Diagnosis and Management of Common Inflammatory Skin Diseases in Older Adults

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KEYWORDS

• Inflammatory • Dermatitis • Psoriasis • Elderly • Geriatric • Skin

KEY POINTS

- Many common dermatoses begin or continue in elderly patients.
- Management considerations in this population need to account for increased likelihood of difficulty with mobility and dexterity, of concurrent medical conditions, and of polypharmacy.
- The primary complaint of inflammatory dermatoses can be pruritis, so a careful history must be obtained to discern the correct diagnosis.

INTRODUCTION

Inflammatory skin conditions affect people of all ages, genders, and races and are frequent causes of visits to the dermatologist. The geriatric population is often afflicted by these conditions because many are chronic and relapsing diseases. Examples include but are not limited to psoriasis, atopic dermatitis (AD), contact dermatitis, rosacea, seborrheic dermatitis, and Grover disease. Chronic inflammatory skin conditions place a large burden on the health care system in the United States and have many associated comorbidities.[1] Herein, we discuss the common inflammatory dermatoses that affect the geriatric population.

RELEVANT CLINICAL QUESTIONS

- When was the onset of the eruption?
- What underlying diseases are present and could be contributing to the eruption?
- Is there an association between the eruption and a certain trigger?
- What type of management is required for this condition based on the associated risks of the condition versus the risks of the treatment options taking into account the patient's comorbidities and current medications?

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- How long is treatment required for the condition?

PSORIASIS

Psoriasis is a chronic inflammatory condition with several subtypes including plaque, guttate, pustular, inverse, and erythrodermic. According to a recent study in 2021, the prevalence of psoriasis of US adults age 20 and older is 3%.[2] Although psoriasis can occur at any age, it most commonly presents before the age of 35 years old.[3] Several studies have shown that psoriasis has a bimodal age of onset with the first peak occurring in the 30s and the second in the early 60s.[4] Kwon and colleagues[5] describes elderly onset psoriasis as those who first develop clinical manifestations of psoriasis at greater than 60 years old. Given the chronicity of the condition, this disease still has a prevalence of 1.23% in the older population from the US Medicare beneficiary database.[2] Associated psoriatic arthritis is debilitating and often hinders ambulation and dexterity for elderly patients. In a study from France, 19.4% of psoriasis patients greater than 70 years old also had psoriatic arthritis.[6]

Clinical Presentation

- Plaque psoriasis is the most common form of psoriasis seen in the elderly population and presents as well-demarcated, thick plaques with silvery scale (**Fig. 1**) most often affecting the extensor surfaces and scalp.
- Plaques can also be seen in the intertriginous regions with maceration and fissuring more often than scaling because of occlusion in these areas.
- Psoriasis also has several associated serious medical comorbidities: psoriatic arthritis, cardiovascular disease, and the metabolic syndrome.

Differential and Diagnosis

- Differentials: Seborrheic dermatitis, AD, nummular eczema, fungal infections, crusted scabies, and secondary syphilis.
- Diagnosis: Often clinical, but skin biopsy is useful in ruling out other conditions.

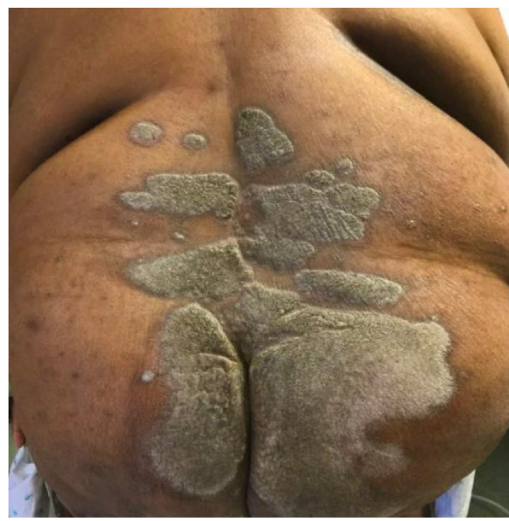

Fig. 1. Well-demarcated plaques on the buttocks of a patient with severe plaque psoriasis.

Pathogenesis

- The cause of psoriasis involves interleukin (IL)-17 and IL-23 and crosstalking between the innate and adaptive immune systems in a feed-forward amplification that leads to the inflammatory cascade occurring in psoriasis.

