Chronic Pruritus in the Geriatric Population

Taige Cao, MD[a], Hong Liang Tey, MD[a,b,*], Gil Yosipovitch, MD[c]

KEYWORDS

• Pruritus • Geriatrics • Therapeutics • Diagnosis • Classification

KEY POINTS

- Chronic pruritus in the geriatric population is defined by pruritus that persists for more than 6 weeks experienced by people 65 year old and above.
- Aging is associated with pathologic changes is the epidermal barrier, immune system, and the nervous system that predispose the elderly to itch.
- In our approach to the clinical problem, we dichotomize pruritus based on its pathogenesis, either histaminergic or predominantly nonhistaminergic.
- Topical treatments are generally safe for the elderly population; systemic treatments are chosen depending on the condition with consideration of comorbid diseases and drug interactions.
- Numerous new medications are currently undergoing clinical trials and they are anticipated to enter the clinics in the near future.

INTRODUCTION

Chronic pruritus in the geriatric population, as defined by pruritus that persists for more than 6 weeks experienced by people 65 years old and above, is an increasing health care problem.[1] Medical advances, decreasing fertility rates, and longer life expectancies have given rise to a rapidly aging population, especially in developed countries. In 2017, there are an estimated 962 million people aged 60 or over in the world, comprising 13% of the global population. The population aged 60 or above is growing at a rate of about 3% per year. By 2050, all regions of the world except Africa will have nearly one-quarter or more of their populations at ages 60 and above. The number of older persons in the world is projected to be 1.4 billion in 2030 and 2.1 billion in 2050.[2] By then, geriatric conditions will impose the greatest strain on global health.

It is believed that the incidence of pruritus increases with age; however, there is a dearth of well-designed epidemiologic research to establish this. Depending on region and sample size, the prevalence of itch in the elderly was estimated to be between 11.5% and 41.0%.[3] A working population survey found that the prevalence of current chronic pruritus increased with age from 12.3% (16–30 years) to 20.3% (61–70 years).[4] A cross-sectional study of 302 geriatric patients in the Hispanic population in Mexico demonstrated the prevalence of chronic itch was 25%.[5] In another study of 68 noninstitutionalized persons, two-thirds of the group and 83% of octogenarians reported medical concerns regarding their skin, with pruritus being the most frequent complaint.[6] In a study conducted in Thailand involving 149 elderly patients, pruritic diseases were the most commonly reported disease (41%), of which xerosis (synonymous with senescent itch) was the most frequent (38.9%).[7]

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[a] National Skin Centre, 1 Mandalay Road, Singapore 308205, Singapore; [b] Lee Kong Chian School of Medicine, Nanyang Technological University, 11 Mandalay Road, Singapore 308232, Singapore; [c] Department of Dermatology and Cutaneous Surgery, Itch Center Miller School of Medicine, 1295 NW 14th Street, University of Miami Hospital South Building Suites K-M, Miami, FL 33125, USA

* Corresponding author. National Skin Centre, 1 Mandalay Road, Singapore 308205, Singapore.
E-mail address: teyhongliang111@yahoo.com

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The consequences of chronic pruritus are especially significant in the elderly population, which include a decreased quality of life, diminished mental health, and an impact on the health care economy. A recent German study by Makrantonaki and collegues[8] on 110 hospitalized geriatric patients revealed that pruritus showed a positive correlation with the duration of hospitalization and a negative correlation with the Barthel index and Tinetti score on the day of discharge. The latter indicates that pruritus has a significant impact on the physical condition and on the static and dynamic balance abilities of the elderly with multiple morbidities, respectively.[8] Insomnia and depression can result, and this condition is especially worse in the elderly with interrupted sleep.[9] In addition, pruritus can be aggravated by the complex psychosocial factors that are often present in the elderly.[10]

PATHOPHYSIOLOGY OF ITCH IN THE ELDERLY

Aging is associated with pathologic changes is the epidermal barrier, immune system, and nervous system, which predisposes the elderly to itch.

Epidermal Barrier

The epidermal barrier of the skin deteriorates with aging. The epidermal barrier is formed in the stratum corneum with corneal cells and intercellular lipids, forming a watertight configuration.[11] Xerosis of aging skin is not only caused by deficient sebum production, but also by a complex dysfunction of the stratum corneum. With aging, there are decreased skin surface lipids, increased transepidermal water loss, and decreased corneal hydration, which ultimately contributes to xerosis.[12] Aquaphorin-3 (AQP3), a membrane channel, allows the passage of glycerol and water. *AQP3* gene expression was found to be significantly reduced in the skin of people aged 60 years and over.[13] A reduction of facilitated water permeability and glycerol transport translates to a decrease in epidermal and stratum corneum hydration and glycerol content, leading to xerosis. Sweat and sebum production are also reduced and barrier repair is diminished.[14,15] Elderly patients with pruritus are found to have clinically drier skin than age- and sex-matched control subjects. Xerosis may be causally implicated in up to 38% of generalized pruritus.[7] This finding is further supported by another study demonstrating that, although 69% of patients with itch presented with xerosis, only 18% of patients without itch presented with xerosis ($P<.001$).[5]

The decrease in skin surface lipids has been attributed to the increase in epidermal surface pH, which leads to reduced activity of lipid-forming enzymes and production of ceramide in the stratum corneum.[16] The decrease in the lipid formation capacity and fluid loss affect the epidermal barrier function and could contribute to pruritus of advanced aging. A study revealed that intracorneal cohesion is increased in pruritus of advanced aging, similar to that observed in dermatoses such as psoriasis, ichthyosis, and atopic eczema.[17] This feature predisposes the elderly to the development of other skin conditions, such as contact dermatitis.

Immune System

The age-associated alteration in systemic immunity with aging is referred to as immunosenescence. It is characterized by both a decrease in cell-mediated immune function and humoral immune responses.[18] Immunosenescence consists of defects within the innate immune system with a pathologic shift toward proinflammatory activity with an allergic phenotype. This dysregulation is hallmarked by a change toward type 2 dominance (Th2), with chronically increased levels of interleukin-6 and tumor necrosis factor-α.[19] It has been proposed that, during the aging process, there is a thymic involution with a loss of naive T cells, a decrease in T-cell regeneration, and a decrease in T-cell receptors. Impaired T-cell regulatory function leads to the change to Th2 dominance. Th2 dominance, in turn, may possibly predispose elderly individuals to allergic and mast cell disorders,[20] which in turn result in itch. In a recent report of 4 patients with chronic idiopathic pruritus, there was a decrease in serum immunoglobulin (Ig)G level, but not IgA or IgM levels. It has been suggested that the B cell defect could be considered a form of common variable immunodeficiency, acquired as a consequence of the aging process.[21]

It has also been proposed that there can be an autoimmune component to the pruritus of advanced aging. Aging is associated with a loss of self-tolerance against cutaneous autoantigens.[22] Macrophages are affected by aging, resulting in a compromise of the inductive phase of the immune response and phagocytic capabilities.[23] An example is bullous pemphigoid, in which patients can have generalized itch for years without blistering. Pruritus may be the consequence of antibodies developing against the basement membrane zone antigens.[24]

Nervous System

Aging also affects the nervous system and predisposes one to pruritus. Although no significant