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Dermatoporosis – The Chronic Cutaneous Fragility Syndrome

Uwe Wollina[1*], Torello Lotti[2], Aleksandra Vojvotic[3], Andreas Nowak[4]

[1]Department of Dermatology and Allergology, Städtisches Klinikum Dresden, Academic Teaching Hospital, Dresden, Germany; [2]Department of Dermatology, University of Rome "G. Marconi", Rome, Italy; [3]Military Medical Academy of Belgrade, Belgrade, Serbia; [4]Department of Anesthesiology and Intensive Care Medicine, Emergency Medicine and Pain Management, Städtisches Klinikum Dresden, Dresden, Germany

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***Correspondence:** Uwe Wollina. Department of Dermatology and Allergology, Städtisches Klinikum Dresden, Academic Teaching Hospital, Dresden, Germany. E-mail: uwollina@gmail.com

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Abstract

Dermatoporosis is an important clinical condition leading to chronic skin fragility. It can be separated into primary and secondary subtypes, with the latter induced by medical drugs and environmental factors. Dermatoporosis can be classified into 4 major stages with increasing morbidity and mortality with the advanced stages. Its aetiology has been related to the epidermal hyalusome. Dermatoporosis is a cause of mortality in the intensive care unit and should be known not only by a dermatologist but another medical speciality as well. Prevention is of major importance. Therapeutic options are limited but available.

Introduction

Cutaneous ageing has been divided into intrinsic and extrinsic ageing based upon the origin of changes leading to the ageing process. Cutaneous ageing is characterised by pigmentary, vascular, connective tissue and adipose tissue aspects that are contributing to the complex process. The ageing process is genetically determined but can be largely be influenced by environmental factors such as ultraviolet radiation, air pollution and smoking [1].

Dermatoporosis

Dermatoporosis is the term coined by Saurat that covers all the aspects of the chronic cutaneous fragility syndrome [2]. Dermatoporosis describes a loss of function that eventually results in a breakdown of the protective mechanisms of human skin. We differentiate primary forms due to increased age and extensive exposure to sunlight from secondary forms due to certain medications (see below).

The prevalence of dermatoporosis in 202 elderly French hospital in-patients aged between 60 to 80 years has been calculated as high as 32% [3]. A prospective trial of the department of dermatology of Helsinki University Central Hospital analysed 176 consecutive outpatients aged ≥ 60 years. Dermatoporosis was evident in 30.7% of patients, mainly on the upper limbs (94%). The authors performed multivariate analysis for possible risk factors. Dermatoporosis was significantly associated with ultrapotent topical corticosteroids (odds ratio (OR) 5.34), oral corticosteroids (OR 3.22), concomitant corticosteroid therapy, anticoagulant and chronic renal failure (OR 4.02) while age had only a marginal impact (OR 1.05). Patients with bullous pemphigoid were those with the highest prevalence of dermatoporosis in their cohort (64%) [4].

The prevalence is slightly higher in another

French study performed in a representative sample of the population (n = 533): Here, the estimated overall prevalence of dermatoporosis was 37.5% in subjects aged older than 65 years with a predominance of women [5].

Dermatoporosis has been staged into 4 stages (Table 1).

Table 1: Staging of dermatoporosis (according to [5])

Stage I:	Skin atrophy, senile purpura and pseudo-cicatrices
Stage IIa:	Localised and small superficial lacerations (< 3 cm) due to skin fragility
Stage IIb:	Larger lacerations (> 3 cm)
Stage IIIa:	Superficial hematomas
Stage IIIb:	Deep dissecting hematomas without skin necrosis
Stage IV:	Large areas of skin necrosis with potentially lethal complications

Aetiology

In the case of corticosteroid-induced skin atrophy, the hyalurosome of filopodia of epidermal keratinocytes becomes weakened [6]. This organelle is composed of hyaluronic acid receptor CD44, heparin-binding epidermal growth factor (HB-EGF), HB-EGF receptor erbB1 and hyaluronic acid synthase 3. The hyalurosome is involved in different functions such as secretion of hyaluronic acid and epidermal growth factor-receptors signalling. It is anchored on F-actin fibres. Investigations in a mouse model suggested that the hyalurosome is the target of corticosteroids and involved in corticosteroid-induced epidermal atrophy and dermatoporosis [7].

As a consequence of these molecular mechanisms, dermatoporosis skin demonstrated peculiaritis in the viscoelastic properties of the affected skin. In the steep suction mode using the 4 mm aperture probe, the comparison with normal skin showed that residual deformation (RD) was significantly increased ($P < 0.05$) in dermatoporosis. In the progressive suction mode using the same aperture probe, the comparison with normal skin revealed a significant increased RD in dermatoporosis ($P < 0.05$). A combination of the 2 mm aperture probe with the outer guard ring yielded significant ($P < 0.05$) hysteresis increase in dermatoporosis compared to normal skin [8].

