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Nummular Dermatitis

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Introduction

Nummular dermatitis is a pruritic eczematous dermatosis characterized by multiple coin-shaped lesions. It may occur as a feature of atopic dermatitis, asteatotic eczema, or stasis dermatitis. The prognosis of this condition is excellent. Most cases can be treated successfully with conservative measures and topical corticosteroids and a majority of patients will eventually achieve remission. **Nummular** dermatitis may also be referred to as **nummular** eczema, discoid eczema, and microbial eczema.

Etiology

The exact etiology of **nummular** dermatitis is unknown. Numerous factors have been implicated such as xerosis, contact allergy sensitization [1], reactivity to environmental aeroallergens [2], *Staphylococcal* colonization [3], use of irritating and drying soaps, frequent bathing with hot water, environments with low humidity, skin trauma (Koebner phenomenon), exposure to rough fabrics such as wool, breast implantation [4], and certain medications (antivirals, interferon, isotretinoin [5], retinoids, ribavirin, and gold compounds). Chronic venous stasis may be a predisposing factor to development on the lower extremities [2]. A minority of patients with **nummular** dermatitis may also have a history of atopic dermatitis.

Epidemiology

Nummular dermatitis has a bimodal distribution, affecting predominantly females 15-25 years of age and males 50-65 years of age. Prevalence ranges from 0.1% to 9.1% [1].

Pathophysiology

The cutaneous lipid barrier may be compromised by the natural aging process, chronic venous stasis, bacterial colonization, certain drugs, and sensitization to contact allergens, most commonly metals (nickel sulfate, potassium dichromate, cobalt chloride) [1]. The release of cytokines (i.e. IFN-g and IL-17) results in increased recruitment of T cells, dendritic cells, and Langerhan's cells eventually leading to epidermal hyperplasia and the development of characteristic lesions [6].

Histopathology

Histopathologic findings are not specific and share features with other eczematous dermatoses with spongiotic dermatitis that may be subacute or chronic. The following features may be present: parakeratosis, acanthosis, intracellular edema, eosinophils, and exocytosis of lymphocytes.

History and Physical

Acutely, lesions may begin as papules or vesicles that coalescence into plaques. When established, lesions will appear as symmetrically distributed, sharply defined, round or coin-shaped, erythematous, and eczematous plaques ranging in size from 1-10 cm. Late-stage lesions may develop a drier scale and lichenification. Lesions may be associated with mild to intense pruritus. Lesion and symptom severity are exacerbated by behaviors that decrease the natural moisture barrier of the skin such as harsh soaps and long, hot, frequent showering. The lower extremities are most commonly

involved, followed by the upper extremities and trunk. The face and scalp are spared. Post-inflammatory pigmentary changes commonly persist after resolution. Dermoscopic findings may reveal scales, shiny yellow clods, and irregularly distributed brownish-red globules [7].

Evaluation

Nummular dermatitis is a clinical diagnosis based on its classical presentation of pruritic coin-shaped erythematous and eczematous plaques symmetrically distributed in a person with diffusely dry skin. Dermoscopic evaluation may assist with the diagnosis; other laboratory tests are not generally necessary [8]. Ancillary tests to help distinguish it from other causes of annular erythematous plaques, if indicated, include potassium hydroxide wet-mount examination of skin scrapings, skin bacterial culture, biopsy, and patch testing.

Treatment / Management

Management focuses on restoring the natural skin barrier and avoiding behaviors that dry and irritate the skin [9]. Frequent moisturization with thick emollients such as petroleum jelly is recommended. Patients should be instructed to take short (5 minutes or less) lukewarm showers, use gentle hydrating soaps, and apply emollients immediately after showering while skin is still slightly wet. Encourage patients to avoid tight clothing and irritating fabrics.

If there are signs of a secondary bacterial infection or a bacterial swab of lesional skin is positive, treat with topical or oral antistaphylococcal antibiotics depending on whether lesions are localized or diffuse. Sensitivities and local antibiotic resistance patterns may impact the choice of antibiotic.

