

Contact Dermatitis in Older Adults

A Review of the Literature

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Abstract

Contact dermatitis is a significant health problem affecting the elderly. Impaired epidermal barrier function and delayed cutaneous recovery after insult enhances susceptibility to both irritants and allergens. Exposure to more numerous potential sensitizers and for greater durations influences the rate of allergic contact dermatitis in this population. Medical co-morbidities, including stasis dermatitis and venous ulcerations, further exacerbate this clinical picture. However, while these factors tend to increase the degree of sensitization in the elderly, waning immunity can actually decrease such a propensity. This interplay of both intrinsic and extrinsic factors makes a generalization on trends for contact dermatitis in older adults challenging. The literature has varying reports on the overall incidence of allergic contact dermatitis with advancing age. Nevertheless, it does clearly show that sensitivity to topical medicaments increases with age. Irritant contact dermatitis studies are more consistent, with less reactivity (to irritants) in older compared with younger skin.

Diagnosis of both irritant and allergic contact dermatitis is based on a thorough history, complete skin examination, and comprehensive patch testing. The mainstay of therapy is avoidance of the offending chemical substances and the use of topical along with systemic therapies, depending on the severity of the condition.

In the US, the sector of the population aged 55 years or more is expected to double between 2000 and 2040. It is anticipated that there will be approximately 63.4 million people in this age

group, representing 31% of the total population.^[1] As a corollary, medical expenditures will also rise, including those related to dermatologic conditions such as contact dermatitis.^[1,2]

1. Definition of Contact Dermatitis

Contact dermatitis is defined as inflammation of the skin, resulting in a constellation of clinical findings, produced by the direct interaction of the skin with a chemical substance. This condition can be further subdivided into two major pathways: irritant or allergic. Irritant contact dermatitis predominates, accounting for approximately 80% of all contact dermatitis cases, while allergic contact dermatitis accounts for the remaining 20%. Over 4000 chemical agents are known to be capable of inducing an allergic contact dermatitis reaction, and that number is several times higher for irritant reactions.^[3] In addition, many allergens can also serve as irritants and, similarly, chemical irritants can sometimes produce sensitizing allergic reactions.^[4,5]

2. Allergic and Irritant Contact Dermatitis: Distinguishing and Predisposing Factors

The fundamental difference between the two forms of contact dermatitis is also the basis of their pathogenesis: immunologic versus non-immunologic mediation. An allergic reaction is specific to the inducing agent, requires sensitization, and only occurs in a genetically determined host capable of being sensitized to that given antigen. In contrast, an irritant reaction is nonspecific, does not require prior sensitization, and can occur in anyone. However, individual susceptibility to irritants varies greatly and has recently been shown to also have underlying immune mechanisms, including upregulation and recruitment of chemokine genes, regulated by T-cell effector cytokines.^[6-8] Furthermore, the properties of the contact substance, environmental conditions, and exposure factors, such as concentration, pH, vehicle, temperature, humidity, occlusion, and repetitiveness and duration of contact, also play a role. Finally, genetic factors including polymorphisms in cytokine genes, atopic diathesis, and race, along with other host factors such as age and sex, also contribute to the response to allergens and irritants.^[4,5,9]

3. Clinical Presentation, Symptomatology, and Histology

Despite their differing pathogenesis, allergic contact dermatitis and irritant contact dermatitis may be impossible to distinguish clinically, especially in their chronic forms. Both processes can produce an acute, subacute, and chronic dermatitis. Acute eczematous dermatitis is characterized by erythematous papules with vesicles and weeping. Subacute eczema presents with erythema, scaling, and a serous exudate.

Finally, as the process evolves into a more chronic dermatitis, it is associated with epidermal hyperkeratosis, lichenification, and fissuring. However, high-potency irritants can also produce distinctive clinical findings such as severe skin necrosis with ulceration, sometimes referred to as a chemical burn.

