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Case Report

Therapeutic options in dermatoporosis: A case report

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Abstract Dermatoporosis is a chronic skin insufficiency syndrome with typical manifestations of extreme skin atrophy, senile purpura, stellate pseudoscars, skin lacerations, and skin dissecting hematomas at the age of 70–90 years old. It is caused by aging process, and triggered by environmental factors and drugs which lead to the skin's protective mechanical function loss. The prevalence was 32% in French Hospitals and 30.7% in the Central Hospital of the University of Helsinki, Finland. We report a dermatoporosis case to raise awareness in clinical practice and trace study literature for choosing the most appropriate therapy. An 89-year-old man complained of red and itchy patches on his skin since the last six months. Physical examination shows dry skin, atrophy, purpura, stellate pseudoscars, and multiple arms and lower leg lacerations. The patient had been given ceramide moisturizer, but there is no significant improvement. Structural and functional skin damage in elderly is not just cosmetic problem, it significantly impacts life quality and increases morbidity and mortality risk if it develops into a serious condition. Therefore, clinicians have an essential role in proper management to prevent complications. Several studies have shown that hyaluronic acid and retinaldehyde improve skin function in dermatoporosis.

Key words
Dermatoporosis; Senile purpura; Skin aging; Moisturizers.

Introduction

The term 'dermatoporosis' is analogous as 'osteoporosis' which was first stated by Kaya and Saurat in 2007.[1] Along with age, the structure and function of the skin as a barrier against external exposure progressively decline, leading the skin to become drier, wrinkled, sagging, and lose its elasticity, which increases susceptibility to irritation and infection.[2] Dermatoporosis is divided into primary and secondary types. Primary dermatoporosis is a common form caused by chronological aging, while secondary is caused by prolonged UV exposure, the use of topical and systemic corticosteroids, or other medical drugs.[1-2] Furthermore, typical manifestations of dermatoporosis are classified into 4 stages: stage 1-skin atrophy, senile purpura, and stellate pseudoscar; stage 2-skin lacerations <10 lesions; stage 3-lacerations ≥10 lesions; and stage 4-skin dissecting hematoma caused by massive subcutaneous bleeding leading to ischemia and extensive skin necrosis.[3,4]

Demographic data on dermatoporosis is still limited. A study at a hospital in France shows the prevalence of dermatoporosis to be 32% in the elderly. Meanwhile, other studies reveal a national prevalence in France of 37.5%, which increases with age and shows a female-to-male ratio of 3:2.[5,6] We present one case of dermatoporososis to raise awareness of this clinical condition and to assist clinicians in choosing appropriate therapy for elderly patients.

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Case report

An 89-year-old man complained of red and itchy

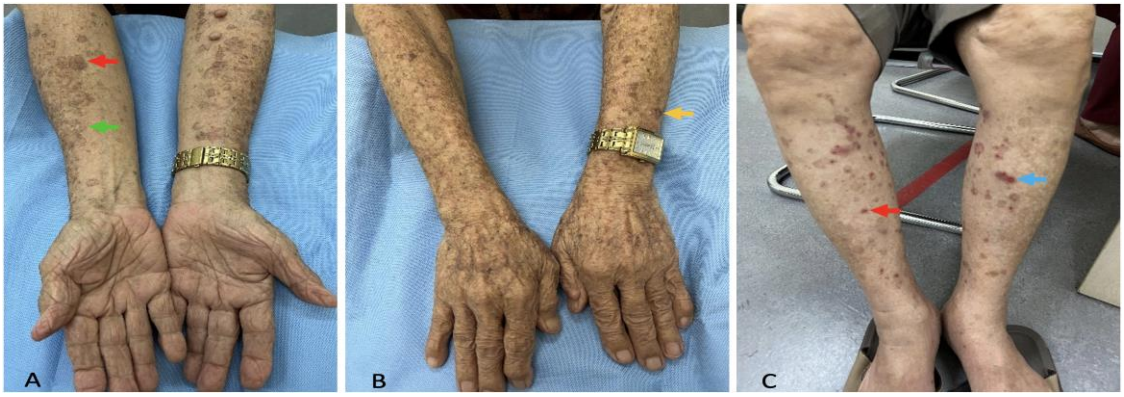

Figure 1 89 years old male patient shows signs of skin aging and stage II dermatoporosis; A) extensor hands, B) flexor hands, C) legs, arrow descriptions: skin atrophy (green), pseudoschar (yellow), senile purpura (red) and lacerations <10 lesions (blue).

