

Skin Ageing and Dermatoporosis

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Dermatoporosis is a recent term that defines the clinical and functional aspects of chronic cutaneous insufficiency/fragility syndrome related to skin ageing. Dermatoporosis is frequently seen in geriatric medicine. It mainly affects patients in the age range 70–90 years, and leads to important functional disabilities and high management costs. Management of dermatoporosis involves the prevention of trauma in elderly patients. Topical treatments, such as hyaluronic acid and retinaldehyde, may help to correct the skin atrophy. Topical vitamin C has recently proved beneficial in this indication.

Key words: ageing; dermatoporosis; fragility; haematoma; hyaluronic acid; purpura; vitamin C.

Skin ageing

Skin ageing is an unavoidable natural process, which varies considerably from one individual to another, depending on genetic/ethnic and environmental factors (mainly sun exposure and tobacco consumption) (1). Chronological or intrinsic ageing is responsible for skin that gradually thins, dehydrates and depigments, appearing withered, with a loss of elasticity (Tables I and II). Menopause and physical or psychological illnesses (such as depression) accentuate chronological ageing (2). In addition, extrinsic ageing is mainly related to photo-ageing due to repeated exposure to solar radiation (Table III, Figs 1–5) (2, 3), which may be aggravated by smoking (4, 5). Other factors may eventually affect ageing, such as alcohol intake, drug use (e.g. crack cocaine), or possibly atmospheric pollution (2).

Dermatoporosis: an emerging concept

“Dermatoporosis” is an emerging clinical condition due to increased life expectancy and ageing of the population. This recent neologism was first proposed by a Swiss dermatologist, Pr Jean-Hilaire Saurat (6), and can be compared to (rheumatological) osteoporosis. The term dermatoporosis refers to various manifestations related to chronic fragility of the skin and its consequences. It implies the need to treat, but also to prevent, this age-related condition, as rheumatologists endeavour to do with osteoporosis (6–10). The first manifestations of dermatoporosis occur at approximately 60 years of

Table I. Morphological changes in normal aged skin, after Fenske & Lober (1)

Epidermis
↓ Thickness
↓ Vertical height and ↑ Surface of keratinocytes
↓ Corneocyte adhesion
Flattening of the dermal-epidermal junction
Duplication of lamina densa and fibrillar anchorage complex
↓ Number of melanocytes
↓ Number of Langerhans cells
Dermis
↓ Number of fibroblasts
↓ Number of mast cells
↓ Capillary network
Alteration of blood vessels
Abnormal nerve endings
Adnexal structures
↓ Number of eccrine glands
Attenuation of eccrine and apocrine glands
Hyperplasia of the sebaceous glands
↓ Number of scalp and facial hair follicles
↓ Hair shaft thickness
Hair greying, canitia
Nail thinning
↓ Size of the lunula
Subcutaneous tissue
↓ Especially in the face, hands, legs and feet
↑ Abdominal belt (man) and thighs (woman)

Table II. Clinical correlation of normal skin ageing after Fenske & Lober (1)

Pallor and laxity of the skin
Hair greying
Scalp and beard alopecia
Xerosis
↑ Terminal hair follicles: face, ear, nose
Nail fragility
↑ Treatment time of onychomycosis
Fragility of the skin
Purpura and haematomas, vulnerability of subcutaneous tissues
↑ Susceptibility to skin tears and bubbles
↓ Dermatoglyphs
↓ Naevus
Irregular pigmentation
↓ Sensation
↓ Ability to make fine gestures
↓ Body odour
Benign and malignant skin tumours

Table III. *Signs of skin photoageing (dermatoheliosis)*

Face and neck
Thickened yellowish and citrus-coloured skin with dilated follicular orifices
Comedones and cysts of the face (cutaneous nodular elastoidosis, Favre-Racouchot disease)
More or less deep wrinkles of the face
Cutis rhomboidalis nuchae
Sebaceous hyperplasias (forehead, nose, cheeks)
Rosacea
Erythrosis interfollicularis coli
Solar lentigos
Actinic keratoses
Limbs
Thinned skin
Bateman's senile purpura
Stellar pseudoscars
Solar lentigo
Guttate hypomelanosis of the lower limbs
Skin cancers (basal cell carcinoma, squamous cell carcinoma, melanoma)

age, but the condition takes its complete form between 70 and 90 years of age (7).

The prevalence of dermatoporosis is unclear. According to a prospective study of 202 patients over 60 years of age, conducted in the Geriatric Department of the University Hospital of Toulouse (south-east France), 32% of hospitalized patients had signs of dermatoporosis (11). A more recent study of

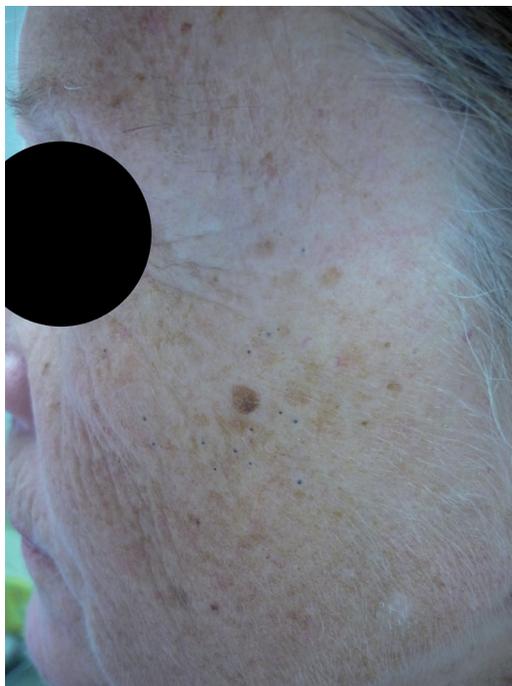


Fig. 1. Multiple signs of skin photoageing in a woman: crow's feet and cheek wrinkles, terminal hairs, sun lentiginos and black comedones of Favre-Racouchot disease.