Early in the course of disease, allergic contact dermatitis and irritant contact dermatitis may be differentiated by their symptomatology and histology. Irritant reactions are more likely to be associated with burning and stinging sensations, whereas the *sine qua non* of allergic reactions is pruritus. Also, early irritant reactions are characterized histologically by necrosis of epidermal keratinocytes. Over time, though, both processes reveal similar features: a combination of epidermal spongiosis, acanthosis with mild hypergranulosis and hyperkeratosis, and superficial dermal perivascular lymphocytic infiltrate. The clinical and pathologic overlap in the later phases of these reactions is due to similar patterns of inflammation, including the types of infiltrating inflammatory cells and soluble chemical mediators.^[4]

4. Differential Diagnosis

The differential diagnosis for contact dermatitis is broad. Sub-categorization based on chronology, where the condition is divided by its stage (acute, subacute, or chronic) can be helpful. Early on, inflammatory and bullous conditions such as drug eruptions, viral exanthems, other infectious processes (i.e. erysipelas, impetigo, eczema herpeticum, etc.), bullous pemphigoid, and bullous erythema multiforme should be considered. With time, less inflammatory and more persistent processes such as tinea, psoriasis, Bowen disease, Paget disease, early mycosis fungoides (cutaneous T-cell lymphoma), and secondary syphilis are of diagnostic consideration. Finally, chronic forms of contact dermatitis must be differentiated from lichen simplex chronicus (neurodermatitis) and Norwegian crusted scabies.^[2] The location of the eruption is another useful way of narrowing the differential (table I). These methods of categorization, based upon the stage and anatomic location of the condition, can also be used together.

5. Diagnosis

The diagnosis of contact dermatitis is often made based upon history and pattern of distribution. For example, nickel dermatitis classically presents with well defined eczematous plaques underlying the patient's watch, pant snap, or belt buckle. Rhus dermatitis results in linear lesions in unprotected

Table 1. Differential diagnosis based on location

Location	Differential diagnoses
Scalp/face	Psoriasis
	Seborrheic dermatitis
	Atopic dermatitis
	Tinea faciei
	Dermatomyositis
	Lupus erythematosus
	Herpes simplex
	Impetigo (including bullous form)/erysipelas
	Lichen simplex chronicus
Trunk/extremities	Bowen disease
	Psoriasis
	Seborrheic dermatitis
	Atopic dermatitis
	Asteatotic dermatitis
	Nummular dermatitis
	Stasis dermatitis
	Tinea corporis
	Tinea versicolor
	Dermatomyositis
	Lupus erythematosus
	Viral/bacterial exanthems (including vesicular/bullous forms)
	Lichen simplex chronicus
	Bowen disease
	Early mycosis fungoides (cutaneous T-cell lymphoma)
	Page disease
	Secondary syphilis
	Drug eruptions
	Pityriasis rubra pilaris
	Bullous pemphigoid
	Bullous erythema multiforme
Hands/feet	Psoriasis
	Atopic dermatitis
	Tinea manuum/pedis
	Id reaction
	Secondary syphilis
	Norwegian scabies
	Viral/bacterial exanthems (including vesicular/bullous forms)
	Lichen simplex chronicus
	Bowen disease
	Dyshidrotic eczema
	Bullous pemphigoid
	Bullous erythema multiforme
	Bullous impetigo
Groin	Porphyrria cutanea tarda
	Psoriasis
	Yeast intertrigo
	Tinea corporis/cruris
	Erythrasma
	Herpes simplex
	Lichen simplex chronicus
	Bowen disease
	Page disease
	Lichen planus
	Drug eruption

areas, commonly on the extremities, whereas aerosolized allergens also affect exposed sites, but usually in a more diffuse pattern. Conversely, formaldehyde releasers and dyes in clothing cause reactions in covered sites. Irritant reactions are often most concentrated at the site of exposure but, like allergic-type reactions, can be transferred to distant sites (usually by the hands).