patches on the skin which had been very disturbing for the last six months. The patient visited several doctors for five months and it was diagnosed as dermatitis. He had been given topical steroids and glycerine moisturizer, but there was no change. Dermatological examination of the extremities, superior and inferior, showed dry skin, atrophy, purpura, stellate pseudoscar, and laceration with more than 10 lesions, as shown in **Figure 1**, leading to signs of stage II dermatoporosis. He has hypertension and takes regular antihypertensive medication but he does not take any anticoagulant or antiplatelet drugs. The therapy given was a moisturizer containing ceramide and glycerol twice a day, petroleum jelly at night, topical antibiotics for wound lesions, and SPF 50 sunscreen.

Discussion

Skin aging is intrinsically caused by genetic and hormonal factors that affect the components of the extracellular matrix of the dermis (collagen, elastin, fibrillin, glycosaminoglycans (GAGs), and hyaluronate (HA), shortening of rete ridges, nutritional deficiencies, and a decrease in subcutaneous lipid levels, which cause atrophic skin, fine wrinkles, dryness, and sagging, so that the skin is easily torn after light trauma.[2] Extrinsically, it is triggered by exposure to UV light, which induces metalloproteinases (MMP) to degrade collagen and elastin fibers, resulting in the accumulation of abnormal elastin tissue (elastosis) that is characterized by deeper wrinkles, brittleness, roughness, and changes in skin pigmentation.[2]

Primary dermatoporosis is associated with chronological aging factors, while secondary dermatoporosis is due to UV light exposure, use of drugs such as topical or systemic corticosteroids, antiplatelet, anticoagulant, and EGFR inhibitors, as well as disease comorbidities such as chronic kidney failure.[7] HA is the main component of the extracellular matrix in the dermis, which functions to maintain hydration and skin viscoelasticity. It also plays a role in inflammatory processes, angiogenesis, tumor progression, and skin tissue repair.[8] The pathogenesis of dermatoporosis is associated with a reduction in HA levels, dysregulation of CD44 receptor expression, and dysfunction of the hyalurosome macromolecular complex, which is involved in HA metabolism and cell signaling in keratinocytes. This complex includes CD44, heparin-binding epidermal growth factor (HB-EGF), and the erbB1receptor, which leads to dermal atrophy.[7]

The manifestations of dermatoporosis are characterized by atrophy, purpura, pseudoscars,

and skin lacerations in areas that are exposed to UV rays, such as the head, neck, arms, legs, and chest, as well as areas that are easily accessible and show excoriated lesions due to scratching.[3] Moreover, diagnosis of dermatoporosis includes clinical manifestations, physical examination, and ultrasound images. The scoring dermatoporis created by Kaya, by assessing skin manifestations and dermal thickness, was classified as: 1–7 early stages (I), 8–9 early intermediate stage (IIa), 10–12 late intermediate stage (IIb), 13–16 early advanced stage (III), >16 advanced stage (IV).[1] Based on this score, our patient is classified in the early stage (II).

The histological appearance of dermatoporosis shows shortening of the rete ridges, solar elastosis, and dermal atrophy; compared to the typical range of 1.4–1.5 mm, ultrasonography reveals a reduction in dermal thickness of 0.7–0.8 mm.[7] Previously our patient used moisturizer containing glycerine, vitis vinifera seed oil, sodium lactate, tocopheryl acetate, biosaccharide Gum-1, and panthenol and it did not improve the skin because of the use of topical steroids at the same time, which reduced the therapeutic effect.

Topical corticosteroids reduce the expression of CD44 receptors, especially the CD44v3 receptor, which is a special isotype located in the keratinocyte membrane and has an impact on skin thickness.[7] It is believed to be a secondary risk factor in patients as reported by a cohort study in Japan with 149 elderly participants. The incidence of easy skin tearing, which was observed for 8 months, was found to be a predictor of epidermal thickness, with a cutoff point of 0.80 mm being an associated predictor.[9]

