesion. No significant complaints in family. Cutaneous examination revealed single well defined annular erythematous plaque of size approximately 8x10 cm surrounded by erythematous border with central atrophy and hypo pigmentation over right breast. (Figure 1) Dermoscopy done with Dermalite DL 4 under 100 magnification revealed diffuse erythema with globular

vessels, yellowish to whitish thick scales, and white structureless areas. (Figure 2)



Figure 1: Single well defined erythematous plaque with central atrophy and hypo pigmentation over right breast. (Dermoscopy 100X magnification)

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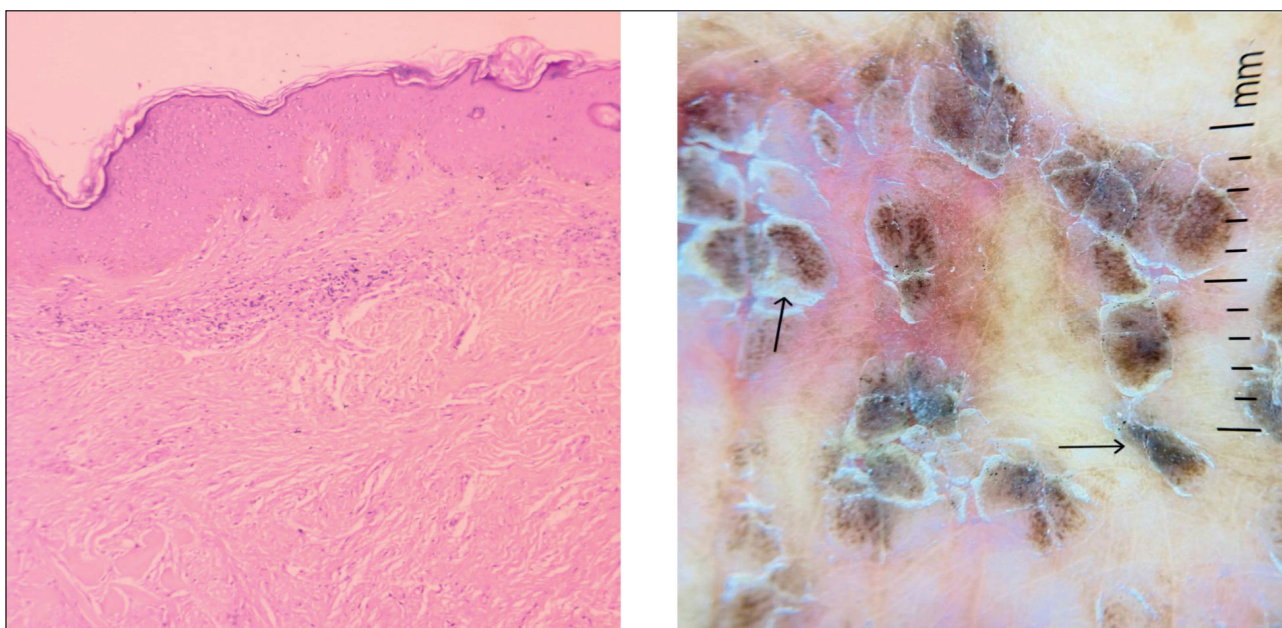


Figure 2 a&b: Dermoscopy done with DermLite DL 4 under 100x magnification revealed diffuse erythema with few linear vessels, yellowish to whitish thick scales, and white structureless areas (black arrow) (Intralesional triamcinolone 10mg)

Punch biopsy taken from edge of lesion with differentials of keloid and granuloma annulare showed perivascular lymphoplasmacytic infiltrates in the reticular and papillary dermis. The reticular dermis showed long, broad, haphazard proliferation of collagen bundles with glassy eosinophilic appearance with increased fibroblastic proliferation and destroyed dermal adnexa. (Figure 3) As per the morphology, dermatoscopy and histopathology, case was labelled as annular keloid.