One of the most difficult and sometimes impossible clinical diagnoses is the distinction between irritant and allergic contact dermatitis, especially of the hands and feet. A thorough history is necessary to elucidate potential exposures to both substances. As irritant and allergic contact dermatitis have similar histologic features, a skin biopsy is not useful in differentiating these conditions. However, patch testing, using a standard series of allergens, is often utilized to look for common potential sensitizers. The patch test was introduced to the American Dermatologic Society by Sulzberger and Wise in 1931 and was initially developed by Jadassohn 35 years earlier. Correctly performed, it is the only scientific proof of contact allergy.^[4] More extensive allergen testing (i.e. cosmetic, corticosteroid, textile, metal, plastic, plant series, etc.) is also performed based upon the history and distribution of the dermatitis.

Diagnostic criteria for irritant contact dermatitis, established by Rietschel in 1990,^[10] are also helpful with this diagnostic dilemma. Features suggestive of irritant contact dermatitis include both subjective and objective features. Subjective criteria include onset of symptoms within minutes to hours of exposure; pain and discomfort exceeding itching; onset of dermatitis within 2 weeks of exposure; and many people in an environment similarly affected. Objective criteria include erythema/hyperkeratosis/fissuring predominating over vesicular change; glazed or scalded appearance of epidermis; sharp circumscription without tendency of spread of dermatitis; evidence of gravitational influence (dripping effect); small differences in concentration or contact time produce large differences in skin damage; healing process proceeds without plateau upon removal of exposure; and patch testing is negative.^[10]

6. Age-Related Susceptibility to Contact Dermatitis

6.1 Literature Search Methods

An exhaustive review of the literature was performed using medical search engines including Ovid and PubMed. Keywords such as 'contact dermatitis,' 'allergic contact dermatitis,' 'irritant contact dermatitis,' 'young,' 'elderly,' 'age related,' 'treatment,' and 'trends' were utilized, along with combinations of search terms. The resulting articles were then scrutinized for how the patients were divided in terms of age. We originally hoped to

subdivide the patient population into clearly defined age strata (i.e. <55 years, 55–70 years, and >70 years). However, all of the studies have differing age ranges as detailed in tables II and III. Therefore, the data were collected and presented in table format with age-related trends outlined.

6.2 Alterations in Chronologically Aged Skin

As mentioned in section 2, there are many factors that predispose an individual to developing an irritant and/or allergic contact dermatitis. Chronologically aged skin enhances one's susceptibility to these factors due to impaired epidermal barrier function, delayed recovery after barrier damage, and decreased epidermal lipid synthesis. Stratum corneum lipids, particularly cholesterol but also ceramides and free fatty acids, are decreased in the elderly allowing greater risk for irritant reactions and penetration of potential sensitizers for allergic reactions.^[30] Xerosis, with its resultant cutaneous defects, also compounds potential exposure to irritants and allergens.^[31] Photodamage adds to the inherent delay in recovery time in aged skin. Furthermore, alteration of the epidermal barrier has been shown to prime the inflammatory response, with an associated increase in the concentration of Langerhans cells and cytokine production.^[32]

On the contrary, the density of epidermal Langerhans cells has been shown to not only decrease in aged skin but also to diminish with UV irradiation (photodamage). This depletion of Langerhans cells contributes to a decreased reactivity to both allergens and irritants in the elderly.^[33,34]

6.3 Immune Mechanisms

Alterations in immune mechanisms also contribute to dermatitis in the elderly. A decrease in the density of antigen-

presenting Langerhans cells and the production of proinflammatory cytokines has been shown.^[35] These factors make allergic contact dermatitis less likely and, if present, less inflammatory, in the geriatric population.^[36] However, increased chronologic years equates to both a greater number of exposures and longer exposures to potential sensitizing agents and, thus, an increased risk for allergic contact dermatitis.^[33] Also, aging skin is slower to turn over, preventing the natural sloughing of bound antigens and resulting in greater rates of sensitization.^[2]

