

Solar Elastotic Syndrome

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Date:	8 December, 1999
Venue:	Yaumatei Skin Centre
Organizer:	Social Hygiene Service, DH; Clinico-pathological Seminar

CASE SUMMARY

History

A 68-year-old man complained of itchy erythematous rash over his face, neck and upper chest for two years. There was no history of drug intake. He worked as a farmer for 30 years and was retired. There was no recent weight loss, muscular pain or weakness. The rash flares up after sun exposure. His past health is unremarkable. There is no allergic history. He is a non-drinker and non-smoker.

Physical examination

On examination, there were diffuse erythematous rash with telangiectasia, scattered comedones, wrinkling and lichenification over his face, neck and upper chest, sparing the periorbital and postauricular area (as he was wearing a pair of sunglasses). There were some deep furrows noted over the back of his neck (Figure 1). The clinical differential diagnosis included chronic actinic dermatitis, photoallergic contact dermatitis, solar elastotic syndrome, lupus erythematosus, dermatomyositis, and other photoaggravated dermatoses.

Investigations

Photopatch test was performed and were all negative (including olaquinox which is a growth stimulant in pig feeding). Blood tests included baseline haematology and biochemistry were normal. Immune markers showed that ANF titre was 1/120 and the anti-double-strand DNA was not raised. The anti-ENA was negative. The creatinine phosphate kinase and lactate dehydrogenase were normal. A full thickness incisional skin biopsy was performed over his neck. There was extensive deposit of altered elastic fibres, some in

homogenous nodules in the papillary and upper reticular dermis with associated telangiectasia and overlying epidermal atrophy (Figure 2). There was also prominent comedone formation (Figure 3). The histological finding was compatible with solar elastotic syndrome (Nodular Elastosis with Comedone and Cysts).

Management

After the diagnosis of solar elastosis was confirmed, the patient was treated with topical retinoid (tretinoin 0.025% cream). He was also advised to use sunscreen with SPF \geq 15 and avoid further sun exposure.

REVIEW ON SOLAR ELASTOTIC SYNDROME

Definition

This is a gross and microscopic cutaneous degenerative changes that are a consequence of prolonged exposure to chronic solar radiation. It is characterized clinically by yellowish discoloration and histologically by massive elastosis (deposition of abnormal elastic fibres), collagen degeneration, and twisted, dilated microvasculature.

Aetiology

The most important cause is chronic exposure to sunlight (which includes ultraviolet, visible and infrared radiation). Ultraviolet-B (UVB) radiation causes erythema, sunburn, DNA damage and skin cancer. Ultraviolet-A (UVA) radiation causes erythema, (up to 15% of the erythematous response at noon); and because of its deeper penetration into the dermis, it causes disproportionately more chronic photodamage compared to UVB. It can also damage blood vessels, producing endothelial cell enlargement, and vasodilatation. Visible light has been shown to cause UVB tumor enhancement in animals. Infrared radiation produces a typical reticulated pattern of pigmentation of the skin's surface that is a reflection of the underlying



Figure 1: Diffuse erythematous eruption with telangiectasia, comedones, and deep furrows on neck.

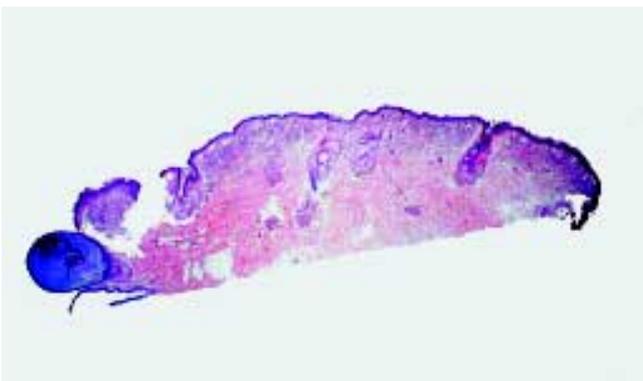


Figure 2: Low power of entire lesion. There is thickening of the entire papillary dermis by extensive deposit of altered elastic fibres associated with sun damage, contrasting the underlying normal eosinophilic reticular dermis. At the edge of the biopsy is comedone consisting of a distended follicular infundibulum. (H&E x20) (By courtesy of Dr. W. Y. Lam, Department of Pathology, TMH)

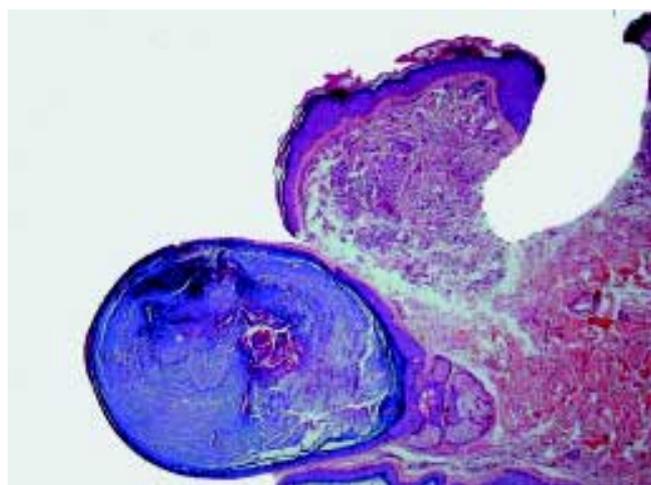


Figure 3: High magnification showing comedone and adjacent skin with sun altered elastic fibres in the papillary dermis. (H&E x100) (By courtesy of Dr. W. Y. Lam, Department of Pathology, TMH)

dilated, leaking blood vessels. It can also induce elastosis, telangiectasia, epidermal atypia, and keratosis. The clinical cutaneous damage is related to the cumulative dose of radiation and individual variation in susceptibility (for example skin type). Photoaging, particularly wrinkling and skin cancer, is exacerbated in smokers.