Treatment

Treatment of psoriasis is individualized to meet the severity of the case and the willingness of the patient to use different therapeutic regimens with the primary goal being to improve quality of life in conjunction with interval improvement in disease status. First-line treatments for limited plaque psoriasis in the elderly are topical agents, such as topical steroids; however, special attention should be given to monitoring of the cutaneous side effects including purpura (Fig. 2), telangiectasia, atrophy, and secondary skin infections. Vitamin D analogues are other topical treatment options (calcipotriene/calcipotriol) that are used alone or in combination with topical steroids. It is also important to assess for any risk factors for hypercalcemia when using vitamin D_3 analogues. Topical retinoids, such as tazarotene, are another option for treatment, but use is often limited by skin irritation. Topical calcineurin inhibitors are beneficial in intertriginous areas. Phototherapy is an effective option that circumvents any drug interactions or side effects but is often cumbersome in the elderly because frequent visits several times weekly are needed and caregivers may need to provide transportation.

Systemic agents are widely effective and convenient options for those with a larger body surface area involved. Methotrexate is a folic acid antagonist that is a traditional first-line medication used in moderate to severe psoriasis.[7] However, extra care should be given to monitoring renal function and hepatic function because the medication is renally excreted and is hepatotoxic and older patients are more likely to have renal insufficiency. Because of this, smaller doses may be required in the geriatric population when compared with younger populations.[8] Dosing is done orally;

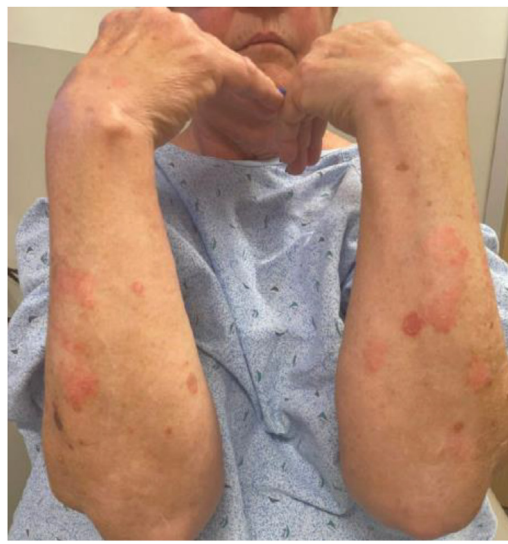

Fig. 2. Patient with psoriasis with focal concurrent purpura, likely partially caused by recent topical steroid use.

however, the intramuscular injection is better tolerated in the geriatric population with difficulty swallowing. A newer systemic agent is apremilast, a phosphodiesterase-4 inhibitor. This is an oral option that is also approved for psoriatic arthritis. It has a known side effect of causing diarrhea and often causes modest weight loss, which is beneficial in the right patient.[9] Apremilast is less immunosuppressive than conventional immunosuppressants or biologic agents and therefore is useful in patients with a history of malignancy or chronic infection. Acitretin is another option that is considered in a patient with a history of malignancy or chronic infection, but it can cause significant xerosis and dyslipidemia and hepatotoxicity, which may be of particular concern in older patients.

The next systemic agents are the biologic medications. In terms of safety profile, biologics typically cause less organ dysfunction as compared with the conventional immunosuppressants, which is beneficial in the geriatric population, but overall, these drugs have been less studied in the elderly population. The biologics are broadly classified by targeted elements of the immune system and are broken down into the tumor necrosis factor (TNF)-α inhibitors, IL-17 inhibitors, IL-23 inhibitors, and IL-12/23 inhibitors.