6.4 Irritant Contact Dermatitis

The published literature demonstrates decreased reactivity to irritants in older (>65 years) than younger subjects (<30 years) [table II]. This trend was reproducible in several studies with different chemical irritants including sodium lauryl sulfate (sodium laurilsulfate) in different concentrations^[11,12,14,15] and a series of irritants including histamine, dimethyl sulfoxide (DMSO), compound 48/80, chloroform-methanol, lactic acid, and ethyl nicotinate.^[13]

6.5 Allergic Contact Dermatitis

A summary of the literature shows varying reports on whether the incidence of allergic contact dermatitis increases or decreases with advancing age (see table III). Induction of allergic contact sensitization in previously naive subjects is the only method to truly evaluate this condition. Although these types of studies are not conducted today, in the early 1950s, Schwartz^[37] studied the induction of sensitization to dinitrochlorobenzene in previously naive subjects of varying ages. Approximately 50% of the 174 subjects became sensitized, but

Table II. Age differences in susceptibility to irritant contact dermatitis

Study	Design	Findings
Lejman et al. ^[11]	Compared two age groups (18–25 and 65–84 y) for reactivity to a strong irritant, sodium lauryl sulfate (sodium laurilsulfate) 5%	Reaction in older age group was less than that of younger age group
Cua et al. ^[12]	Compared two age groups (mean age 25.9 and 74.6 y) for reaction to sodium lauryl sulfate 0.25%	Reaction in older age group was less than that of younger age group
Grove et al. ^[13]	Examined the response to a variety of irritants (histamine, dimethyl sulfoxide [DMSO], compound 48/80, chloroform-methanol, lactic acid, and ethyl nicotinate) in two age groups (18–30 and 65–75 y)	In all cases, reaction in older age group was less than that of younger age group
Marrakchi and Maibach ^[14]	Compared two age groups (mean age 25.2 and 73.7 y) for reaction to sodium lauryl sulfate 2%	Reaction in older age group was less than that of younger age group
Schwindt et al. ^[15]	Compared two age groups (mean age 27.7 and 69.8 y) for reaction to sodium lauryl sulfate 7.5%	Reaction in older age group was less than that of younger age group

Table III. Age differences in susceptibility to allergic contact dermatitis (ACD)

