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Review

Aging of the skin. From molecular mechanisms and clinical aspects to chronic skin failure and social impact

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Abstract: The process of aging population is increasing the relevance of comprehending age-related changes in skin structure and function, as well as their implications for clinical care. This narrative review explores aspects of the intrinsic and extrinsic mechanisms of skin aging, their molecular and morphological consequences, and their clinical expression, with particular attention to chronic cutaneous insufficiency. The intrinsic aging process is reflecting declining cellular functionality, impaired tissue homeostasis, and endocrine changes, especially regarding estrogen deficiency after menopause. These processes are reducing collagen synthesis, hydration, and elasticity and thus contributing to epidermal and dermal atrophy and barrier dysfunction. On the other hand, extrinsic aging is driven mainly by cumulative ultraviolet exposure, with additional contributions from pollution, smoking, diet, and behavioral and psychosocial factors. Processes such as oxidative stress, inflammation, DNA damage, glycation, and increased matrix metalloproteinase activity promote extracellular matrix degradation, linking the environmental exposure to the photoaging and cutaneous carcinogenesis. Main clinical manifestations include xerosis, chronic pruritus, pigmentary changes, benign lesions, actinic keratoses, and basal cell and squamous cell carcinomas. Inflammatory, allergic, autoimmune, and drug-induced dermatoses, including psoriasis, lichen planus, and bullous pemphigoid, are complicating healthcare in older adults, while systemic comorbidities and medication use are influencing clinical presentation and treatment. Dermatoporosis is characterized by cutaneous atrophy, purpura, pseudoscars, and susceptibility to injury after minor trauma. Reduced hyaluronic acid availability and CD44 expression contribute to impaired hydration and mechanical resilience, potentially leading to ulceration and deep dissecting hematomas. Management measures are emphasizing the role of photoprotection, skin hydration, trauma prevention, and reduction of modifiable exposures, with corticosteroid use warranting reassessment in secondary dermatoporosis. Other discussed approaches are topical hyaluronic acid, retinoids, alpha-hydroxy acids, and vitamin C, but the proposed interventions have limited supporting evidence. Therefore, it is considered that early recognition of these interconnected changes may be supportive for planning prevention, a timely diagnosis, and individualized care, based on considerations like skin fragility, associated disease, and treatment burden in older patients.

Keywords: skin aging; endocrine aging; sun exposureȘ dermatoporosis; photoaging; oxidative stress; skin barrier dysfunction

EPIDEMIOLOGICAL ASPECTS OF POPULATION AGING

According to recent statistics, 10% of the world's population is over the age of 60, and this percentage is expected to double by the end of the 21st century. Aging is considered a global phenomenon with an estimated increase in the number of individuals aged 80 and above in the near future. The researchers describe it as "aging of the elderly." Worldwide, the elderly are the fastest-growing population group. By the year 2050, it is expected that the percentage of the elderly will surpass that of children. Economic growth and rising education levels have some consequences, such as significant disruption in the children-to-elderly ratio, particularly in Western Europe and the United States [1,2].

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The situation in Romania aligns with the global aging trend, especially in the past two decades. In 2018, the country's population reached 22,194,000 citizens. The phenomenon of demographic aging has increased, with the elderly population aged 65 years and older exceeding the population aged 0–14 years by 350,000 people, according to the National Institute of Statistics (INS). Life expectancy approximately doubled, from 36.4 years at the beginning of the 20th century to 75 years by 2015. Romania ranks 67th in terms of life expectancy, with Monaco in 1st place, and Angola and Sierra Leone in last place (source: WHO, 2016).

In Romania, we are facing a decrease in the population aged 0–14 years and an increase in the population over 65 years old; this aspect is attributed to the decrease in the birth rate, rising economic levels, and improvements in the quality of healthcare services. This gap between the two groups is found especially in rural areas due to migration toward urban areas and outside the country (about 10% of Romania's population has emigrated, particularly following the country's accession to the European Union). This phenomenon also has an impact on the birth rate, with most of the children of those who emigrated being born outside the country, especially in Germany, Spain and Italy. Based on these data, it is not difficult to conclude that in about three decades, the Romanian medical system is likely to face a real problem regarding treatment for the elderly (according to the INS, as of January 1, 2018, the largest age group in Romania was composed of those aged 45–49 years) [3,4].

INTRINSIC SKIN AGING

Intrinsic, or endogenous, aging results from the sum of two major factors. The first involves a slow and gradual decline in physiological cellular functions; it has individual variations and is genetically determined. On top of these gradually accumulated changes sits the reduction in the activity of the endocrine glands, especially involving the gonads. Skin aging due to endogenous factors is a slow and inevitable process, unlike extrinsic aging, which can be significantly delayed by avoiding the factors involved in its genesis, such as exposure to the sun [5].