Bateman purpura

Bateman purpura is a classical sign of photo-ageing, characterised by hemorrhagic areas with purpuric eruptions like petechial or confluent ecchymoses, by stellar scars, and a fragile skin due to thinness of the dermis [9].

These features are mainly localised and the back of the hands and the forearms (Figures 1 and 2).

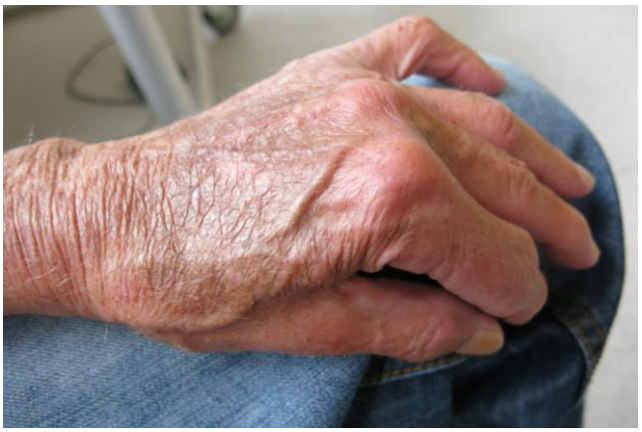

Figure 1: Dermatoporosis stage I with pronounced skin atrophy and pigmentary changes

It has been demonstrated, that in the affected parts of the skin, there is a depletion of the photoprotective vitamin C due to chronic ultraviolet light (UV)-exposure [10].

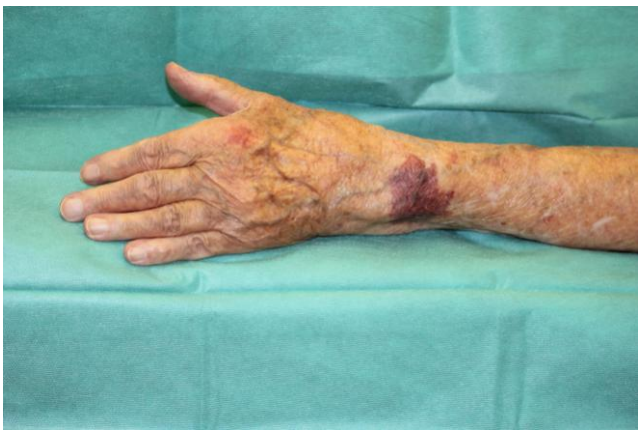

Figure 2: Dermatoporosis with advanced skin atrophy and Bateman purpura in a patient with bullous pemphigoid

Complications of dermatoporosis

Laceration and delayed wound healing (Fig. 3)

From stage IIa onwards, laceration (skin tears) is a common feature of dermatoporosis. This symptom is caused by blunt trauma. Underlying mechanisms are age-related skin changes, but also dehydration, malnutrition, sensory changes, mobility impairment, pharmacological therapies and mechanical factors related to skincare practices [11]. Due to the delayed wound healing, it has a risk of soft tissue infections [12].

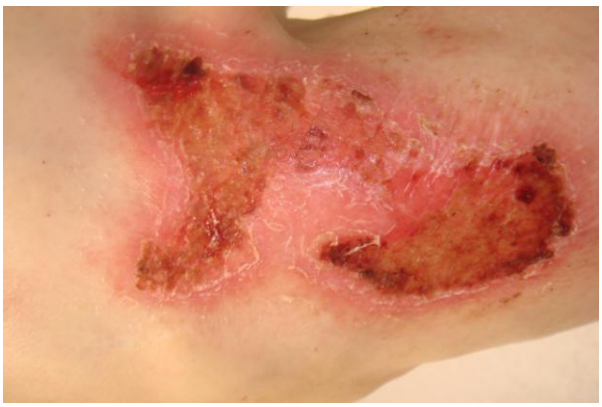

Figure 3: Delayed wound healing after laceration in a 76-year-old male patient with Parkinson's disease

Deep dissecting hematoma

Deep dissecting hematoma (syn. chronic expanding hematoma) is an emergency [13]. A Swiss study reported a close connection to dermatoporosis. The legs were affected in all patients, most frequently in older women (mean age 81.7 years). Risk factors were long-term treatment with systemic corticosteroids and anticoagulation. Deep dissecting hematoma presents with pain and swelling, erythema and oedema without fever (Figure 4). Skin necrosis was a late symptom. Magnetic resonance imaging and histopathological analysis confirmed the deep anatomical location of the hematoma. Treatment consisted of deep incision and/ or debridement followed by direct closure, skin grafting, or wound healing by second intention [14].