Study	Design	Findings
Prystowsky et al. ^[16]	Examined the response to four common contact allergens (nickel, neomycin, benzocaine, and ethylenediamine)	No age-related differences in the incidence of allergic patch test reactions
Goh ^[17]	Examined the response to common patch test allergens	Increased incidence of reactions in subjects >40 y vs those <40 y and an even greater trend in those >59 y vs <20 y
Young et al. ^[18]	Examined the incidence of sensitization with individual allergens	Found different patterns based on the allergen: cobalt and nickel reactions greater in those aged <30 y vs those aged >50 y, but the opposite was true for wood tar
Wantke et al. ^[19]	Looked at sensitization rates from birth to >70 y	Patch test reactivity to standard series allergens was comparable in the 0–7, 8–14, and 20–50 y cohorts (40–65%) but dropped in the elderly age group 70–94 y (33–35%)
Uter et al. ^[20]	Analyzed allergic contact sensitivity data for three age groups (≤60, 61–75, ≥76 y)	Majority of allergens in the standard series displayed an age-related increase in sensitization (i.e. fragrance mix, Balsam of Peru, neomycin sulfate, amongst others). A few allergens exhibited a decline in proportion of patients sensitized with age (potassium dichromate, epoxy resin, thiomersal, nickel sulfate, cobalt chloride). Some allergens did not demonstrate a trend in either direction
Tosti et al. ^[21]	Analyzed allergic contact sensitivity data for two age groups (65–74 and >75 y)	Frequency of positive reactions in elderly patients was 43%, with more numerous reactions in the older cohort
Piaserico et al. ^[22]	Compared response patterns to patch testing in elderly subjects (>65 y) and a younger cohort (20–40 y), both with suspected ACD	Overall, prevalence of a positive patch test was significantly lower in the elderly patients compared with the younger adult cohort
Gupta et al. ^[23]	Compared patch test results from 2 separate years (1982 and 1997) to look for trends in the elderly (>65 y)	Frequency of ACD in the elderly was less than in younger patients (in both 1982 and 1997)
Wohrl et al. ^[24]	Analyzed patch test data from 2776 patients using a revised standard series (with 34 allergens) for age- and sex-specific differences	The overall sensitization rate was highest in children aged <10 y (62%) and declined steadily to be lowest in elderly patients aged >70 y (34.9%)
Schafer et al. ^[25]	Analyzed allergic contact sensitivity data for four age groups (28–39, 40–49, 50–59, and 60–78 y)	Overall, found decreasing sensitization with age (43.6%, 42.0%, 36.7%, and 37.9%, respectively; results not statistically significant). Also found a significant decrease for sensitization to nickel but an increase to fragrance mix with age
Green et al. ^[26]	Reviewed patch test results of 4384 patients for medicament allergies	Overall prevalence of allergy to topical medicaments and their constituents was 20.6%. Prevalence increased with age: 14.8% in patients <30 y, 21.3% in those 30–70 y, and 31.9% in patients >70 y
Goh and Ling ^[27]	Analyzed allergic contact sensitivity data for three age groups (50–59, 60–69, and 70–79 y)	No significant difference in the proportion of patients with positive patch test reactions or ACD in the three age groups, but the numbers did seem to trend upward with age (68% vs 75% vs 75% for the former and 32% vs 45% vs 39% for the latter, respectively). There was a significant decrease for sensitization to nickel but an increase to medicament (cloquinoxil mix) and medicament-related allergens (Balsam of Peru, fragrance mix, para-phenylenediamine) with age
Walton et al. ^[28]	Analyzed allergic contact sensitivity data for age distribution	Patch test reactivity steadily declined after the sixth decade of life and was <5% in the eighth decade
Nethercott ^[29]	Analyzed allergic contact sensitivity data for age distribution	Peak prevalence of patch test reactivity seen in the fourth decade (35%) and a steady decline with age, with 8% reactivity in the eighth decade

there was no significant difference in the incidence of sensitization among the three age groups (21–59, 60–79, and >80 years).^[37,38]

As discussed in section 6.3, aged skin shows reduced reactivity secondary to waxing immunity. Nevertheless, increas-

ed years of exposure to more numerous allergens, intrinsic/extrinsic cutaneous factors, and compounding variables (such as stasis dermatitis, venous ulcerations, incontinence, etc.) can result in greater allergen contact and sensitization. Therefore,

although the overall incidence of allergic contact dermatitis varies by report and may decrease with age, specific allergens such as topical medicaments and fragrances are associated with increased rates of sensitization with advancing age (table III).

7. Age-Related Trends in Contact Dermatitis

Allergic contact dermatitis in the elderly displays several trends. The frequency of multiple allergies (defined as three or more allergies) found on patch testing increases with age.^[39] Nickel and topical medicament-related agents, including fragrances, preservatives, antibacterials, and emulsifying agents/emollients, are the most frequent allergens.^[19,26,40,41] High nickel positivity may result from exposure and sensitivity acquired at a young age. However, the prevalence of sensitivity to topical medicaments increases with age, owing to frequent use of these substances and longer periods of exposure. The most frequent individual medicament allergens in the elderly included: fragrance mix (10–12.9%), *Myroxylon pereirae*/Balsam of Peru (8%), neomycin (7–8.8%), gentamicin (6.6%), and lanolin – detected by wool alcohols and Amerchol L-101® (Amerchol Corporation, Edison, NJ, USA) [1–4.5%].^[26,41] Patients are also at risk for sensitization to topical corticosteroids used in treatment. Contact dermatitis is more frequent on the extremities, especially the lower extremities, in older patients, compared with the face and hands in younger patients. This location difference is secondary to medical conditions unique to the elderly, while the younger age group is more likely to be affected by occupational and cosmetic dermatitides.^[27]