TNF-α inhibitors were the first biologics to be approved for psoriasis and typically have an onset of action at 12 to 16 weeks.[10] These include etanercept, adalimumab, certolizumab, and infliximab, with the first three being offered as subcutaneous (SQ) injections and the last given intravenously.[11] This class of medications are approved for psoriasis and psoriatic arthritis, but are contraindicated in patients with comorbidities, such as congestive heart failure class III and IV and multiple sclerosis.[12] Although chronic infection is a contraindication, in some circumstances, they are used in patients with HIV, hepatitis B, and hepatitis C infections if the patient is actively treated with concurrent antiviral therapy and if they are being followed by infectious disease specialists.[10,12] IL-12/23 inhibitors include ustekinumab, which is available in an SQ formulation. Full effect of therapy is usually seen after 12 weeks. This class of drugs can also be used in combination with other systemic agents to increase efficacy and the only absolute contraindication to use is history of allergic reaction to the therapeutic agent. Of note, ustekinumab is less effective than the TNF-α inhibitors in treating psoriatic arthritis.

IL-17 inhibitors include secukinumab, ixekizumab, and brodalumab, which are all available in SQ formulations. As a class, the response to therapy is best appreciated after 12 weeks. Efficacy is comparable among the agents in this class.[10] The IL-17 inhibitors are associated with increased mucocutaneous *Candida* infection, and this class should be avoided in patients with a history of irritable bowel disease. Injection site reactions are seen in up to 20% of patients with pruritis at the injection site as the primary complaint.[10] Brodalumab stands alone in that rare cases of suicidal ideation have resulted in a black box warning and brodalumab should not be used in patients with a history of or current suicidal ideation.

IL-23 inhibitors are the last class of biologics that include guselkumab, Risankizumab, and tildrakizumab. Response to the IL-23 class is seen after 12 weeks of therapy and increasing the dose is considered in patients who partially respond. Rare cases of increased liver transaminases have been seen with this class of medications. Temporary discontinuation of the drug is recommended if the patient has a febrile illness, particularly if they are receiving systemic antibiotics.[10] The drug is restarted after symptom resolution.

Newer classes of medications called tyrosine kinase and janus kinase (JAK) inhibitors are emerging as treatment options for psoriasis and psoriatic arthritis. Deucravacitinib, an oral tyrosine kinase-2 inhibitor, is currently the one medication approved for

psoriasis in this class.[13] Because this is a newer medication just approved in Fall of 2022, not all side effects are known. It is known that deucravacitinib may increase the risk of infections with the most common being mild to moderate including upper respiratory infections, herpes simplex infections, and folliculitis.[13,14] Other JAK inhibitors are approved for psoriatic arthritis, but are not yet approved for psoriasis alone. Tofacitinib and upadacitinib are Food and Drug Administration (FDA)-approved for psoriatic arthritis and may be approved for psoriasis in the future. Side effects of JAK inhibitors include increased risk of infections and increased risk of thromboembolic events, which must be considered if using in the geriatric population.[15]

For all biologics and immunomodulators, baseline laboratory studies should include a complete metabolic panel, complete blood count with differential, tuberculosis test (purified protein derivative, Quantiferon Gold, or T-spot), serologic tests for hepatitis B and C, and possible HIV testing depending on physician discretion and risk factors of the patient.[10]

ATOPIC DERMATITIS

AD is another chronic, inflammatory disease that can impact the elderly. Because many older adults see their physicians for dry, flaky skin, diagnosing and treating AD correctly is paramount in this population. There is a degree of age-related skin barrier breakdown and a switch to primarily type 2 helper T cellular responses with age, but this should not exclude proper diagnosis and treatment of AD in the geriatric population. It has been estimated that 2% to 7% of adults have AD.[16,17]

Clinical Presentation

- Adult type AD: Lichenified plaques involving the flexural areas of the extremities, the head, and the neck.
- Elderly type AD: Lichenified plaques more commonly seen affecting the buttocks or genitals. Lesions are less likely to be located within the flexural regions, but may involve the areas surrounding the skinfolds.[18]

Differential and Diagnosis

- Differential diagnosis: Asteatotic dermatitis, nummular eczema, contact dermatitis, cutaneous T-cell lymphoma, and adverse drug eruptions.
- Diagnosis:
  - Often based on the clinical findings of eczematous dermatitis with lichenification that involves flexural regions of the extremities (**Fig. 3**) or chronic eczematous patches involving the face and neck.
  - Positive family history of atopic conditions (asthma, seasonal allergies, AD) or personal elevated total IgE and allergen-specific IgE is supportive of the diagnosis.
  - Skin biopsy is recommended for older patients without a history of prior AD to rule out cutaneous T-cell lymphoma.