Fig. 2. Multiple cysts and open comedones of Favre-Racouchot (or cutaneous nodular elastosis, Favre, 1932, Racouchot, 1951) in a man.

a representative sample of the French population ($n=533$) aged 65 years and over, reported that 37.5% of respondents presented with dermatoporosis (12).

Clinical manifestations of cutaneous fragility include atrophy, mainly on sun-exposed areas (upper limbs, pre-tibial regions), Bateman's senile purpura, and spontaneous asymptomatic white porcelain stellar pseudoscars. The lesions are more often located around the cleavage/décolleté area and anterior aspect of the legs in women, and forearms and forehead in men (Fig. 6) (10).



Fig. 3. Erythrosis interfollicularis coli (Leder, 1944 or "red neck") in a woman. Note that the shaded triangle under the chin is spared.



Fig. 4. Idiopathic guttate hypomelanosis of the limbs (Costa, 1951).



Fig. 5. Cutis rhomboidalis nuchae (Jadassohn, 1925).

The functional consequences of this cutaneous fragility are the development of cutaneous lacerations, delayed wound healing and deep dissecting haematomas between the subcutaneous fat and the muscular fascia, which can lead to large areas of necrosis due to vascular ischaemia (Fig. 7) (9). Of note, Saurat et al. (12), developed a 14-item questionnaire for evaluating dermatoporosis in adults, which is relatively easy to perform

Four stages of dermatoporosis were defined by Kaya & Saurat in 2007 (8) (Table IV). In a study by Mengeaud et al. (11), 82–92% of patients had stage 1 lesions, 80–14% stage 2 lesions, and 4% stage 3 lesions. No patient presented with deep dissecting stage 4 haematomas (9).

Local or systemic corticosteroids are considered an aggravating factor in dermatoporosis (8). However, Mengeaud et al. did not find such correlation in their study, but instead found a significant association between dermatoporosis and severe chronic renal failure (11). Long-term anticoagulation can promote the development of dissecting haematomas and various co-morbidities (diabetes, arteriovenous insufficiency of limbs, polyneuropathies) contribute to delayed healing (9). Finally, vitamin C deficiency must not be neglected. Indeed, vitamin

C deficiency is responsible for purpura and haematoma, and elderly people are at risk of vitamin C deficiency. Vitamin C deficiency may be an aggravating factor in dermatoporosis, as we have observed (Fig. 7) (13).

Histologically, dermatoporosis is characterized by a cutaneous atrophy affecting all skin components. There is a striking decrease in hyaluronic acid in the extracellular matrix of the dermis, affecting the viscoelasticity of the skin (14). Amyloidosis is ruled out by negative specific staining, such as Congo red.

Dermatoporosis is primarily a consequence of ageing and prolonged sun exposure, but some genetic factors may be relevant (8). It is linked to a progressive alteration of the extracellular matrix of the dermis and, in particular, of its major component, hyaluronic acid and its cellular receptor, CD44 (8). Indeed, hyaluronic acid is an active molecule that induces intracellular signalling after binding to the CD44 membrane receptor. Activation of CD44 regulates keratinocyte proliferation and controls the homeostasis of hyaluronic acid.

Care involves prevention of minimal trauma, which can be responsible for major bruising in elderly people. This is ena-



Fig. 6. Examples of dermatoporosis presentations. A) Bateman senile purpura (Bateman, 1818) and pseudoscars of the hands. B) Marked cutaneous atrophy, senile purpura and pseudoscars of the forearms. C) Extensive dermatoporosis of the décolleté and right upper limb with purpura, pigmentation, pseudoscars and cutaneous lacerations.



Fig. 7. Extensive spontaneous hematomas of the lower limbs in a patient with low levels of vitamin C (13).

bled through the education of nursing staff, protection of the lower limbs, adaptation of furniture at home and in public places, correction of visual disorders, etc. On the other hand, topical treatments including intermediate-sized fragments of hyaluronic acid (ranging from 50,000 to 400,000 Da, but not smaller or larger) could correct the cutaneous atrophy of dermatoporosis induced by age or local corticosteroid therapy through a dependent CD44 mechanism (15). In addition, a synergistic action has recently been shown in mice *in vitro* and *in vivo*, but also in patients with dermatoporosis, when applying retinaldehyde and fragments of hyaluronic acid of intermediate size (16, 17). Recently, Humbert et al. (18) showed that the application of a 5% vitamin C topical ointment twice daily for 12 weeks was efficient in the treatment of Bateman's purpura: it corrected the purpura and increased dermal thickness. Vitamin C deficiency may play a role in the pathogenesis of senile purpura and may be related to local scurvy (18).

With increasing life expectancy and an ageing population, dermatoporosis could become a public health problem, a

Table IV. Different stages of dermatoporosis according to Kaya & Saurat (8)

Stage I	Extreme skin atrophy, purpura, stellate pseudoscars
Stage II	Stage I lesions and some skin lacerations due to minor trauma
Stage III	Numerous and large cutaneous lacerations with a notable delay of wound healing
Stage IV	Deep dissecting haematoma (s) associated with any lesion of another stage, which may result in skin necrosis

source of healthcare costs and hospitalization. It is important that caregivers can recognize this pathology in order to avoid unnecessary, or even harmful, explorations and therapies. In addition, new local topical treatments should emerge in the future for the management of dermatoporosis and, more generally in cosmetology, for cutaneous atrophy induced by ageing and/or corticosteroid therapy (15, 19).

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