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Commentary

The Cosmic Resilience-Solar Elastosis

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Preface

Solar elastosis occurs due to chronic, persistent cutaneous damage arising on account of ultraviolet radiation affecting ageing cutaneous surfaces. Solar elastosis exhibits an accumulation of anomalous elastin or elastic tissue within cutaneous dermal layer or ocular conjunctiva. The exceptional solar elastosis engendered by actinic-induced cutaneous damage exhibits thickened, wrinkled, dry, furrowed, bumpy and yellowish cutaneous surfaces. Solar elastosis is also designated as actinic elastosis or elastosis senilis [1,2].

Preliminary lesions of solar elastosis demonstrate dermal proliferation of elastic fibres. Gradually, papillary and reticular dermal collagen is replaced by thick, curled, basophilic fibres configuring tangled masses. Thus, solar elastosis may be contemplated as a biological indicator and a protective, compensatory reaction to proportionate, ultraviolet-induced cutaneous injury. Confirmation by cogent histological assessment is mandated.

Disease characteristics

Solar elastosis emerges as a feature of photo-ageing and occurs in individuals with extended exposure to sun. The lesion initially appears beyond > 40 years. Elderly subjects depicting solar elastosis are associated with enhanced possible emergence of cutaneous malignant melanoma [3,4].

Although devoid of racial predilection, incriminated Caucasian population predominantly exhibits a yellow cutaneous hue. Immunocompromised individuals or Caucasians with minimally tinted irises and blonde hair subjected to excessive exposure to sun or pursuing an outdoor occupation depict a predilection for solar elastosis, rapidly emerging freckles or sunburn [3,4].

Accumulation and denaturation of elastic tissue ensues on account of cumulative, prolonged and excessive actinic exposure which induces photo-ageing. Extended exposure to sunlight engenders solar elastosis wherein no cutaneous subtype is spared [3,4].

Solar elastosis engendered by smoking is indicative of premature cutaneous ageing. Tobacco consumption alters collagen production with elevated configuration of tropo-elastin and matrix metalloproteinases (MMPs) [3,4].

In fact, smoking enhances production of tropo-elastin fibres along with degradative enzymes constituted by matrix metalloproteinases (MMPs). Consequently, matrix proteins are degraded with dermal accumulation of anomalous elastic substance. Alteration within elastic fibres may extend towards deep-seated reticular dermis [3,4].

Alternatively, singular actinic-induced damage in the absence of tobacco consumption is associated with alterations of elastic fibres predominantly restricted to superficial papillary dermis [3,4].

Of obscure aetiology, configuration and accumulation of elastotic substance within the dermis is posited to arise due to actinic stimulation of fibroblasts, a process which stimulates synthesis of the substance. Besides, elastotic substance emerges as a degradation product of collagen and elastin [3,4].

Dermal elastotic substance is posited to arise from degradation of collagen and elastic fibres, fibroblasts activated due to actinic induced injury with syntheses of fresh elastic tissue and an anomalous, synthetic, degradative tissue process [3,4].

Clinical elucidation

Solar elastosis is associated with minimal clinical symptoms and may exemplify a gradually enlarging, reddish, bumpy cutaneous patch with pruritus or burning sensation. Solar elastosis initially incriminates sun-exposed cutaneous zones as the face, lips, ears, neck, forearms or hands. Upon examination, a reddish or yellow papule of variable magnitude is observed [4,5].

Accumulation of deranged elastic tissue ensues within actinic-sensitive upper and mid-dermis. Photo-ageing is associated with cutaneous damage confined to superficial papillary dermis [4,5].

Tobacco smoke induces denaturation of deep-seated reticular dermis. Tropo-elastin and matrix metalloproteinases (MMPs) denature the matrix with consequent, disorderly configuration of elastin fibres within cutaneous layers [4,5].

Solar elastotic syndrome manifests as thickened, dry, coarse, wrinkled cutaneous soft tissue associated with decimated skin tone [4,5].

The commonest clinical variant appears as thickened, intensely fissured cutaneous surface discerned upon dorsum of neck subjected to chronic actinic-induced damage which is designated as cutis rhomboidalis nuchae and configures a segment of cutaneous photo-ageing. Cutis rhomboidalis nuchae exhibits thickened, yellow, leathery cutaneous tissue confined to posterolateral neck surface [4,5].