Pathogenesis

- The pathophysiology of AD is multifactorial including cellular barrier dysfunction, environmental factors, IgE hypersensitivity, in addition to changes in cell-mediated immune responses.[19]
- Loss of function mutations in filaggrin are implicated in classic AD, but a down-regulation of filaggrin occurs with age adding to pathophysiology of AD in the geriatric population.[17,19]
- Cytokine imbalances that lead to increased expression of IL-4 and IL-13.

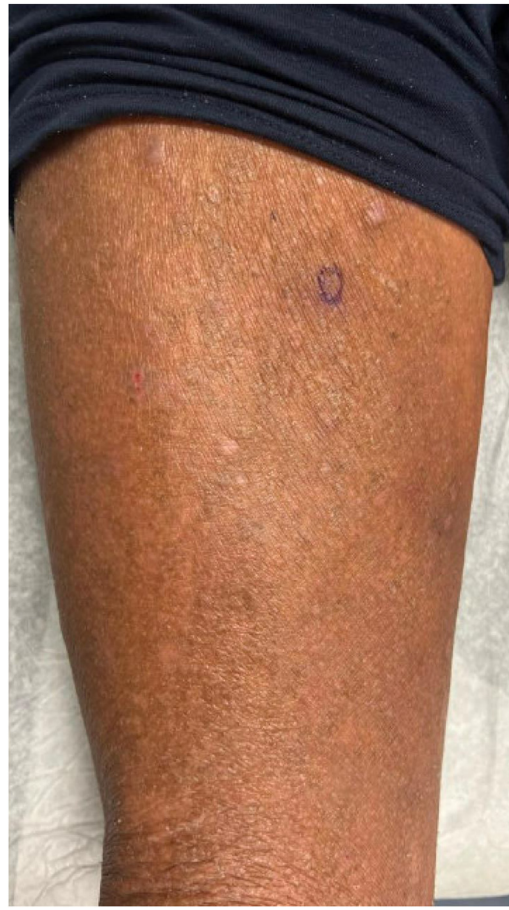

Fig. 3. Thin lichenified papules in xerotic plaques on forearm of patient with atopic dermatitis. Biopsy performed to rule out mycosis fungoides.

Treatment

Initial therapy for AD in the geriatric population should be targeted toward education about the chronicity of the disease, treatments to maintain the skin barrier, avoidance of possible triggers, and anti-inflammatory medications. All patients should be counseled to avoid harsh topical products and to use products that are free of dyes and perfumes. Emollients should be used liberally and frequently to prevent general xerosis of the skin. Topical corticosteroids are first line in AD and should be applied once or twice daily with decreasing steroid potency as the disease status improves.[18] Topical calcineurin inhibitors (tacrolimus and pimecrolimus) are steroid-sparing agents that can be applied to sensitive areas, such as the face, neck, and groin.[20] However, use of these topical steroids or other topical medications may be limited if the patient has a lower self-care ability. Light therapy is viable option for treatment of AD, but logistical complications of transportation can become burdensome in the geriatric population.

Common systemic agents that are recommended for geriatric populations include dupilumab and methotrexate.[18,21] Because randomized controlled trials often exclude

geriatric subjects and those with many complex medical comorbidities the full safety profiles of systemic and biologic agents in older patients are not known.[21,22] Dupilumab is the most commonly recommended first-line systemic agent for geriatric patients with AD and blocks IL-4 and IL-13 signaling.[21,22] It is dosed every 2 weeks and often results in rapid improvement in skin lesions with minimal adverse events. Methotrexate is a treatment option that is available in once-weekly dosing as either oral tablets or SQ injections. This treatment is often referred to as a second-line systemic therapy in geriatric adults after dupilumab and should be started at lower doses with close monitoring.[22]

Newer treatment options include JAK inhibitors, which are available as oral abrocitinib and upadacitinib and topical ruxolitinib.[15,23] The oral options are approved for moderate to severe AD in adults. Risks of the oral JAK inhibitors are still being investigated; however, there is a known increased risk of infection and a potential increase in venous thromboembolism.[15,23] Topical ruxolitinib offers similar efficacy to triamcinolone with less risk of skin atrophy and striae formation, which could be favorable for the geriatric population.[23]