The genetic component has an important influence on the skin aging process. Thus, gender, ethnicity, and individual variations in the composition of the hydrolipidic film bring significant differences in the degree of skin aging. Women have lower amounts of dermal collagen compared to men; these levels remain constant up until menopause, after which a sudden and marked reduction is observed. In men, collagen decreases gradually by 1% per year. This steady decline also affects other skin functions such as elasticity and hydration. While men experience chronological aging directly proportional to age, in women there is a sudden onset of these skin manifestations based on the estrogen deficiency induced by menopause. Ethnicity plays an important role in the clinical degree of chronological cutaneous aging. This is directly related to the level of skin pigmentation, leading to different responses to ultraviolet radiation. Thus, the elderly with Fitzpatrick phototype VI (African Americans) do not show significant differences between extrinsic and endogenous skin aging due to the protective role of high pigmentation. There are large differences in skin morphology and physiology between Caucasians, Asians, and African Americans [6,7].

Endocrine factors play an important role in the aging process of the human body. Menopause is the period characterized by the permanent loss of ovarian function (meno = month in Greek, pause = stop). It represents the transition from the cyclical, monthly secretion of estradiol to a constant and diminished hormonal output. It is a process that sets in gradually, being preceded by a period with polymorphous clinical manifestations called perimenopause or climacteric. These changes are followed by an increased risk of developing cardiovascular disease, osteoporosis, and cognitive impairment. The onset of menopause significantly impacts the skin aging process. Menopause leads to a significant acceleration in skin aging due to a 30% reduction in dermal collagen during the first 5 years after its onset. Estrogen deficiency predisposes to cutaneous xerosis and accentuation of wrinkles, as well as to a reduction in mechanical function, which is clinically observed in the appearance of bruises after minimal trauma. Cutaneous xerosis is caused by a decrease in epidermal hydration, as well as a reduction in the skin's barrier function. Androgenic alopecia can be found in menopausal women and has clinical aspects similar to those found in men. It manifests as hair thinning in the frontal and parietal regions. Unlike in men, androgenic alopecia in menopausal women rarely becomes complete and is frequently associated with hirsutism. Another manifestation linked to menopause is frontal fibrosing alopecia, characterized by the loss of hair in the frontotemporal regions and a fibrotic aspect of the underlying skin. This condition is permanent and irreversible [8,9]. Andropause sets in slowly after the age of 40. The signs are usually subtle and are caused by reduced levels of free testosterone. Clinical manifestations include decreased libido, bone and muscle fragility, and sleep disorders. Reduced testosterone levels correlate with reduced skin elasticity and the formation of wrinkles [10].

EXTRINSIC SKIN AGING

Ultraviolet radiation (UVR) was discovered in 1801 by Johann Wilhelm Ritter. The first correlations between exposure to UVR and the acceleration of skin aging were made by Unna in 1894. Thirteen years later, Dubreuilh made the first observation linking sun exposure to the development of skin cancer (1907). UVR is absorbed into the skin through high-affinity molecules called chromophores. Following absorption, photochemical reactions occur at the cellular level. The main chromophores for UVR in the skin include melanin, beta-carotene, proteins, 7-dehydrocholesterol, protoporphyrin IX, and nuclear DNA. These chromophores absorb radiation depending on its wavelength; for example, nuclear DNA, proteins, and protoporphyrin IX absorb predominantly ultraviolet B (UVB), while beta-carotene and 7-dehydrocholesterol have an affinity for the absorption of ultraviolet A (UVA). Melanin acts as a chromophore for both

UVA and UVB. The absorption of UVR initiates photochemical reactions that are central to the pathophysiological mechanisms involved in acute effects (such as sunburn) or chronic conditions (such as aging and skin cancer) [11]. Exposure to UVR leads to the formation of reactive oxygen species (ROS) that cause oxidative stress, inflammation, DNA lysis or decreased DNA repair capacity, as well as malignant transformation at the cellular level. Although the skin has its own defense mechanisms against these molecules (antioxidant enzymes), chronic exposure to UVR disrupts the balance between aggressors and protective mechanisms. This type of aggression can be quantified by some laboratory techniques such as chemiluminescence, photon emission detection or immunohistochemistry. Immunohistochemical analysis used to evaluate the oxidative stress induced by ROS focuses on measuring the expression of 8-hydroxy-2-deoxyguanosine (8-OHdG), a compound produced by the interaction between the hydroxyl radical (-OH) and the skin [11,12]. Bernerd highlights the fact that UVB has a harmful effect on the epidermis, while UVA radiation reaches deeper layers and alters dermal structures by quantifying pyrimidine dimers that behave as chromophores for ultraviolet radiation at the skin level. Detection of photon emission seems to be a reliable method for assessing the cellular stress induced by ROS [13]. Khabiri investigated these effects by measuring the emission of photons resulting from the oxidation of amino acids such as tryptophan, cysteine, phenylalanine, lysine, or threonine. These amino acids emit photons at various intensities when exposed to ROS [14].