The current patient's therapy was replaced with ceramide but no skin improvement was observed. Glycerol is a hygroscopic compound, while ceramide is a lipid complex consisting of sphingoid bases conjugated with fatty acids through amide bonds, both of which function in maintaining skin barrier homeostasis by reducing TEWL (trans epidermal water loss) and increasing hydration in the stratum corneum of the epidermis.[10,11] However, there are no studies that have studied its effect on dermal structure and function. Therefore, it is necessary to think about other moisturizing options that are suitable for the patient. Dermatoporosis therapy focuses on repairing dermal atrophy with activation of hyalurosome molecules, which increases the amount of HA and expression of CD44 receptors.[1]

In an RCT trial, 15 of the 22 elderly patients with senile purpura who received topical vitamin C 5% twice daily for 12 weeks demonstrated clinical improvement compared to the placebo group's 47%, with clinically significant results of $p<0.05$.[12] Skin elasticity in the active cream group increases (66.7%) compared to the placebo group (16.7%). Additionally there was a gradual increase in skin thickness in the active group while the placebo group remained stable and even decreased.[12] Another study that treated 67 elderly patients with oral bioflavonoids and citrus supplements for six weeks revealed a 50% improvement in purpuric lesions compared to the control group and a statistically significant decline in the frequency of new lesions.[13]

In several studies, topical hyaluronate fragment intermediate (HAFi) and retinaldehyde (RAL) significantly increased dermal thickness up to 1 mm.[1,14] The expression of the CD44 and Hyal2 genes was significantly raised in a different study using topical mixtures of HAFi 1% and RAL 0.05% twice daily for 30 days. This increased expression is associated with increased dermal secretion of collagen and HA.[8] HAFi, or sodium hyaluronate, is a low-molecular-weight HA with a molecular weight of 80–150 kDA that can stimulate keratinocyte proliferation and epidermal hyperplasia.[1,14] In a large multicenter

cohort study, subjects were divided into three groups: topical RAL 0.05%-HAFi 0.5% in 242 subjects, serum RAL 0.05%-HAFi 1% in 177 subjects, and used together in 1003 subjects over three months. All three groups showed significant improvement, as evidenced by decreased Larnier's scores, improved wrinkles in groups 2 and 3, and improved signs of photoaging in groups 1 and 3.[15,16] In addition, purpuric lesions can be a clinical indicator of response to RAL-HAFi therapy with decreased number and improvement of purpuric lesions after one month of topical treatment.[1,8]

A pilot clinical study with topical human epidermal growth factor (EGF) twice daily for 6 weeks in 6 subjects with senile purpura showed an increase in epidermal thickness and reduced the mean number of lesions from 15 to 2,3.[17] Moreover, lactic acid 5% and 12% twice daily showed improvement in the thickness, density, and smoothness of the epidermis and dermis in 42 subjects.[7] Alpha-hydroxy acid (AHA) and citric acid treatment were found to increase skin thickness by 16.3% and 25%, respectively, in a different study.[7] Alternative therapy with intense pulsed light (IPL) and microneedling radiofrequency (RF) can improve clinical senile purpura.[18,19] The number and size of purpuric lesions improved after four IPL treatments were administered weekly to five subjects; in addition, dermal and epidermal thickness also improved statistically, despite the subjects' taking corticosteroids and anticoagulants.[18] After one microneedling (RF) procedure, the quantity of collagen and elastic fibers as well as the average thickness of the epidermis also rise, however this study lacks statistical significance.[19]

Systemic dehydroepiandrosterone (DHEA) therapy can increase skin hydration and reduce skin atrophy after oral supplementation of DHEA 50 mg per day for 1 year as indicated in a randomized placebo-controlled study with 280 elderly subjects.[20] Moreover, the same result is found with estrogen replacement therapy in menopausal women who consume conjugated estrogens.[21] Although there are not many moisturizing preparations containing these ingredients, some are available in Indonesia and can be administered to patients.

Conclusion

Structural and functional skin damage in the elderly is not just a cosmetic problem. It has a significant impact on life quality and increases morbidity and mortality risk. Dermatoporosis can develop to be severe, so clinicians have an important role in proper management to prevent complications and improve the patient's quality of life. The focus of dermatoporosis therapy is to improve skin atrophy and dysregulation of hyaluronic acid and hyalurosome molecules according to the pathogenesis. Moreover, several studies show that a combination of hyaluronate fragment intermediate and retinaldehyde can improve skin function in dermatoporosis. Topical or systemic administration of vitamin C, lactic acid, AHA, IPL measures, microneedling (RF), and hormonal medication may also be potential therapies. However, further clinical studies are needed to evaluate the effectiveness of these various therapies.

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