Cancer Association of South Africa (CANSA)

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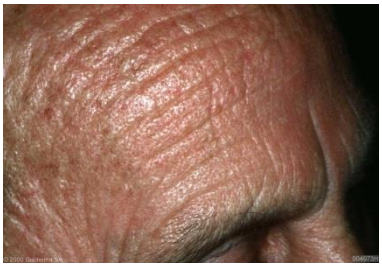
Fact Sheet
on
Solar Elastosis

Introduction

Solar elastosis (also known as Actinic Elastosis) is an accumulation of abnormal elastin (elastic tissue) in the dermis of the skin, and in the conjunctiva of the eye, which occurs as a result of the cumulative effects of prolonged and excessive sun exposure, a process known as *photoaging*. It is most commonly found on the face, lips, ears, back of the hands, forearms, scalp or neck.



[Picture Credit: Solar Elastosis]

Solar Elastosis

Solar elastosis is a medical condition in which the skin modifies its colour (often times it is yellowish) and it becomes thicker, as a direct result of sun damage. It is especially encountered in people with fair complexion.

In the case of elastosis, the collagen layer is damaged and the elastic layer overcompensates by accumulating elastin excessively. The accumulation of abnormal elastin is not only noticed on the skin but also on the eye conjunctiva. It would seem that prolonged and excessive exposure to the sun is the main culprit behind solar elastosis.

Gonzaga, A.K.G., Mafra, R.P., da Silva, L.P., de Almeida Freitas, R., de Souza, L.B. & Pinto, L.P. 2020. "Actinic cheilitis (AC) is a potentially malignant lesion caused by chronic sun exposure. This study aimed to evaluate the relationship between the degree of epithelial dysplasia and morphometric findings in AC. Sixty-eight slides of AC cases were selected and classified according to the grade of epithelial dysplasia, following morphologic criteria of World Health Organization. For morphometric analysis, the slides were scanned and images were analyzed using Pannoramic Viewer software. We obtained vertical measurements of the parameters: thicknesses of the keratin layer, lamina propria and zone of solar elastosis in three selected fields. Thirty-seven (54.4%) of the analyzed cases were classified as none/mild dysplasia and 31 (45.6%) as moderate/severe epithelial dysplasia. Cases with a moderate/severe dysplasia exhibited a thicker layer of keratin (median = 0.055 mm) than none/mild dysplasia (median = 0.045 mm) (p = 0.033). No significant differences in the thicknesses of lamina propria and zone of solar elastosis were observed according to the grade of epithelial

dysplasia. A positive significant correlation between keratin layer and lamina propria thicknesses was found (p = 0.019). Based on our findings, rigorous clinical follow-up should be recommended for patients whose histopathological examination shows a greater thickness of the keratin layer."

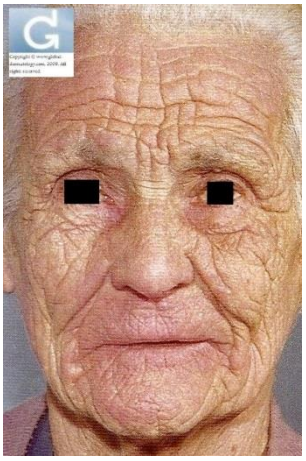
Incidence of Solar Elastosis in South Africa

The National Cancer Registry (2016) does not furnish any information regarding the incidence of Solar Elastosis.

Pathophysiology of Solar Elastosis

Cells within Solar Elastosis show characteristic UV-induced gene mutations.

[Picture Credit: Solar Elastosis 2]



Histologically Solar Elastosis share features with squamous cell carcinoma (SCC). It is an epidermal lesion characterised by:

- Collections of atypical, pleomorphic keratinocytes in the basal layer which can extend to the upper granular and cornified layers.
- The epidermis being abnormal in architecture, with acanthosis, parakeratosis, and dyskeratoses. Cellular atypia is present with keratinocytes varying in size and shape.
- Mitotic figures being present.

It has features of Bowen's disease or carcinoma *in situ*:

- They can be distinguished more by the degree of cellular change and the extent of the lesions rather than differences in the features of individual cells.
- Often, marked hyperkeratosis and areas of parakeratosis with loss of the granular layer are present.
- A dense inflammatory infiltrate is usually present.

Causes of Solar Elastosis

Solar elastosis affects people who have had long term sun exposure and is a feature of photoageing. It affects individuals of all skin types but its yellow hue is more obvious in fair skin individuals. Solar elastosis is also a manifestation of premature skin ageing caused by smoking.

Treatment of Solar Elastosis

The best treatment of solar elastosis is prevention. One can prevent sunburn and the related skin conditions by protecting one's skin whenever outdoors by wearing a broad rim hat, protective clothing, and sunscreen with SPF of at least 30. However, if one sees signs of solar elastosis, there are antiaging skin care products and treatment available.

Charles-de-Sá, L., Gontijo-de-Amorim, N.F., Rigotti, G., Sbarbati, A., Bernardi, P., Benati, D., Bizon Vieira Carias, R., Maeda Takiya, C. & Borojevic, R. 2020.

Background: The major intrinsic cause of facial skin degeneration is age, associated with extrinsic factors such as exposure to sun. Its major pathologic causes are degeneration of the elastin matrix, with loss of oxytalan and elaunin fibers in the subepidermal region, and actinic degeneration of elastin fibers that lose their functional properties in the deep dermis. Therapy using autologous adipose mesenchymal stem cells for regeneration of extracellular matrix in patients with solar elastosis was addressed in qualitative and quantitative analyses of the dermal elastic fiber system and the associated cells.

Methods: Mesenchymal stem cells were obtained from lipoaspirates, expanded in vitro, and introduced into the facial skin of patients submitted after 3 to 4 months to a face-lift operation. In the retrieved skin, immunocytochemical analyses quantified elastic matrix components; cathepsin K; matrix metalloproteinase 12 (macrophage metalloelastase); and the macrophage M2 markers CD68, CD206, and hemeoxygenase-1.

Results: A full de novo formation of oxytalan and elaunin fibers was observed in the subepidermal region, with reconstitution of the papillary structure of the dermal-epidermal junction. Elastotic deposits in the deep dermis were substituted by a normal elastin fiber network. The coordinated removal of the pathologic deposits and their substitution by the normal ones was concomitant with activation of cathepsin K and matrix metalloproteinase 12, and with expansion of the M2 macrophage infiltration.

Conclusion: The full regeneration of solar elastosis was obtained by injection of in vitro expanded autologous adipose mesenchymal stem cells, which are appropriate, competent, and sufficient to elicit the full structural regeneration of the sun-aged skin.

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