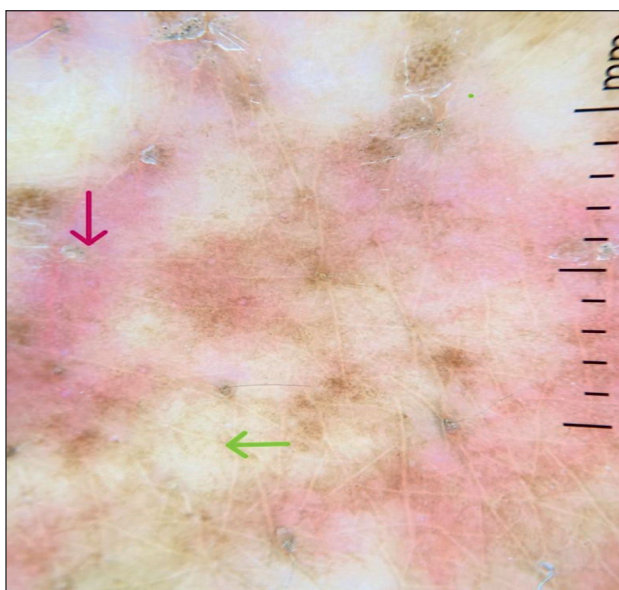


Figure 3: Perivascular lymphoplasmacytic infiltrates in the reticular and papillary dermis. Long, broad, haphazard proliferation of collagen bundles with glassy eosinophilic appearance with increased fibroblastic proliferation and destroyed dermal adnexa in reticular dermis (H&E stain 10x)

Patient was treated with intralesional triamcinolone injection at 3 week interval and cryotherapy weekly. The skin lesions started improving in terms of reduced erythema and regression of elevated border after 4 weeks of treatment.

Keloids are pedunculated to broad-based plaques that rarely regress spontaneously. Its variants includes nodular, flat butterfly, burned out and earlobe keloid. Unusual keloid can present as annular plaques with central clearing, which needs to be differentiated from granuloma annulare (GA), annular sarcoidosis, and nodular scleroderma. They are prone to occur in areas of high skin tension or repeated trauma. They are seen in darker skin types and appears during puberty and pregnancy. The pathogenesis of keloid scar is complex involving both genetic and environmental factors.²

The histologic hallmark of keloid is increased whorls of thickened, hyalinized collagen bundles widely known as keloidal collagen. In its absence; thickened or flattened epidermis; a tongue-like advancing edge in the dermis; haphazard, thick, hyalinized collagen bundles in dermis; loss of the papillary-reticular boundary; increased dermal cellularity; or signs of inflammation are diagnostic.

GA is a benign inflammatory dermatosis characterized clinically by erythematous plaques or papules arranged in an annular configuration on the upper extremities. Clinical

variants includes localized, generalized, subcutaneous, perforating and arcuate dermal erythema. It is caused by a delayed-type hypersensitivity reaction and histologically, is characterized by lymphohistiocytic infiltrates, mucin deposition, and either an interstitial or palisading pattern of histiocytes with necrobiotic collagen forming granulomas.³

In a study by Yoo MG *et al.*, the most common dermoscopic vascular structures in keloids were arborizing vessels followed by linear, irregular and comma shaped vessels.⁴ Dermoscopy of GA shows blurry vessels having variable appearance (dotted, linear-irregular, and branching) over pinkish-reddish background. Additional findings seen can be rosettes, crystalline structures, and white scaling.³ Our case showed diffuse erythema with globular vessels, yellowish to whitish thick scales, and white structureless areas.

GA occurring on or around scars has never been reported previously. There have been reports of GA developing within herpes zoster scars, where it is thought to represent an atypical delayed hypersensitivity immune reactions to the viral antigen(s).³ An association between GA and keloids is rarely reported. Reported literature showed 3 patients with lesions that clinically appeared as GA but histologically were determined to be keloid; It has been hypothesized that there is a “keloid-like stage” in the involution of GA. A more recent case in the literature described GA arising years

after a keloid scar, in which the keloid was secondary to a lightning strike while wearing a gold chain.⁵ Several modalities used for the treatment of keloids include topical and intralesional steroid, cryotherapy, surgical excision, radiotherapy and lasers.

Case is reported as an unusual morphology highlighting the importance of development of GA in long time according to literature which needs vigilant follow up.

Conflicting Interest: NIL

Acknowledgement: NIL

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