Papular elastosis as a terminology was initially adopted by Kwittken wherein asymptomatic, solitary or multiple, shiny, smooth, firm, variably hued papules of magnitude up to 10 millimetres are associated with severe elastosis within the papillary cutis [6].

Elastotic nodules confined to ear characteristically display singular or multiple, bilateral, firm or hard, pale nodules situated upon antihelix or helix of the pinna. Upon histology, irregular, coarse, elastotic substance and clumped, thick fibres are admixed with foci of prominent solar elastosis and are appropriately appreciated with elastic-Van Gieson stain. Clinical segregation is required from basal cell carcinoma, amyloidosis, gouty tophi and chondrodermatitis nodularis helicis. Collagenous and elastotic plaques of the hand appear as a gradually progressive disorder discerned in elderly males. Lesions appear as waxy, linear plaques and are situated upon the junction of dorsal and palmar cutaneous surface of the hand [5,7].

Upon histology, amorphous, basophilic nodules of elastotic tissue and thickened, fragmented, calcified, elastic fibres are admixed within the upper and mid-dermis. Thickened, haphazard collagen bundles appear perpendicular to the cutaneous surface [5,7].

Adult colloid milium is a solar elastotic dermatosis which resembles papular solar elastosis. Upon microscopy, homogenous, amorphous, mildly eosinophilic substance is admixed with cleft-like spaces confined to upper and mid-dermis [5,7].

Of obscure aetiology, dermal colloid deposition ensues due to actinic- induced degeneration of collagen and elastic fibres. Besides, the elastotic substance may be engendered due to actinically injured fibroblasts [5,7].

Histological elucidation

Solar elastosis can be appropriately discerned by cogent tissue sampling of incriminated zone. Decimated staining with eosin within upper dermal layers is associated with a basophilic hue and significant disorganization of dermal elastin fibres [7,8].

Solar elastosis is indicative of pertinent histopathological alterations with degeneration of dermal elastic tissue which ensues within photo-damaged cutaneous surfaces. Predominantly, solar elastosis exhibits basophilic degeneration of dermal elastotic fibres with a narrow band of normal, horizontally disseminated collagen or Grenz zone segregating the dermis from epidermis. Elastosis is proportionate to cumulative exposure of ultraviolet radiation to cutaneous surfaces. Verhoeff's stain can be employed to stain the elastotic fibres black [7,8].

Upon microscopy, decimated eosin staining is observed within superficial dermis along with accumulation of irregular, dense elastic fibres. The elastic fibres are degraded to configure tangled tropoelastin and fibrillin articulations [7,8].

Foci of solar elastosis may be admixed with patchy, chronic, non specific lymphocytic and plasma cell inflammatory infiltrate. Epidermal acanthosis may accompany the lesions [7,8].

Additionally, Favre-Rachouchot syndrome is associated with elevated elastosis of upper and mid-dermis configuring thickened, tortuous elastic tissue fibres [7,8].

Differential diagnosis

Papules of solar elastosis require segregation from lesions such as basal cell carcinoma, sebaceous hyperplasia, molluscum contagiosum or actinic keratosis [3,4].

Solar elastosis upon the hands requires demarcation from keratoelastosis marginalis which is an acquired marginal keratoderma incriminating lateral aspect of index finger or thumb [3,4].

Therapeutic options

Solar elastosis can be minimized with circumvention of sun exposure which is an efficacious prevention strategy. Adoption of appropriate sun protection with broad spectrum sunscreens and suitable clothing is recommended. Also, discontinuation of smoking and preventing exposure to passive smoking appears advantageous. Antecedent disease prevention and therapy is efficacious and recommended [9,10].

Treatment options of variable efficacy applicable for alleviating modifications of photo-aged cutaneous surfaces are comprised of dermabrasion, topical retinoic acid, topical oestrogens, resurfacing with carbon dioxide laser, dermal injections of hyaluronic acid, imiquimod or tacrolimus ointment [9,10].

Cosmetic outcomes of elastotic cutaneous surfaces can be meliorated with employment of laser ablation, topical imiquimod, injectable botulinum toxin which decimates nervous activity with cutaneous relaxation or dermal fillers which permeate cutaneous contours and decrease depth of cutaneous furrows [9,10].

Additionally, ablative and non-ablative laser therapy, dermal fillers and injectable neurotoxins as botulinum toxin can be adopted in order to enhance aesthetic outcomes of solar elastosis therapy [9,10].