CONTACT DERMATITIS

Contact dermatitis is inflammation of the skin that is a direct result of an interaction of the skin with a chemical substance. Contact dermatitis has two forms, irritant contact dermatitis (ICD) and allergic contact dermatitis (ACD), which are dichotomized by their pathogenesis. Eighty percent of all contact dermatitis is irritant and 20% is allergic.[24] Asteatotic and perineal irritant dermatitis are important common variants of ICD in older patients.[25] For ACD, older literature has found nickel, balsam of Peru, rubber accelerators, topical antibacterials, fragrance mix, and potassium dichromate common allergens in the elderly.[26] More recent data from 2023 have shown fragrance mix, nickel sulfate, and preservatives, such as methylchloroisothiazolinone/methylisothiazolinone, to be among the most commonly encountered allergens in the geriatric population.[27] The same study also found that the frequency of patch test positives was lower in the aging population.[27] Patients with leg ulcers, which occur in more than 1.7% of patients aged 65 and older, have a higher risk of ACD with their sensitization rate increasing with duration of the ulceration.[26]

Clinical Presentation

- Despite different pathogenesis, ACD and ICD are difficult to differentiate clinically.
- Acute phase: Erythematous papules and vesicles, often weeping.
- Subacute phase: Erythematous patches, scaling, serous exudate.
- Chronic phase: Fissures, lichenified plaques, and hyperkeratosis.

Pathogenesis

- ACD: The allergic process requires sensitization to a specific allergen, which is determined by a genetic predisposition. On reexposure to the allergen, the host mounts an immune response.
- ICD: An irritant reaction does not require prior sensitization and can occur at any time. The properties of the substance, such as pH, vehicle, humidity, occlusion, and duration of exposure, all contribute to the reaction.

Differential and Diagnosis

- Differentials vary based on phases

Acute: Impetigo, drug eruptions, herpetic infections, viral exanthems, and urticaria.
- Subacute: Dermatophyte infections, psoriasis, AD, early cutaneous T-cell lymphoma, and squamous cell carcinoma in situ.
- Chronic: Lichen simplex chronicus, psoriasis, nummular eczema, and crusted scabies.

- Diagnosis
  - Diagnosis is usually made on the pattern of distribution and history of the eruptions.
  - Well-defined eczematous plaques that align with belt buckles, zippers, or watches are clinical indicators of ACD, but often distinguishing the ACD from ICD depends on a careful history of exposures and timing of eruptions.
  - Patch testing is useful for ACD and can test for specific allergies.

Treatment

The mainstay of treatment of ACD and ICD is avoidance of the allergen or irritant. For ACD, identification of the allergen is critical and is commonly done through patch testing. In addition to avoidance of triggers, the acute dermatitis is treated with emollients; topical corticosteroids; and other steroid-sparing agents, such as topical calcineurin inhibitors. When using topical steroids in the geriatric population, it is especially important to consider the potency used and to be aware of side effects, such as striae, skin thinning, and telangiectasia. Physicians must also be mindful of which topical corticosteroid is prescribed because they may contain allergens.[28] Above all, avoidance of the allergen or irritant must be at the forefront and patients should be counseled on avoidance practices, such as wearing gloves, minimizing exposure, and using alternative products.

SEBORRHEIC DERMATITIS

Seborrheic dermatitis is a chronic inflammatory condition characterized by pruritic erythematous scaly patches or plaques seen in infancy and adulthood.[29] Although this condition is usually mild, it is often chronic and can reoccur frequently if not kept under control. The prevalence of the condition is estimated to be between 2.35% and 11.3%, with increased frequency in immunocompromised individuals and patients with neurologic conditions.[29]

Clinical Presentation

- Erythematous patches, papules, or plaques with fine or greasy-appearing scaling most often seen in regions with high numbers of sebaceous glands.
- Commonly affected areas: Scalp, face, flexural regions, and chest.