Air pollution has serious consequences for the skin, including an accelerated aging process, delayed wound healing, the appearance of precancerous and/or malignant lesions and, last but not least, the triggering of dermatological diseases in predisposed individuals (psoriasis, atopic dermatitis). The skin is the second organ through which the pollutants can penetrate into the body after the respiratory system. Air pollution exerts its harmful effects on the skin system through ozone (O3). Although ozone has no harmful effects per se, its interaction with substances resulting from industrial processes leads to the formation of ROS. O3 is a pale blue, unstable gas with a sharp smell detectable at concentrations of 0.01 ppb (parts per billion). It has great oxidative properties and is normally found in the stratosphere (12-55 km above the ground), where it forms the "air filter" with a protective role against UVR. On the other hand, its presence in the troposphere becomes toxic to living organisms. In highly industrialized areas, ozone concentrations can exceed 1000 ppb (normally the tropospheric concentration is approximately 80 ppb), particularly during the summer months when UVR levels are elevated. It is estimated that during the 21st century the concentration of O3 will increase about fivefold, due to the large number of cars as well as urban crowding. O3 reduces the number of antioxidants present in the epidermis, destroys the lipid film, and activates collagenase-like proteins. The skin's defense mechanisms against these harmful effects include antioxidants (vitamins E and C, glutathione, ubiquinol) and enzymes (peroxidases, superoxide dismutase and catalase). However, the distribution of these substances is not uniform throughout the epidermis, so they are found in lower concentrations at the most superficial layer of the skin, partly because the area is directly exposed to the environment, and partly due to the epidermal turnover. O3 has a pro-inflammatory effect on the skin by activating the nuclear factor kappa B (NF-κB), which determines the expression of pro-inflammatory molecules such as IL-8, TNF-α, as well as adhesion molecules (ICAM, VCAM). Additionally, it also leads to the activation of cyclooxygenase-2 (COX2). It has been demonstrated that these pro-inflammatory molecules can be inhibited by topical application of antioxidants (tocopherol) in both laboratory animals and human subjects. In the deeper layers of the epidermis, ozone activates heat shock proteins (HSP) 32, 27, 70 that are involved in apoptosis, cell proliferation and inflammation. O3 also has effects on the extracellular matrix by activating metalloproteinases (MMPs) 2, 9, which are responsible for the degradation of the extracellular components of the skin. These changes occur both through increasing the expression of these enzymes and decreasing their inhibitors (TIMPs). It seems that the effects of O3 are amplified when acting on previously sun-damaged skin; as a result, wound healing in elderly people is delayed. In summary, O3 accelerates the aging process by disrupting the lipid film, degrading extracellular components, decreasing antioxidants, and inducing pathophysiological mechanisms such as inflammation, apoptosis, and cell proliferation. While UVR affects rural populations with chronic occupational sun exposure, ozone exerts its harmful effects on people from urban and highly industrialized areas. To date, the negative impact of O3 can be decreased by therapeutic or preventive measures such as topical application of antioxidants, especially vitamin E, as well as a diet rich in fruits and vegetables [15–18].

Smoking is believed to have a negative influence on the skin aging process. The role of smoking is still debated, as the impact of this habit on the skin system is most likely related to a multitude of other factors such as age, sex, genetic predisposition, phototype, as well as cofactors: sun exposure, alcohol consumption, stress, or chronic organic diseases. The primary molecular mechanism by which cigarette smoke accelerates skin aging is represented by the increased expression of metalloproteinases (MMPs) 1, 3, 7. MMP 1 is responsible for collagen degradation, while MMPs 3 and 7 have a role in decreasing elastin and proteoglycans. This leads to damage to extracellular matrix components, especially collagen, elastin, and proteoglycans [19,20]. Yin and colleagues demonstrated that fibroblasts exposed to cigarette smoke have reduced synthesis of collagen and its precursors, procollagen I and III. Cigarette smoke directly induces dehydration of the epidermis, which promotes the formation of wrinkles. Smoking is also associated with the development of cutaneous elastosis and telangiectasia [21]. Studies on identical twins with the same lifestyle have shown the influence of smoking on the aging process after a history of more than 20 years of smoking, with more visible clinical signs in men [22,23]. Daniell observed a particular morphology and distribution of wrinkles in smokers, with more defined lines on the lateral parts of the eyes, the temples, as well as around the lips, due to the specific movements of the facial muscles during smoking [24]. It is important to highlight that smoking is a cofactor in the skin aging process in the case of chronic sun exposure, the best example being Favre-Racouchot syndrome. The adverse effects of smoking on the skin can be prevented by topical application of vitamin C, which decreases the expression of smoking-induced metalloproteinases in fibroblasts [19,20].