Clinical features

Clinical changes may vary in different races with different skin type. They include various degree of wrinkles, furrows and yellow discoloration of the skin on light-exposed areas, for example the forehead and the back of the neck (irregular rhomboidal pattern-cutis rhomboidalis nuchae). A combination of senile comedones, infundibular follicular cysts around the periorbital area with elastotic degenerative changes is known as *Favre-Racouchot* syndrome. Other associated solar damages such as laxity, roughness, irregular hyperpigmentation, telangiectasia, solar keratosis and malignancy may also be found.

Histopathology

At early stage, there is a perivenular lymphohistiocytic infiltrate, with degranulating mast cells, degeneration of collagen and deposition of abnormal elastotic material. In later stage, massive quantity of thickened, tangled, and degraded elastic fibres can be seen, with tightly packed collagen fibrils replace elastic microfilaments and finally degenerates into an amorphous elastotic mass. The microcirculation become dilated and twisted (seen as telangiectasia) and finally very sparse, while vessel walls are initially thickened and later thinned.

Prevention and treatment

General measures

Wearing tightly woven clothing and wide-brim hat are simple and practical ways of protecting the skin. Direct sun exposure from 11 am to 2 pm should be avoided as it constitutes nearly half of the daily ultraviolet light.

Specific measures

Sunscreen

Sunscreen can mitigate the harmful effects of UVA

and UVB on the skin. It also permits the repair of photodamaged skin. In both mouse model and in human studies, sunscreens produced dose-dependent reductions in dermal damage as well as reductions in photocarcinogenesis. In practice, uneven application of sunscreen may reduce its protective effect and higher SPF is recommended for those patients who need more protection from UVR as suggested by a recent study.¹ It may need to be reapplied after swimming or transpiration.

Alpha-hydroxy acids

Alpha-hydroxy acids include lactic, glycolic, or citric acid. It has a normalizing effect on hyperkeratinization.² It also increases viable epidermal thickness and dermal glycosaminoglycans. In higher concentrations, alpha-hydroxy acids appear to result in epidermolysis. The mechanism of action is still unknown.

Antioxidants

Double-blind, placebo-controlled studies have failed to show that either oral vitamin E or beta-carotene supplementation modifies the severity of acute cutaneous photodamage in normal subjects.

Tretinoin

A double-blind study by Griffiths³ demonstrated that papillary dermal collagen I formation was reduced by 56% in photodamaged skin compared with the rate in sun-protected skin ($P < 0.001$). Moreover, the reduction correlated with the degree of photodamage ($P = 0.002$). Treatment with 0.1% tretinoin cream for 10-12 months resulted in an 80% increase in collagen I formation, whereas patients treated with vehicle had a decrease in collagen formation.

A dose-dependent improvement in photodamage was observed. In the six-month multicentre trial, 2-mm diameter punch biopsies were taken at baseline and 24 weeks from the same periorbital region, and computerized image analysis of coded samples was used to quantify selected features from histopathological slides.⁴ Statistically significant ($P < 0.002$) differences from vehicle were found in the tretinoin 0.05% groups for three histological features: (i) a greater increase from baseline in epidermal thickness; (ii) a greater increase in granular layer thickness; and (iii) a greater transformation of the stratum corneum from its usual basket-weave pattern into a compact morphology. Melanin content decreased markedly in tretinoin 0.05%-treated patients and was statistically significantly ($P = 0.017$) decreased compared with vehicle-treated patients in one of the two studies.

After 12 months of tretinoin therapy, solar elastosis decreased, and an increased number of fibroblasts were observed in the dermis. In addition, packed collagen fibres became well organized into woven bundles with a normal wavy pattern in half the subjects who formerly had evidenced collagen disorganization. Keratinocytic damage, manifested at baseline by ultrastructural evidence of cytoplasmic degeneration, was negligible after 12 months of therapy.⁵

Overall, tretinoin appears to normalize rather than to stimulate or inhibit cellular proliferation.

The adverse effects of tretinoin include mild-to-moderate skin reactions, such as patchy erythema, burning and mild scaling were commonly recorded during the first 6 months of tretinoin emollient cream therapy. The prevalence of adverse skin reactions in patients treated with the 0.05% tretinoin formulation decreased from a maximum of 71% at week 2 to 47% at week 24.⁶

Learning points:

Solar elastosis is preventable and partially reversible. Photosensitive patients may have to be advised to use high SPF sunscreen because of the inadequate protective effect provided by sunscreen secondary to uneven application.

References

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