Use of mid-to-high potency (class 2-5) topical corticosteroids 1-2 times daily directly to affected skin helps to decrease the inflammation and pruritus. Topical calcineurin inhibitors (tacrolimus, pimecrolimus) may be used as steroid-sparing topical agents. A typical alternating schedule includes topical corticosteroids on weekdays and topical calcineurin inhibitors on weekends.

Sedating antihistamines such as hydroxyzine or diphenhydramine may be used to provide relief for severe pruritus, especially at nighttime.

For widespread disease in which topical treatment may not be feasible, narrowband UVB light therapy should be considered. Light therapy should be administered 2-3 times weekly, slowly titrating up to the appropriate duration and desired clinical response. If light therapy is not available, systemic immunosuppressants and immunomodulators have been used to treat extensive recalcitrant disease. Systemic corticosteroids may lead to rapid improvement but are associated with high rates of rebound when discontinued. Few studies have shown lasting efficacy after the use of methotrexate in children [10]. Recently, dupilumab has also shown promise as a possible emerging treatment [11].

Differential Diagnosis

- Allergic contact dermatitis: may present with **nummular** lesions. Patch testing can help identify the causative allergen.
- Plaque psoriasis: classically presents with well-demarcated erythematous plaques with overlying silvery scale plaques involving the scalp, elbows, and knees, although any site may be involved.
- Stasis dermatitis: is often associated with findings of venous insufficiency including edema, varicose veins, and/or atrophic hypopigmented scars (atrophie blanche). Lesions consist of erythematous patches that may progress to scaly plaques involving the ankle and distal lower leg. Plaques may be exudative, lichenified, or superimposed on varicosities.
- Asteatotic eczema (eczema craquelé): appears as diffuse erythema and fine scale, with small irregular fissures and cracks, most commonly on the bilateral lower legs.
- Lichen aureus: demonstrates small petechiae and well-defined red to brown macules or plaques symmetrically on the bilateral lower legs, or rarely on the trunk and upper extremities.
- Fixed drug eruption: presents with one or multiple well-circumscribed circular red to brown patches or edematous plaques that recur in the same location when the patient is exposed to the implicated drug.

- Erythema annulare centrifugum: appears as arcuate or round plaques with central clearing with a trailing scale at the outermost edge.
- Tinea corporis: presents with one or multiple circular erythematous scaly plaques with central clearing.
- Majocchi granuloma: demonstrates multiple perifollicular pustules coalescing to form erythematous scaly plaques.
- Impetigo: presents as "honey-colored" or golden-crusted papules and plaques with an erythematous base.
- Secondary syphilis: appears as diffuse pink, red, violaceous, or brown papules and plaques with overlying scale that often also involves the palms and soles.
- Pityriasis rosea: presents with pale pink circular or oval patches or thin plaques with a collarette of fine white scale.
- Patch or plaque stage mycosis fungoides (cutaneous T-cell lymphoma): erythematous to slightly hyperpigmented patches or thin plaues with ovelying fine scale. Lesions may measure 2-20 cm and occur primarily in areas of the body that are protected from sun exposure (i.e. the buttocks or axillary folds).
- Squamous cell carcinoma in situ (Bowen's disease): usually presents as a singular (rarely multiple) erythematous, scaly, well-defined thin plaque with a circular or irregular shape.

Prognosis

Nummular dermatitis often follows a chronic course characterized by relapses and remissions over months to years.

Complications

Because of the impaired skin barrier, lesional skin may become secondarily infected. *Staphylococcus aureus* is the most commonly implicated pathogen. Impetiginized lesions can display purulent ooze and thicker golden crusting than noninfected lesions. A bacterial swab should be performed for culture and sensitivities. Doxycycline, or another antistaphylococcal antibiotic based on local antimicrobial resistance patterns, may be selected initially and further treatment tailored according to the resultant sensitivities.

As with any inflammatory condition of the skin, there may be post-inflammatory dyspigmentation of the skin including erythema, hypo- or hyperpigmentation.

Consultations

The majority of cases of **nummular** dermatitis should respond to conservative measures such as gentle skincare and bland emollients used in combination with mid- to high- potency steroids. Consultation to a dermatologist should be considered in refractory, widespread, or atypical cases. Further evaluation may include skin scraping with potassium hydroxide preparation, bacterial swab for culture and sensitivities, biopsy, or patch testing as discussed previously. If narrow-band UVB light therapy, systemic immunosuppressant, or immunomodulator is warranted, this should be also carried out under the supervision of a dermatologist.