Diet has some impact on the degree of skin aging. Thus, sugar consumption accelerates the skin aging process, particularly by deepening wrinkles and promoting skin elastosis. The effects are linked to the formation of advanced glycation end products (AGEs). Certain cooking methods, such as frying, lead to the formation of large amounts of AGEs [25]. Increased consumption of carbohydrates and animal fats increases the risk of wrinkles and skin atrophy. Nutrition also plays a role in developing non-melanocytic skin cancer; therefore, increasing the intake of fruits and vegetables containing vitamins C and E and glutathione significantly reduces the risk of squamous cell carcinoma [26]. Similarly, selenium, zinc, and green tea extract have been shown to be effective in preventing skin tumors, most likely through their photoprotective effects [27]. Other compounds that can reduce the risk of skin cancer, as well as slow down the skin aging process, include tomatoes, curcumin, beta-carotene and genistein. These substances act by reducing inflammation, oxidative stress, and degradation of nuclear DNA. A vitamin C-rich diet significantly reduces the risk of wrinkles [28]. Interestingly, studies carried out on isolated substances have failed to demonstrate significant preventive benefits regarding skin cancer or aging. Only a diet based on combinations of antioxidant and photoprotective nutrients is effective, probably because these substances act through multiple different mechanisms [29].

Alcohol consumption could be a precipitating factor of skin aging, but data from the scientific literature remain limited. Twin studies describe a more youthful appearance in those who have not consumed alcohol during their lifetime without specifying a direct link between the quantity of alcohol consumed and the degree of aging [30]. Most likely, alcohol consumption works as a cofactor often associated with smoking, psychosocial events, and even occupational sun exposure.

Psychosocial events with a negative emotional impact play an important role in accelerating the skin aging process. Direct correlations have been made with experiences such as divorce, death of the spouse, financial situation, or depression [31].

MOLECULAR AND MORPHOLOGICAL ASPECTS

There are clear differences between the two types of skin aging, intrinsic and extrinsic. These differences arise from distinct etiological factors involved in their genesis. Thus, the photoexposed skin will develop changes due to the chronic effects of the sun, pollutants and behavior (diet, smoking, alcohol consumption). By contrast, the photoprotected skin will develop slow changes due to a physiological decrease in homeostasis and endocrine factors. The sun-exposed skin in elderly people shows specific changes in the epidermis and dermis, as well as the selective reduction of subcutaneous fat in areas such as the face, the backs of the hands and the anterior calves. The upper part of the epidermis frequently displays a compact appearance or contains vesicles with fibrillar protein material, along with the disappearance of the transition zone. The number of melanosomes in the basal layer is increased. The dermo-epidermal junction tends to flatten. Dysplastic changes, vacuolization and necrosis can occur within the squamous layer, accompanied by a reduction in the number of Langerhans cells. The papillary dermis develops elastotic changes with the formation of amorphous material, fragmentation of collagen fibers, along with a decreased number of fibroblasts. Aging of the photoprotected skin is characterized by epidermal atrophy, a flattened basement membrane and decreased melanocytes. At the dermal level, there is a noticeable reduction and degeneration of elastic fibers and a slight decrease in proteoglycans and blood vessels in the papillary dermis. Collagen synthesis is also reduced due to diminished fibroblast activity. In contrast to sun-exposed skin, photoprotected skin shows no inflammatory infiltrate [32,33].

CLINICAL ASPECTS OF SKIN AGING

Aging skin is clinically notable for its polymorphic manifestations. Skin aging reflects the sum of chronological changes due to chronic exposure to UVR and other exogenous factors such as pollution, smoking and diet. The intensity of the manifestations is generally correlated with extrinsic factors, especially in males, where hormonal changes associated with old age do not have such an obvious clinical impact as in females [34].

Degenerative skin changes

Solar elastosis occurs as a result of the accumulation of degraded elastin fibers under the dermal-epidermal junction, accompanied by disorganized, non-functional collagen. This condition is more evident in smokers, manifests as a thickened and yellowish appearance of the facial skin with deepening of wrinkles, and has a chronic evolution. On the neck, these wrinkles intersect to form rhomboidal areas, often referred to as rhomboidal neck skin or sailor's neck. Other rare manifestations of solar elastosis include elastic nodules of the helix and Raimer's bands on the dorsal surfaces of the forearms [35,36].