Pathogenesis

- Proposed pathogenesis involves disruption of the skin microbiome and an abnormal immune response to the fungus *Malassezia* sp on the skin.[30]
- It is thought that there is abnormal keratinocyte shedding and increased amount of unsaturated fatty acids in the skin.[31]

Differential and Diagnosis

- Differentials: AD, psoriasis, contact dermatitis, dermatophyte infection, lupus erythematosus, rosacea, pityriasis rosea, Grover disease, and Darier disease.
- Diagnosis

- Seborrheic dermatitis is usually a clinical diagnosis that does not require biopsy.
- Clinically, distribution of lesions is in regions with high number of sebaceous glands, particularly the scalp and face. The pink patches often have greasy scale.
- If considering other differentials, a KOH examination, bacterial swab, serum antinuclear antibodies/extractable nuclear antigen/erythrocyte sedimentation rate, and histology with or without direct immunofluorescence can help rule out other etiologies.[31]

Treatment

Treatment of seborrheic dermatitis depends on the extent and severity of the condition and the desire of the patient to treat this chronic relapsing condition. Treatment rotation is helpful to maintain effectiveness and minimize adverse events. Typical first-line therapies are topical antifungals, such as ketoconazole, miconazole, clotrimazole, ciclopirox olamine, selenium sulfide, or zinc pyrithione. These work to decrease the *Malassezia* burden and reduce inflammation and show similar remission rates to topical corticosteroids but with lower risk of adverse events.[32] The vehicle for the agent selected depends on the area of the body to which it is being applied. Shampoo formulation should be applied to the scalp or body and left on the skin for 5 to 10 minutes before rinsing. Topical corticosteroids of low to mid potencies help mitigate the inflammatory component of seborrheic dermatitis but should be used intermittently to minimize the risk of skin thinning, atrophy, and telangiectasia. The use of steroid-sparing agents, such as calcineurin inhibitors (tacrolimus and pimecrolimus), can also be considered.

Oral systemic agents should only be used in refractory and severe cases. Oral systemic antifungals include itraconazole, fluconazole, and terbinafine.[32] Oral fluconazole is a systemic option that is well tolerated and is used in the elderly, with attention to the risk of the QT prolongation.

ROSACEA

Rosacea is another chronic inflammatory condition that affects 16 million American adults and is estimated to affect 10% of fair-skinned people.[33] Rosacea is typically diagnosed after the age of 30 and is more common in females except for phymatous rosacea, which is more common in men younger than 60 years old.[34] Although more common in Fitzpatrick type I-II, rosacea also affects skin of color.[33]

Clinical Presentation

- Four clinical subtypes of rosacea: Erythematotelangiectatic, papulopustular, phymatous, and ocular.
  - Erythematotelangiectatic: Persistent central facial erythema, with or without telangiectasia and flushing.
  - Papulopustular: Persistent central facial erythema, facial papules, and/or pustules in the central face.
  - Phymatous: Thickening of the skin with resultant surface nodularities and enlargement. Often affects nose, chin, forehead, cheeks, or ears and is most commonly seen in older men.[33]
  - Ocular: Gritty sensation, burning/stinging, dryness and itching of the eye. Often with blurred vision and telangiectasia of the sclera. Can have periorbital edema.

- Variant: Granulomatous rosacea presents with firm, indurated, yellow, brown, or red papules and nodules.

Pathogenesis

- Rosacea is a multifactorial disease that is comprised of genetic predispositions that cause neurovascular dysregulation and immune dysregulation in combination with aberrant responses to environmental triggers.
- Those with genetic predisposition have exaggerated responses to temperature changes, emotional stress, ultraviolet light, and microbial antigens.[33]

Differential and Diagnosis

- Differentials: Adult acne vulgaris, seborrheic dermatitis, contact dermatitis, photodermatitis, actinic damage, and systemic lupus erythematosus.
- Diagnosis
  - Rosacea is diagnosed clinically and can present in a variety of ways. According to the National Rosacea Society, it is defined by one or more of the following primary features: flushing, papules, pustules, or persistent erythema.[33,35]
  - Secondary features include burning, stinging, itching, edema, dryness, erythematous plaques, ocular manifestations, and phymatous changes.[33,35]