8. Reactivity of Chronologically Aged Skin

Studies have shown a 37% prevalence of patch test reactivity in an elderly population without any evidence of dermatitis,^[33] and that patch test reactivity can persist for at least 10 years.^[42] Therefore, it is of utmost importance to assess whether reactions are of current, past, or questionable relevance. Furthermore, elderly patients have been found to have a slower onset of reactivity, less intense reactions, and prolonged recovery with patch testing. An additional reading after 7 days and the interpretation of weak reactions as true positives are important in this population.^[11,22] Irritant reactions in older skin also display these features of delayed reactivity, decreased intensity of reactions, and slower recovery periods compared with younger skin.^[15]

9. Medical Co-Morbidities in Older Adults

There are also special medical considerations in the elderly that make them more prone to contact dermatitis. As discussed in section 7, topical therapy for stasis dermatitis and lower extremity ulcerations dramatically influences the spectrum of allergens found in older populations.^[20,43,44] Uter et al.^[20] stratified patients into three age groups, ≤60, 61–75, and ≥76 years, and found that dermatitis of the legs was most prevalent in the oldest group and that “the number of allergies (against a fixed set of allergens) actually increases almost linearly with the duration of the (lower extremity) ulcer.” Thus, they concluded that allergic contact sensitivity is a function of not only age but age-related co-morbidity.

Other conditions such as xerosis and asteatotic dermatitis can increase exposure and thus sensitivity to topical medicaments. There are also more obscure exposures unique to the elderly detailed in the literature including benzyl alcohol in hearing aid impression material,^[45] acrylic monomer in a denture,^[46] coronary stent materials,^[47] and implant materials for total knee and total hip arthroplasty.^[48,49] Finally, conditions such as urinary/fecal incontinence and ostomies following gastrointestinal or urologic procedures disrupt the natural skin barrier, predisposing to both irritant and allergic dermatitides.^[50,51]

10. Preventative Measures

In Europe, legislative measures have been enacted specifically for the prevention of sensitization and elicitation of nickel dermatitis. The Nickel Directive became law in the EU in January of 2000. Following implementation of this law, nickel content and nickel release from items intended for direct and prolonged contact with the skin such as jewelry and clothing fasteners have been shown to be decreased compared with levels in 1999 (pre-legislation).^[52] Similarly, the Danish Ministry of Environment implemented regulations in 1992 that have been shown to result in decreased nickel sensitization in Danish schoolgirls with pierced ears.^[53] Both of these studies suggest that, with further adaptation of these laws, the risk of sensitization and subsequent allergic contact dermatitis can be dramatically reduced.

11. Treatment

Identifying and avoiding pertinent irritants and allergens is the first line of therapy. As mentioned in section 5, it is often

impossible to differentiate irritant and allergic contact dermatitis and sometimes there is a component of both conditions. Once relevant allergens are determined through comprehensive patch testing and irritants through careful history, patients should be given detailed information on avoidance therapy. Handouts explaining the chemical substance, listing its uses/where it is commonly found, and detailing the various chemical names and potential cross-reactors are very helpful in educating patients. The Contact Allergen Replacement Database (CARD) developed at the Mayo Clinic is useful in helping patients avoid potential allergens.^[54] This Internet-based system provides patients with an individualized list of topical skincare products free of the various allergens and known cross-reactors identified by their specific patch test results. It is important to explain to patients, especially elderly patients whose skin does not have the integrity of their younger counterparts, that it may take as long as 3 months of vigilant allergen avoidance to see any improvement.