Senile or solar comedones occur in the elderly, being located predominantly on the nose and zygomatic region. They are not related to acne or seborrheic syndrome. They appear as open (black) or closed (white) lesions that do not become inflamed, being directly related to aging, sun exposure and smoking. These lesions result from cellular degradation and enlargement of pilosebaceous orifices caused by ROS under the long-term action of ultraviolet rays [37,38]. Favre-Racouchot syndrome is a chronic, asymptomatic dermatosis associated with smoking, long-term sun exposure and male sex. Its incidence of 1.4% is reported in individuals over 50 years of age and it increases to 2.8–5% in patients with chronic kidney disease. It presents as cysts appearing on plaques of solar elastosis and is frequently located on the face, especially in both temporal regions [39]. The lesions require periodic evaluation, and cases of squamous cell carcinoma have been described [40].

Sebaceous hyperplasia (SH) is commonly found on the facial skin of individuals with chronic sun exposure. Typically located in areas such as the forehead, infraorbital region or temporal region, it occurs around the age of 40, its incidence increasing with age. SH appears as centrally umbilicated papules, yellowish in color, with a diameter of about 2-6 mm. Long-term exposure to UVR, advanced age, or immunosuppressive therapy (cyclosporine A, corticosteroid therapy) are considered to be contributing factors. The diagnosis is based on clinical assessment, with dermatoscopy and confocal microscopy playing an important role in differential diagnosis with basal cell carcinoma. SH treatment is indicated, most often, for aesthetic reasons [41,42].

Idiopathic guttate hypomelanosis (IGH) is characteristic of older women, occurring as hypopigmented macules on the forearms and calves. It can also be found in other areas such as the anterior chest or shoulder region. At the molecular level, the condition is associated with atrophy and focal disappearance of melanocytes. From a pathophysiological point of view, a decrease in the melanogenesis process has been identified both in terms of melanin synthesis and its transport to keratinocytes [43]. Recently, it has been described that IGH melanocytes have an increased volume, contain more melanin than normal ones and have reduced dendritic extensions (Rani et al., 2018). These findings clearly suggest an impairment in melanin transportation to keratinocytes, this being the principal mechanism involved in the genesis of IGH lesions. The disease implies only aesthetic risks [44].

Lentigines are benign flat lesions formed by the proliferation of melanocytes induced by UVR action. They appear as pigmented macules that persist during the cold season (unlike ephelides), being located on the face, the backs of the forearms and hands, as well as the posterior thorax. A solitary lesion is called a solar lentigo and should be differentiated from lentiginous melanoma and lentigo maligna [45].

Skin tumors

Seborrheic keratoses are benign skin lesions that are caused by the proliferation of basal epidermal cells (basal cell papilloma). They are asymptomatic and appear predominantly on the seborrheic areas (on the face and posterior thorax), developing after the age of 30 and mainly affecting women. They are never found on the mucous membranes or at the palmoplantar level. Seborrheic keratoses are usually multiple lesions and their number tends to increase with age. Clinically, they appear as pigmented macules or plaques, some with a rough surface. While the lesions are asymptomatic, some patients report pruritus. Their etiology remains uncertain, the only demonstrated cause being skin aging. It is extremely rare that seborrheic keratoses appear before the age of 20 [46,47].

Actinic or solar keratoses are the result of the proliferation of the squamous layer of the epidermis due to chronic exposure to UVR. Histologically, they are characterized by epidermal dysplasia without involvement of the basal layer of the epidermis with hyperkeratosis, nuclear pleomorphism and sometimes with mitoses. Lesions present as erythematous spots or erythematous-squamous plaques, usually less than 2 cm in size. While asymptomatic, some may be associated with mild itching. They are located on the face and back of the hands, less often in other areas, occurring most frequently in patients with Fitzpatrick skin phototypes I and II. The condition begins in the form of erythematous macules that over time enlarge and become covered with adherent scales. Interestingly, spontaneous remission of these lesions has been described [48].

A cutaneous horn is a hyperkeratotic lesion and is considered a precursor to squamous cell carcinoma. It often occurs in elderly patients (over the age of 70) and is located in photoexposed areas such as the face, forearms, decolletage or the back of the hands. The condition is more prevalent in people with skin phototypes I and II, and the risk of malignant transformation into squamous cell epithelioma is higher among male individuals [49,50].

Keratoacanthoma is considered an early form of squamous cell carcinoma, presents as a nodular formation with central ulceration and tends to develop on the face or back of the hands. The risk of progression to squamous cell carcinoma is about 15%. It has a sudden onset and shows rapid growth over a few weeks or months. The treatment of choice is surgical excision, especially to rule out an invasive squamous cell carcinoma [51,52].