Treatment

Treatment depends on the severity of the disease presentation and clinical phenotype of the condition. Topical therapies should be chosen based on the signs and symptoms, tolerability, and past treatments used. The FDA-approved topical therapy options include azelaic acid (15%) gel, metronidazole (1% cream and gel; 0.75% gel, cream, and lotion), sodium sulfacetamide/sulfur (gel, cleanser, lotion, suspension, and cream), brimonidine tartrate (0.33%) gel, oxymetazoline hydrochloride (1%) cream, and ivermectin (1% cream). Azelaic acid or metronidazole treat mild-moderate rosacea and reduce erythema and inflammatory lesions through decreasing reactive oxygen species activity.[35] Sodium sulfacetamide/sulfur wash is also anti-inflammatory, but should be avoided in those with renal disease and known sulfa allergy.[35] Also, the odor is off-putting for patients.[35,36] Topical medications brimonidine tartrate and oxymetazoline hydrochloride are approved for treatment of persistent erythema associated with rosacea, with fast onset of action.[35] Ivermectin cream is thought to have anti-inflammatory actions because it upregulates anti-inflammatory cytokine IL-10; decreases phagocytosis of neutrophils; and targets demodex mites, which are thought to play a role in rosacea activity. It is thought to have less itching and burning compared with azelaic acid and once-daily use of ivermectin 1% cream was more effective than 0.75% metronidazole cream applied twice daily.[37] Topical calcineurin inhibitors are a treatment option for steroid-induced rosacea and lead to improvement in erythema.

Oral therapy for rosacea includes tetracyclines, macrolides, metronidazole, and isotretinoin. Doxycycline (100–200 mg/day) and minocycline (100–200 mg/day) are most used because of tolerability and can be combined with topical agents, such as azelaic acid or metronidazole to achieve control of papulopustular rosacea. Low-dose doxycycline (40 mg) has also recently become available and is another systemic treatment. Ocular rosacea tends to respond to lower doses of doxycycline for long-term treatment.[35] Macrolide antibiotics (azithromycin and clarithromycin) and metronidazole are used in patients with papulopustular rosacea who are intolerant to tetracyclines or have an allergy. Oral isotretinoin is used in erythematotelangiectatic and papulopustular rosacea that have been refractory to other treatments. Although antibiotics

have a faster onset of action when compared with oral isotretinoin, oral isotretinoin is an alternative treatment option with doses of 0.3 mg/kg/day being best tolerated and most effective.[35] Additionally, oral ivermectin has been used off-label because it is not approved by the FDA for rosacea at this time.[35] Laser therapy is used to treat telangiectasia including pulsed dye laser, intense pulsed light, and Nd:YAG laser.

GROVER DISEASE

Grover disease, also known as transient and persistent acantholytic dermatosis, is an inflammatory condition that most commonly affects older White men with a history of extensive photodamage.[38] Some patients report flaring in the winter, whereas others report flares in the summer. The lesions themselves usually last 2 to 4 weeks, but relapses and reoccurrences are common.

Clinical Presentation

- Skin-colored to pink papules and papulovesicles, often excoriated, on the trunk and proximal extremities.
- Lesions can also have crusting or hyperkeratosis and associated pruritis.

Pathogenesis

- Direct pathogenesis is unknown, but exacerbating factors include heat, sweating, low humidity, cold, friction, and radiation (ultraviolet and ionizing).

Differential and Diagnosis

- Differentials: Folliculitis, multiple lichenoid keratoses, arthropod bites, pemphigus foliaceous (early), and Darier disease.
- Diagnosis:
  - Although often diagnosed clinically, biopsy is sometimes needed to differentiate from other dermatoses (discussed previously). Direct and indirect immunofluorescence are negative.

Treatment

There is no cure to Grover disease, but avoidance of triggers and providing symptomatic treatments is standard practice. In mild cases, xerosis should be treated with emollients. Topical corticosteroids (triamcinolone) are recommended to decrease inflammation and reduce pruritis. Other topical treatments include vitamin D analogues and calcineurin inhibitors. Oral antihistamines, such as fexofenadine and hydroxyzine hydrochloride, is added to help ease pruritis, but do not prevent new lesions from forming. Second-line therapy includes oral retinoids, such as isotretinoin. Careful monitoring of liver function and lipids is important, particularly in this geriatric population. For severe cases of Grover disease in which systemic therapies are not tolerated, another treatment option includes phototherapy.

DISCLOSURE

The authors have nothing to disclose.

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