It is posited that imiquimod therapy may ameliorate morphological countenance of solar elastosis in accompaniment with elastotic dermal modifications [9,10].

Certain lesions may progress into cutaneous squamous cell carcinoma. Thus, annual monitoring of solar elastosis is mandated [9,10].

Organ transplant recipients on immunosuppressive therapy exhibit possibility of actinic damage and emergence of diverse cutaneous malignancies. Besides, deceleration of cutaneous carcinogenesis may ensue with discontinuation of immune-suppressive therapy [9,10].

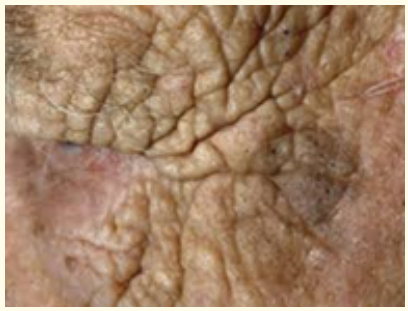

Figure 1: *Solar elastosis exhibiting a wrinkled, corrugated, bumpy, dry, yellowish cutaneous patches [11].*

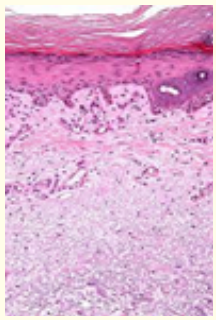

Figure 2: *Solar elastosis exemplifying degeneration of dermal elastic layer with a specific grenz zone and epidermal acanthosis [12].*

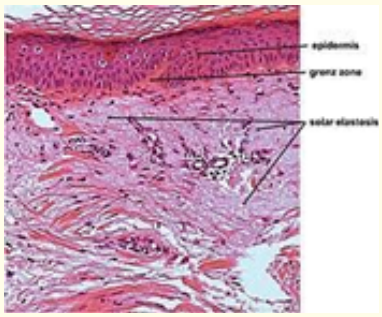

Figure 3: *Solar elastosis enunciating elastotic degeneration separated by a definitive grenz zone and acanthosis of superimposed epidermis [12].*

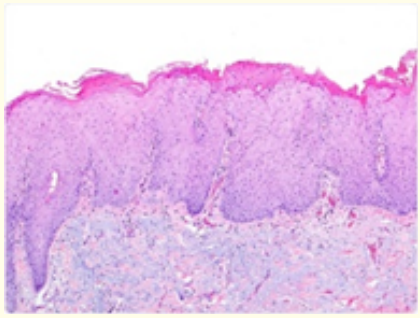

Figure 4: *Solar elastosis depicting significant acanthosis, denaturation of dermal elastic layer, a normal layer of collagen and mild, chronic inflammatory infiltrate [13].*

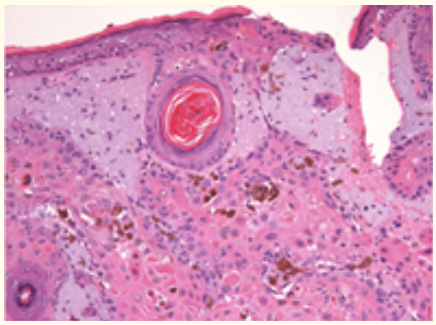

Figure 5: *Solar elastosis displaying denatured dermal elastic layer, horizontal dissemination of collagen fibres, numerous congested vascular articulations and an acanthotic epidermal layer [14].*

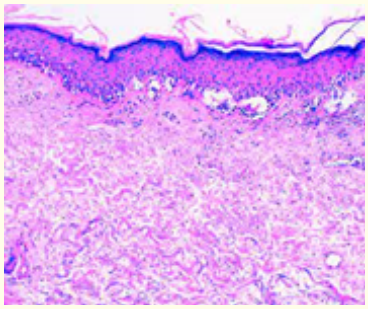

Figure 6: *Solar elastosis demonstrating degeneration of dermal elastic layer, distinct grenz zone and acanthosis of superimposed epidermis with hyperkeratosis [15].*

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11. Image 1 Courtesy: Dermnet NZ.
12. Image 2 and 3 Courtesy: Wikipedia.
13. Image 4 Courtesy: News medical.
14. Image 5 Courtesy: Research gate.
15. Image 6 Courtesy: Academic.oup.com.

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