Basal cell carcinoma is the most common type of skin cancer [53] and develops from epidermal basal cells or cutaneous adnexal structures, without affecting the mucous membranes. While it is considered to be only locally malignant, histologically it has the characteristics of a cancer such as cell dysplasia, pleomorphic nuclei and atypical mitoses. Clinically, it frequently occurs after the age of 50, especially in people with a light phototype. Chronic exposure to UVR is the primary risk factor for its development [54]. The mechanism by which UVR induces BCC includes inactivation of tumor suppressor genes (e.g., the p53 gene with a role in inducing apoptosis) and changes in DNA manifesting as pyrimidine dimers. Therefore, mutation of the p53 gene is found in more than half of the patients with basal cell epithelioma, regardless of the clinical or histopathological form. UVB also induces mutations in the PTCH1 gene, which controls cell growth and differentiation [55]. BCC is often located on the face and less commonly on the chest or limbs. The lesion begins as a papule, nodule, erythematous plaque or an ulceration covered by a hemorrhagic crust [56].

Squamous cell carcinoma typically develops on chronically irritated or damaged skin, particularly in areas exposed over time to external noxious agents such as UVR, toxic substances, radiotherapy, cigarette smoking, or post-burn scarring. It is highly aggressive, and it spreads via both lymphatic and hematogenous routes. It frequently arises after the age of 65 in people with fair skin, being more common among men (male-to-female ratio of 2-3:1). Unlike BCC, this condition can affect the mucosal and semimucous membranes, frequently affecting the lower lip (in patients with a history of chronic solar cheilitis or heavy smoking). Clinically, it has

polymorphism, being characterized by nodular or ulcerated lesions, sometimes covered by scales and having no tendency for spontaneous resolution. The lesions are frequently located on the head and neck areas. It metastasizes to the visceral organs like the brain, the liver and the lungs [55–57]. Alterations in EGFR/MAPK signaling have also been investigated in cutaneous squamous cell carcinogenesis [58].

Senile angiomas (cherry angiomas, Campbell de Morgan spots) are vascular proliferations with a benign nature that present as asymptomatic papules, predominantly on the trunk. They frequently begin after the age of 30 and have been linked with pregnancy and exposure to pollutants (bromides), as well as genetics. As patients age, they can spread to the limbs, face and scalp. Some clinical variations of the classical form have been described, such as ruby spots and senile hemangioma of the lip. Ruby spots represent small, sometimes palpable vascular proliferations, frequently found on the trunk and limbs among people over 80. Senile angioma of the lip appears as a soft, painless, reddish-purple nodule of the lower lip, more often observed in females. All clinical forms are closely related to chronological aging rather than extrinsic factors [59,60].

Aspects of inflammatory, allergic, autoimmune or idiopathic dermatoses in elderly patients

Pruritus is one of the most common skin symptoms in older patients, with a prevalence of approximately 20%. It can have various causes, ranging from underlying dermatological conditions (e.g., xerosis) and medications to systemic diseases like anemia, thyroid diseases, liver or renal disease and malignancies. Pruritus in the elderly is most often a chronic condition (over six weeks). The approach requires a careful clinical examination, as well as paraclinical and laboratory investigations to identify an underlying cause. Despite investigations, 30% of cases do not have a clear cause, and treatment remains symptomatic with topical antipruritic agents [61,62].

Skin xerosis is clinically important, not only because of the constant association with pruritus, but also because it predisposes to the development of secondary dermatoses (eczema, skin infections). Although xerosis presents with a widespread distribution, it is better outlined on the extremities, especially on the anterior area of the calves, where it exhibits the characteristic appearance of cracked eczema. Exacerbating factors include excessive washing and chronic use of certain medications such as diuretics and antihyperlipidemic drugs. Therapeutic management is focused on preventing dryness and ensuring skin hydration [63,64].

Atopic dermatitis is characterized by the presence of pruritus, xerosis and eczema and is associated with a personal or family history of atopic conditions such as bronchial asthma, allergic rhinitis and a high incidence of food allergies. It is considered a disease of children and adolescents; its appearance in the elderly is not very well defined. When it comes to older patients, atopic dermatitis is more common in males (male-to-female ratio of 3:1). In general, the clinical features resemble those found in adults, in the form of cutaneous xerosis and eczematous areas with lichenification. In most older individuals, the condition is a continuation of the symptoms first experienced in early childhood, adolescence or adulthood [65,66].

Contact dermatitis manifests differently in the elderly, having a milder clinical appearance and an extended duration. It appears as subtle erythematous lesions with a tendency toward lichenification, while bullous forms are rare. The condition can have both allergic and irritative mechanisms, developing in patients with a history of skin xerosis. The most frequent sensitizers described are topical medications and detergents. Elderly people with urinary incontinence frequently develop contact dermatitis of the inguinal area [67].