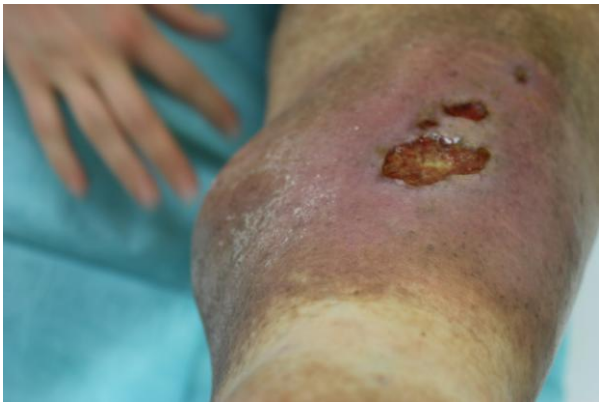

Figure 4: Chronic deep dissecting hematoma of the lower leg leading to secondary ulcerations of the skin

Dermatoporosis in the Intensive Care Unit (ICU)

Skin failure is defined as loss of normal temperature control combined with the inability to maintain the core body temperature, percutaneous loss of fluid, electrolytes and protein, and failure of the mechanical barrier to prevent penetration of germs [15]. In an analysis of 552 adult patients admitted to the ICU, a logistic model was developed to differentiate pressure sores from acute skin failure. The identified risk factors for acute skin failure were peripheral arterial disease (odds ratio [OR] 3.8), mechanical ventilation greater than 72 hours (OR 3.0), respiratory failure (OR 3.2), liver failure (OR 2.9), and severe sepsis and/or septic shock (OR 1.9) were independent predictors (Figure 5) [16].

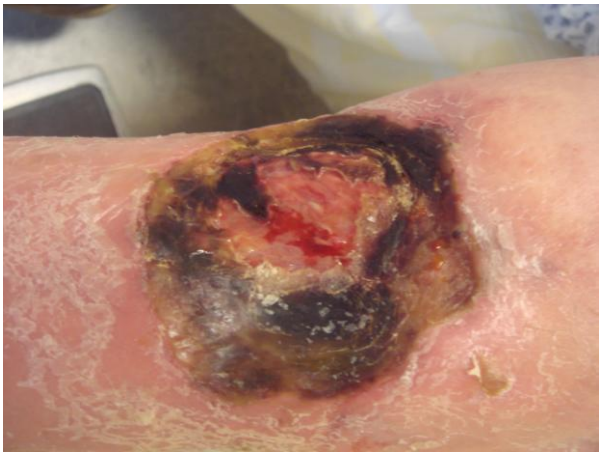

Figure 5: Ulceration and delayed wound healing after removal of a saphenous vein graft for coronary artery bypass graft surgery

Dermatoporosis is a condition that can lead to acute skin failure in the ICU. Dermatoporosis has a high prevalence in autoimmune bullous disorders such as pemphigoid, and bullous disorders are among the dermatological diagnoses that can lead to an ICU admission (Figure 4 and 5) [4], [17], [18].

Prevention and treatment of dermatoporosis

Prevention of dermatoporosis is possible by limiting the exposure to known inducers of this skin condition such as extrinsic factors like ultraviolet radiation, pollution or smoking, and medical drugs like topical and systemic corticosteroids [19].

Treatment of dermatoporosis is principally possible, although best results are obtained in stage I. In a mouse model, intermediate size hyaluronic acid fragments (HAFi) inhibited the downregulation of filopodia and skin atrophy induced by clobetasol propionate. Topical treatment of atrophic forearm skin of dermatoporosis patients with HAFi 1% for 1 month resulted in a significant clinical improvement. Also, the expression of hyalurosome molecules was induced. Topical retinaldehyde 0.05% and HAFi 1%

demonstrated synergy in hyaluronic acid production and heparin-binding epidermal growth factor in mouse skin and in dermatoporosis patients [6], [7], [20].

Other topical modalities to increase skin thickness include alpha-hydroxy acids twice daily for at least three months [21] or topical dehydroepiandrosterone 1% cream (in women) twice daily for four months [22].

In a single-centre, intra-individual randomised, double-blind and placebo-controlled clinical trial, topical vitamin C was used to improve Bateman pupura. The patients received either an active cream containing 5% of vitamin C (L-ascorbic acid) vs a neutral cream twice daily.

In this trial, topical vitamin C led to a clinically apparent improvement of purpura and measurable improvement of skin elasticity (Cutometer SM 575®; Courage and Khazaka, Köln, Germany) and thickness (Harpenden skin-fold calliper) [23].

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