If avoidance of irritants and/or allergens is impossible or if the causal agent cannot be ascertained, topical therapy with creams/ointments, corticosteroids, and other immune-modulating therapies may be helpful. Besides providing emollient properties, topical creams and ointments can also serve as artificial barriers preventing the passage of noxious irritants or sensitizing antigens and allowing for epidermal healing. Topical corticosteroids and immune-modulating treatments, such as the calcineurin inhibitors, pimecrolimus and tacrolimus, are often helpful in moderating the extent and intensity of dermatitis. Topical corticosteroids are the mainstay of treatment for both irritant contact dermatitis and allergic contact dermatitis. However, therapy is limited by adverse effects including adrenal suppression, tachyphylaxis, cutaneous atrophy, striae, and telangiectasia.^[55] These latter adverse effects are especially deleterious in chronologically aged skin. The recently introduced calcineurin inhibitors are particularly useful for their corticosteroid-sparing properties, especially when treating chronic dermatitides.^[56] With regard to topical corticosteroids, it is also important for the clinician to be mindful of the potential development of sensitization to these topical agents themselves, especially in the setting of pre-existing dermatitis. Fluorinated corticosteroids (e.g. clobetasol propionate and fluocinonide) are less likely to sensitize and are preferred over non-fluorinated corticosteroids, such as hydrocortisone and hydrocortisone-17-butyrate.^[57] More intensive topical regimens utilizing occlusive wet dressings can also be helpful.

Recalcitrant conditions may require systemic therapy including phototherapy (UVA, UVB, psoralen plus UVA, and Grenz rays), oral glucocorticoids, and oral immunosuppres-

sants (such as azathioprine, cyclosporine [ciclosporin], and mycophenolate mofetil).^[55]

Recently, oral therapy with alitretinoin (9-cis-retinoic acid) has also been found to be beneficial.^[58,59]

Systemic therapy also carries a host of potential adverse effects including the risk of burning and increased cutaneous carcinogenesis with phototherapy and possible sepsis or internal malignancy with oral immunosuppressants. Oral corticosteroids are especially hazardous in the elderly given the myriad of potential adverse reactions, including osteoporosis, osteonecrosis, cataracts, hyperglycemia, hyperlipidemia, weight gain, hypertension, adrenal insufficiency, peptic ulcer disease, infection (including reactivation of tuberculosis), mood changes, and insomnia, amongst others.^[55]

Finally, treatment of secondary impetiginization with topical and/or oral antibacterials is imperative. Compresses using Burow solution (containing aluminum [aluminium] acetate) are helpful in controlling weeping, exudative conditions, and oral antihistamines are often utilized for pruritus control. Strontium salts have also been used for treatment of sensory irritation.^[60,61] Elderly patients should also be educated on proper skin care and avoidance of injudicious use of topical medicaments, which can result in sensitization.^[27]

12. Conclusions

Contact dermatitis is a significant cause of skin disease in older adults. Impaired epidermal barrier function and repair, long history of exposure to numerous potential irritants/allergens, and medical co-morbidities can all increase susceptibility with age. Conversely, waning immunity actually decreases such a propensity. Therefore, a determination of the overall rate of this condition with advancing age is difficult. Reports in the literature for allergic contact dermatitis in the elderly are varied, with some demonstrating an increase while others a decrease in overall incidence. However, specific allergens including topical medicaments and fragrances are associated with increased reaction rates in the elderly in multiple studies. Irritant contact dermatitis trends are more consistent in the literature, with reduced responsiveness in older compared with younger patients. A thorough history, complete skin examination, and extensive patch testing is necessary for diagnosis of both forms of contact dermatitis. Even with these diagnostic measures, it can oftentimes be impossible to distinguish between these processes. Avoidance of the offending chemical substance is the primary treatment, along with topical and systemic therapies, if necessary.

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