Granulomatous dermatoses are defined by the presence of a chronic inflammatory infiltrate as part of a delayed-type immune response. The most common skin lesions with granuloma formation are cutaneous sarcoidosis, granuloma annulare, interstitial granulomatous dermatitis and gouty tophi. They are more frequently encountered in the elderly secondary to therapeutic interventions (drug therapy or surgery), systemic diseases (gout, systemic sarcoidosis, diabetes) or even malignancies [68,69].

Drug-induced cutaneous reactions tend to occur more frequently in elderly patients than in other population groups. Certain clinical features are relatively specific, such as eczematous reactions in response to angiotensin-converting enzyme inhibitors and calcium channel blockers or reactions associated with chemotherapy (hand-foot syndrome, flagellate erythema, follicular or acneiform eruptions). Also, lichenoid drug eruption is particularly common in the elderly due to its tendency to occur after prolonged administration of certain medications [68–70].

Psoriasis presents some specific aspects in the elderly. Due to its chronic nature, the disease tends to be at least more pronounced, if not more severe, in the elderly. This can be attributed to a higher risk of developing psoriatic arthropathy, as well as superimposed factors such as comorbidities and use of antihypertensives (beta-blockers, angiotensin-converting enzyme inhibitors) or nonsteroidal anti-inflammatory drugs (NSAIDs). Although the condition appears after the age of 70 years in only 3% of cases, about 10% of patients with psoriasis are older than 70 years and often present with secondary conditions like type II diabetes, hypertension and dyslipidemia. Older adults with psoriasis are more likely to develop certain forms or complications such as inverse psoriasis, guttate, pustular, or erythrodermic psoriasis [71,72]. In a study of 90 patients with psoriasis, hepatic steatosis was present in 58.9% and was more frequent in older patients and those with diabetes [73].

Lichen planus is a chronic inflammatory disease that affects the skin, appendages and mucous membranes. The incidence varies between 0.2 and 5%, being more common among the elderly and female patients. The increased frequency in the elderly is largely

due to the presence of some triggering factors such as viral infection with hepatitis B or C, psychiatric disorders, immunosuppression and certain medications. Specific forms in the elderly include verrucous lichen (associated with chronic venous insufficiency) and oral mucosal lichen planus (favored by various denture materials) [74]. Lichen planopilaris occurs more frequently in older women and manifests as frontal fibrosing alopecia that is linked with postmenopausal estrogen deficiency or Graham-Little-Piccardi-Lassueur syndrome, which combines scarring alopecia of the scalp with follicular rash and non-scarring alopecia of the large folds [75,76]. Lichenoid drug eruptions are more common in older patients due to the use of antihypertensive medication (ACE inhibitors, beta-blockers, diuretics) and nonsteroidal anti-inflammatory drugs (NSAIDs) [77].

Bullous pemphigoid is the most common autoimmune blistering dermatosis in the elderly population. Its incidence increases significantly after the age of 60, with the average age of patients in Europe with bullous pemphigoid varying between 77 and 83 years [78]. Although no direct causal link has been made, the increase in the number of cases among the elderly is associated with other comorbidities. Thus, most patients with bullous pemphigoid suffer from diabetes mellitus or neurological diseases such as dementia, Parkinson's disease or multiple sclerosis, or have a history of stroke. Neurological pathology is present in 30% of patients with bullous pemphigoid. Many cases of localized bullous pemphigoid begin in hemiplegic areas. Additionally, orthopedic prostheses are reported in 25% of cases. The disease has an increased incidence in patients with bipolar psychiatric disorders [79]. The role of certain medications such as spironolactone and neurological drugs has also been discussed [80].

Aging of the mucous and semi-mucous membranes

Like the skin, the mucous membranes suffer a decrease in wound healing ability as part of the aging process. The risk of oral mucosal lesions of any type in the elderly increases significantly when the patient also has diabetes, smokes, or has poorly fitted or morphologically inadequate dental prostheses. The most common changes are xerostomia and sublingual varicosity.

Stomatitis due to dental prostheses occurs in 10% of the elderly, with squamous cell carcinoma being the most common tumor of the oral cavity. Angular cheilitis is commonly encountered in the elderly and its appearance is influenced by several factors such as the deepening of commissural wrinkles, weakening of the facial muscles, as well as alterations in the dental apparatus. Actinic cheilitis occurs more frequently in men and is mainly caused by chronic exposure to UVR, but also by smoking.