Deterrence and Patient Education

Patients should be counseled that behavior modification can result in significant improvement of **nummular** dermatitis. Patients should be instructed to take short (5 minutes or less) lukewarm showers, use gentle hydrating liquid cleansers, and apply emollients immediately after showering while the skin is still damp. The use of a bland ointment such as petroleum jelly is recommended. Encourage patients to avoid tight clothing and irritating fabrics such as wool. Otherwise, patients must be instructed to carefully follow their prescription regimen as directed.

Enhancing Healthcare Team Outcomes

Inflammatory dermatoses such as **nummular** dermatitis are often treated by primary care physicians, nurse practitioners, physician assistants, or dermatologists. Although the differential diagnosis of **nummular** dermatitis is quite broad, is it most often clinically diagnosed by recognizing its patterns of morphology, distribution, and epidemiology. Ancillary testing such as skin scraping with potassium hydroxide preparation, bacterial swab for culture

and sensitivities, or biopsy may be performed either in the primary care or dermatologic setting. If the **nummular** dermatitis is unresponsive to conservative management and topical corticosteroids, it is appropriate to consult a dermatologist. Patch testing, narrowband UVB treatment, or systemic immunosuppressant or immunomodulator therapy should be supervised by a specialist.

Treatment of **nummular** dermatitis can be optimized with an interprofessional team approach. The patient's nurse, primary care provider, or dermatologist should monitor for therapy compliance, report any adverse effects, and relay the response to therapy to the remainder of the team. This type of interprofessional teamwork will enhance patient outcomes and minimize adverse reactions in the care of patients with **nummular** dermatitis. [Level 5]

