Raimer's Bands: Case Report with a Review of Solar Elastosis

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Raimer's solar elastotic bands of the forearm are a rarely described clinical variant of severe solar elastosis. We report a case of Raimer's bands on the forearm of a 43-year-old woman. Light microscopy demonstrated solar elastosis and electron microscopy revealed activated fibroblasts appearing to secrete the elastotic material. The clinical variants of solar elastosis are outlined and the possible pathogenesis of the elastotic material is discussed. Key words: actinic elastosis; fibroblast secretion.

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Solar elastosis represents a spectrum of clinically recognisable forms with a similar underlying histology, demonstrating elastotic material in the dermis (1). Primary forms result from chronic sun exposure (2) but secondary forms associated with heat (3) and chemicals (4) have been described. Primary solar elastosis in its simple form is characterized by yellow, inelastic, coarsely wrinkled and diffusely thickened skin (2). Several special forms have been recognised (5–18) (Table I), some having been described in Austalia (1, 10, 12, 18) where solar elastosis is commonly seen amongst the fair-skinned population, particularly from middle-age onwards.

Raimer et al. (11) in 1986 described 3 patients with soft,

flesh-coloured to yellowish papules and nodules that tended to merge in a cord-like band extending from areas of severe actinic damage to less damaged areas of the forearm. The histology revealed a dermis containing haemotoxylinophilic homogeneous material with clefting as well as fibroblasts and inflammatory cells. Electron microscopy showed abundant elastotic fibres composed of granular matrix and electron-dense condensations. Closely apposed to the elastotic fibres were fibroblasts rich in rough endoplasmic reticulum and histiocytes with multiple phagoliposomes.

We report a case of Raimer's plaques in a middle-aged woman and discuss the possible pathogenesis of solar elastosis in the light of the ultrastructural features of this case.

CASE REPORT

A 43-year-old Caucasian housewife of skin phototype II presented with a 2-month history of a linear lesion on the dorsum of her right forearm. A similar lesion was beginning on the dorsum of her left forearm. She had a history of atopic eczema since infancy that was controlled with topical corticosteroids, apart from a single admission with generalised erythroderma 6 years previously that necessitated oral corticosteroids. Her eczema typically flared in the summer months and this was complicated by poor compliance with topical sunscreens.

Slightly raised yellowish bands were evident on the dorsum of both forearms (Fig. 1). These extended along the interface of areas of severe and less severe actinic damage. Purpura had been noted on one occasion.

Table I. Specialized forms of primary solar elastosis, the main sites affected and distinguishing clinical features

Clinical form		Main site	Main features	Ref.
1.	Cutis rhomboidalis nuchae	posterior neck	furrows	
2.a) b)	Nodular elastoidosis Actinic comedonal plaque	a) periorbital; malar b) arms	a), b) plaques studded with cysts and comedones	(5, 6) (7)
3.a) b)	Elastoma Nasal elastotic plaque	a) neck; anterior chest b) nasal dorsum	discrete focal plaques	(8)
4.	Elastotic nodules of the ear	antihelix (helix)	discrete pale elevations	(9, 10)
5.a) b)	Collagenous plaques of the hand Keratoelastoidalis marginalis	a) palm-dorsum junction b) radial index finger; lateral hand	a), b) discrete or confluent groups of hyperkeratotic papules	(11) (12)
6.a) b)	Adult onset colloid milia Nodules & plaques of colloid degeneration	a), b) exposed sites	a) yellowish or translucent papules b) nodules of variable colour & consistency within erythematous plaques	(13) (14–17)
7.	Actinic granuloma	exposed sites	red papules evolving into annular lesions with atrophic centres	(18)
8.	Solar elastotic bands	forearm between actinically damaged and less damaged areas	flesh-coloured to yellowish papules & nodules merging into cordlike band	(11)
9.	Unilateral facial plaque	cheek	indurated plaque; post-palpatory erythema	(1)



Fig. 1. Raised yellowish bands on the dorsum of the right forearm extending along the interface between areas of severe and less severe actinic damage (indicated by *arrows*).

Histology of a skin biopsy specimen revealed florid solar elastosis in the dermis and epidermal atrophy with effacement of the rete ridges (Fig. 2).

Electron microscopy showed moderately electron-dense masses in the mid-dermis. These had an amorphous and filamentous component at higher magnification. Fibroblasts were commonly seen and displayed abundant rough endoplasmic reticulum, mitochondria and a prominent Golgi apparatus. On one view small secretory-like vesicles were evident within a fibroblast, some of these opening along the cell membrane where it apposed the elastotic material (Fig. 3).

DISCUSSION

Solar elastotic bands of the forearm have only been described once before in the literature to our knowledge. The younger age of this patient in comparison to the 3 described by Raimer et al. (11) (63 to 83 years) may relate to her infrequent use of topical sunscreens, in addition to her fair skin and life-long residency in Australia. Epidermal atrophy, as seen in our patient and those of Raimer et al. (11), is indicative of end-stage photoageing (19), in keeping with the concept that this condition is a form of severe solar elastosis. Although Raimer et al. (11) described senile purpura in all their cases, prolonged topical corticosteroid use may have contributed to the purpura noted in our younger patient.

The pathogenesis of elastotic material in the dermis in solar elastosis remains controversal. Elastotic fibres are now known to originate from elastic fibres rather than collagen due to their staining with anti-elastin antibodies and disappearance with elastase but resistance to collagenase, in addition to the high desmosine content (2). It has been variously suggested that they may arise from elastic fibre degradation (2, 20) or de novo synthesis by sun-damaged fibroblasts (21, 22), or from both processes (23). There are two aspects of our case that favour the secretory origin theory: firstly, the presence of many activated fibroblasts and paucity of macrophages on light microscopy, and secondly, the electron microscopic picture of multiple secretory-like vesicles opening along the fibroblast membrane where it apposes the elastotic material.

In conclusion, solar elastosis comprises a spectrum of clinical forms that may arise due to the secretion of abnormal material

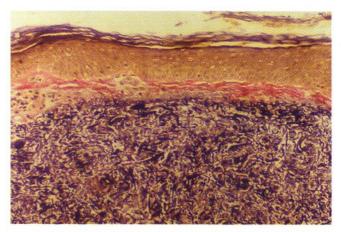


Fig. 2. Light microscopy showing florid solar elastosis in the dermis and epidermal atrophy with effacement of rete ridges (Verhoeff-van Gieson; ×62.5).

into the dermis by sun-damaged fibroblasts. Raimer's bands of the forearm is a rare clinical form of severe solar elastosis.

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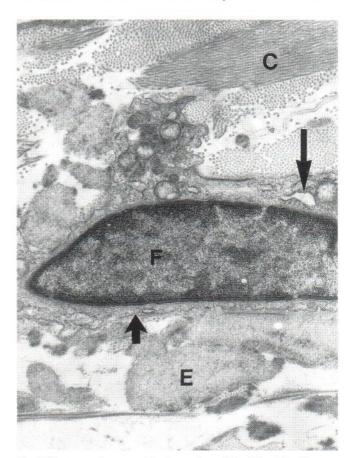


Fig. 3. Electron microscopy showing activated fibroblast (F) adjacent to elastotic material (E). Cytoplasm shows dilated rough endoplasmic reticulum $(large\ arrow)$ and numerous secretory-like vesicles, some fused with the plasma membrane and containing amorphous material $(small\ arrow)$. Normal collagen fibre also shown (C) $(\times 12,500)$.

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