In the genital mucosa, atrophy appears with age, particularly in women. Atrophy of the vulva and vagina correlates with postmenopausal estrogen deficiency. There is also a higher incidence of urinary incontinence and perineal dermatitis in women. Genital lichen sclerosus et atrophicus is the most frequent cause of circumcision in older men [81–83].

Nail aging

Due to keratinocyte turnover disorders as well as decreased blood flow in the nail bed, the elderly develop certain dystrophic nail changes. Changes in coloration of the nail plate that mimic onychomycosis are frequently encountered. The nails become thickened and dull in appearance and sometimes onychogryphosis can be present. Changes associated with medication or repeated trauma may also be encountered in the context of distal sensory impairment. With age, nails lose their length and become more convex. Their resistance decreases, and they become friable, especially toward the distal ends. A particular manifestation seen in senile nails is the arrangement of three horizontal bands, which are white, pink and opaque from proximal to distal. Other features associated with age are localized hypertrophy of the nail (onychauxis), subungual clavus formation, and an increase in subungual hemorrhages and hematomas compared to the young population. These dystrophic changes contribute to a higher incidence of onycholysis [84,85].

Clinical aspects of hair aging

Hair aging involves two aspects: physiological canities (graying) and diffuse alopecia (hair thinning). Men with androgenic alopecia have an earlier tendency to exhibit graying. It is a gradual physiological process that frequently begins after the age of 35, caused by a reduction in the melanogenesis process. So far, canities remains an irreversible condition. Diffuse alopecia of the elderly occurs after the age of 60 and is linked especially to hormonal status. In women, the process is slower and becomes noticeable at a more advanced age. In men, hypertrichosis can be observed in the eyebrows and external auditory canal. Furthermore, hair density on the limbs decreases with age in both sexes [86,87].

Chronic cutaneous insufficiency or dermatoporosis

We are witnessing an increase in the average life expectancy in developing countries. This aspect has amplified both intrinsic and extrinsic factors involved in the skin aging process. Up until recently, we could not discuss chronic organ insufficiency when it comes to the skin, but the term chronic cutaneous insufficiency or fragility has emerged in recent years. Kaya and Saurat were the first to introduce the term dermatoporosis in 2007, describing it as the association between senile purpura and pseudoscars that developed on atrophic skin of the elderly [88]. Injuries occur as a result of minimal trauma caused by decreased mechanical function of the skin. However, the clinical features of senile purpura had been described as early as two centuries ago by Thomas Bateman, while Colomb described the stellate pseudoscars on the forearms in the elderly in 1967 [88–90]. The main pathophysiological mechanism is based on the reduction in extracellular matrix components, particularly hyaluronic acid (HA) and its main receptor, CD44 [88]. Based on this phenomenon, viscoelasticity and skin hydration decline [91]. Clinical changes include skin atrophy, purpura, pseudoscars,

hyperpigmentation and skin ulcerations. These clinical aspects lead to an increased risk of developing deep dissecting hematomas (DDHs) due to the accumulation of blood between the hypodermis and the muscular fascia after minimal trauma [88,92]. The lesions are mainly located on the forearms and calves [88]. Other locations can include the presternal region and the dorsum of the hands, as well as the face and scalp in some isolated cases [93,94].

The prevention of dermatoporosis lesions begins with eliminating or reducing the patient's exposure to the triggering factor. Thus, it is necessary to use photoprotection strategies and avoid trauma in patients with primary dermatoporosis. In cases of secondary dermatoporosis, prevention may be achieved by reducing the dose and/or duration of corticosteroid therapy [88,94].

The first therapeutic measures were developed by Kaya in 2007, along with the initial description of the disease. Topical application of 1% hyaluronic acid leads to improvement of the lesions [88]. Topical retinoids have proven effective in the treatment of dermatoporosis, and their role is to restore the cellular differentiation process. Combining hyaluronic acid and retinaldehyde is more effective than the separate application of either compound [93]. Alpha-hydroxy acids (AHAs) work in reducing the degree of skin atrophy [95]. Humbert et al. claim that dermatoporosis, especially Bateman's senile purpura, is related to vitamin C deficiency in the skin, and they demonstrate a reduction in lesions following topical application of 5% vitamin C [96]. Kluger suggests that systemic administration of vitamin C is also effective [97]. There are limited data on the efficacy of topical applications of epidermal growth factor or dehydroepiandrosterone [98,99].

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Authors' contribution

Conceptualization, MAB and MLS; methodology, BAM; software, BAI; validation, DOC, ACC and MAB; formal analysis, ABG; investigation, MAB; resources, MAB; data curation, DOC; writing—original draft preparation, MAB; writing—review and editing, MAB; visualization, DOC and ACC; supervision, DOC and MLS. All authors have read and agreed to the published version of the manuscript.

Ethics approval and consent to participate

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