Continuing Education / Review Questions

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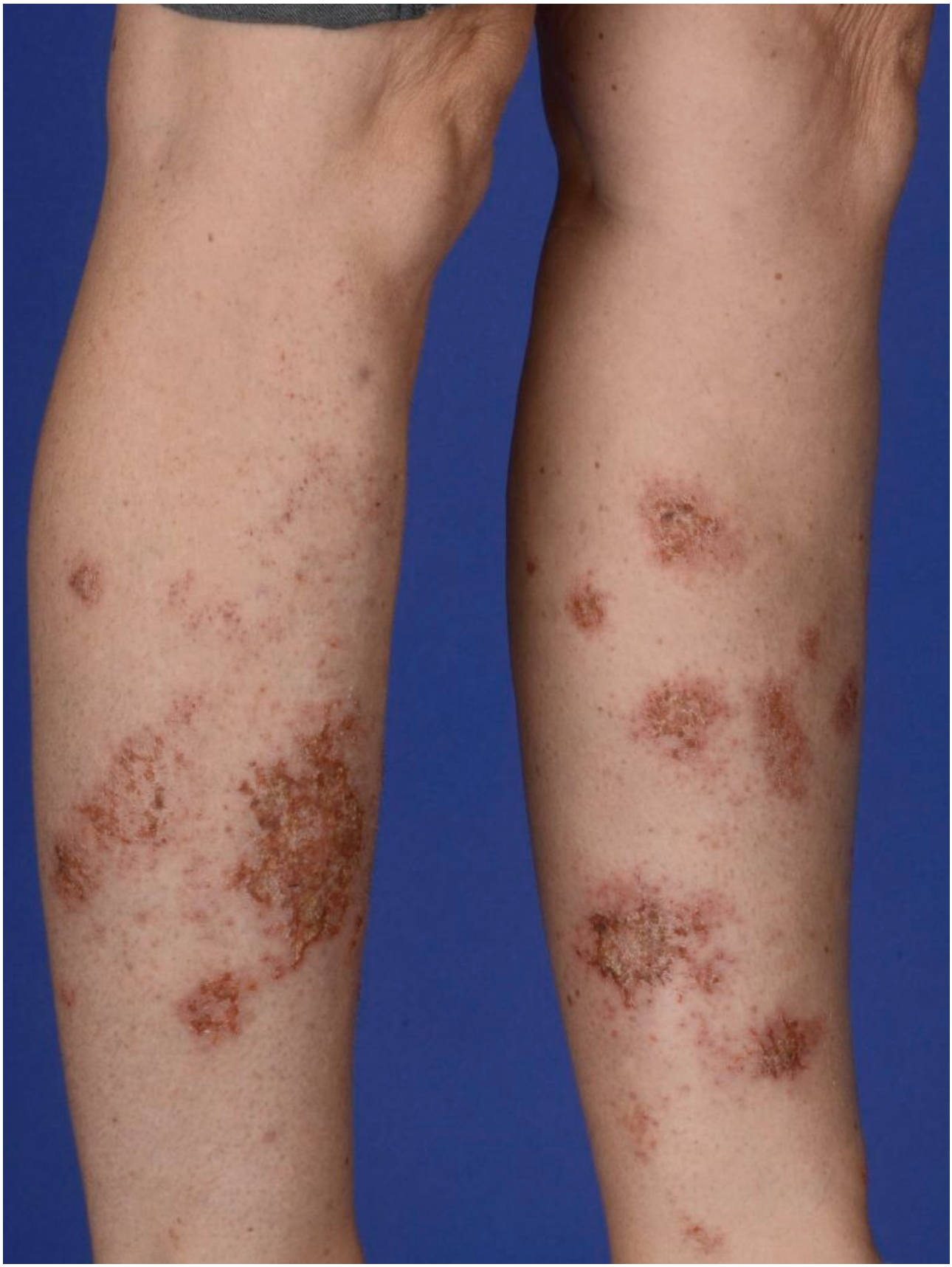
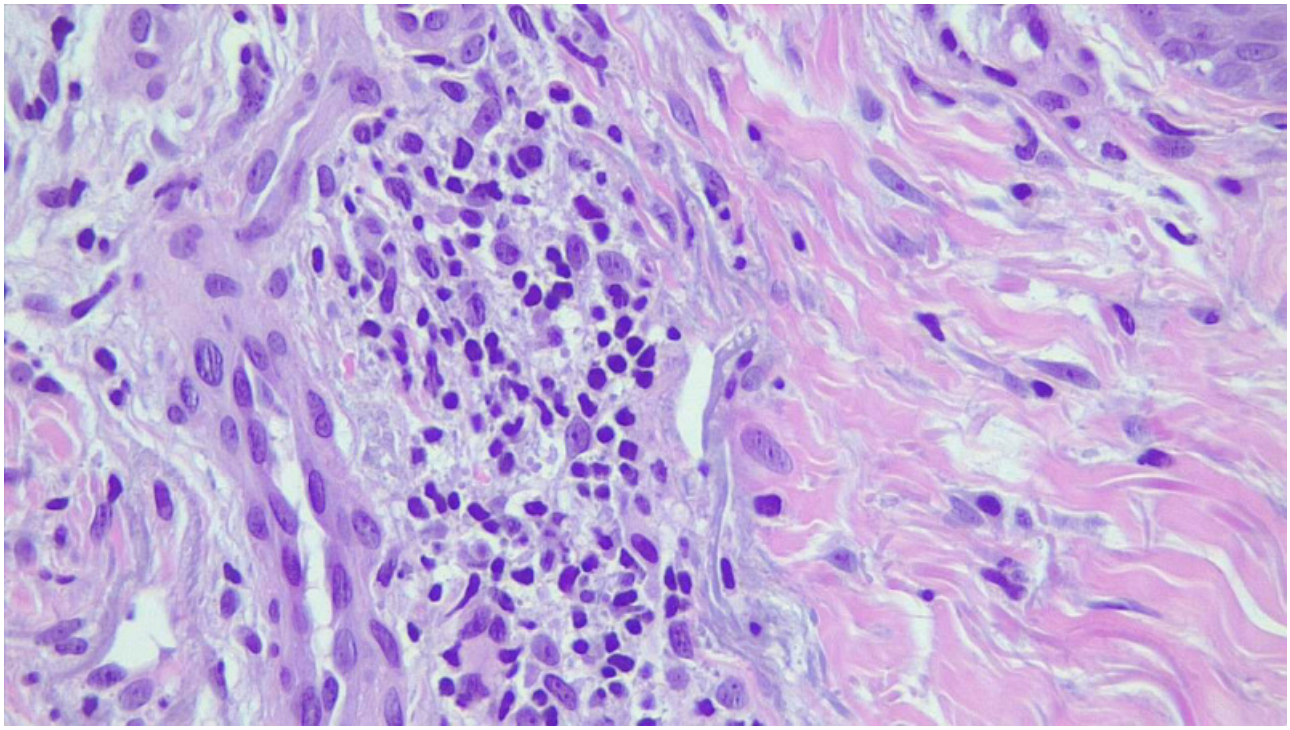
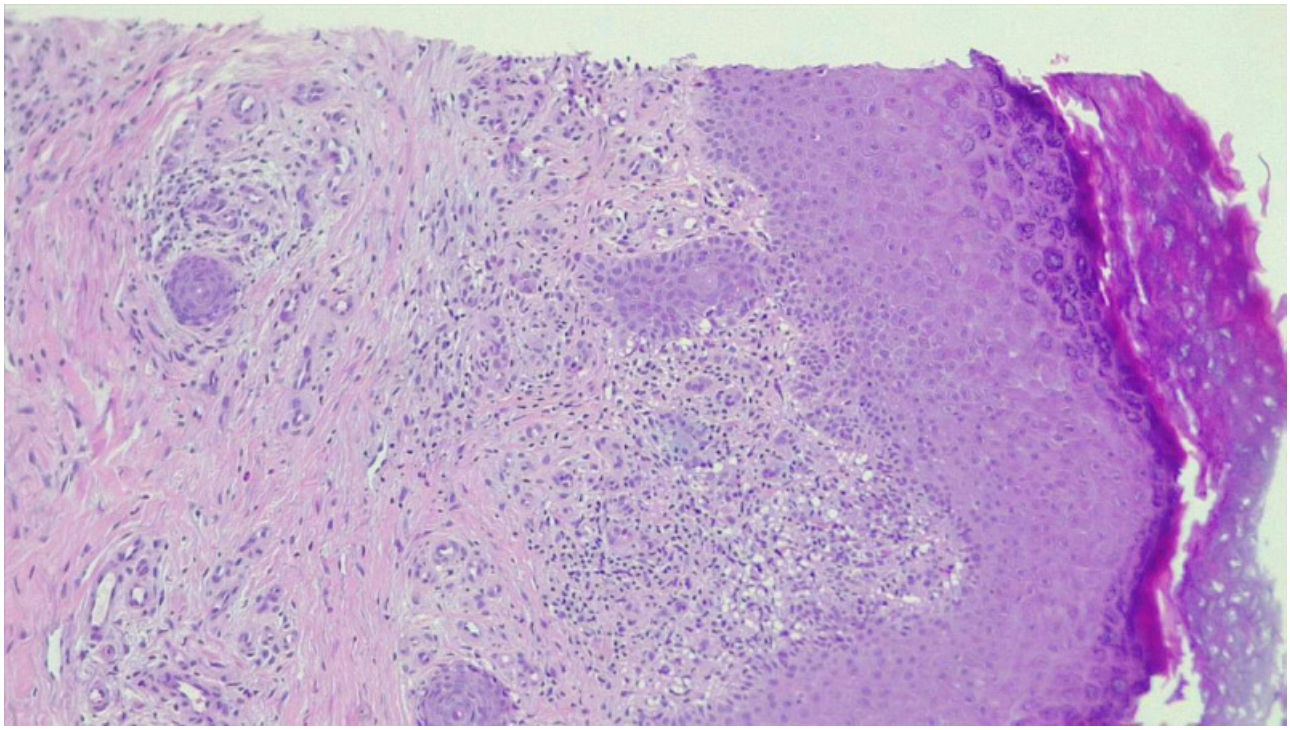
Figures



Nummular Dermatitis. Contributed by DermNetNZ



Dermatitis. Histological demonstration of lymphocytic perivascular infiltrate causing reactive hypertrphic changes in the endothelium. H/E 20x. Contributed by Fabiola Farci, MD



Dermatitis, H/E 4x. Contributed by Fabiola Farci, MD

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