Atopic and Contact Dermatitis of the Vulva

Rita Pichardo-Geisinger, MD

KEYWORDS

• Atopic dermatitis • Eczema • Lichen simplex chronicus • Contact dermatitis
• Irritant dermatitis

KEY POINTS

- Pruritus is a common symptom in a patient with atopic, irritant, or allergic contact dermatitis.
- The diagnosis requires a careful history and physical examination, at times supplemented with a biopsy and/or patch test.
- The 5 most common allergens from the standard series to cause a positive reaction were gold sodium thiosulfate 0.5%, nickel sulfate hexahydrate 2.5%, balsam of Peru 25%, fragrance mix 8%, and cobalt chloride 1%.

Pruritus, or itch, is a common vulvar complaint that is very often treated empirically as a yeast infection; however, yeast infections are just one of the many conditions that can cause vulvar itch. Ignoring these other conditions can do a disservice to your patient by prolonging their pruritus unnecessarily. Atopic dermatitis, irritant contact dermatitis, and allergic contact dermatitis are extremely common noninfectious causes of vulvar itch that are often underdiagnosed by nondermatologists. Up to 50% of cases of chronic vulvovaginal pruritus in adult women can be attributed to irritant and allergic contact dermatitis,[1] and atopic and irritant contact dermatitis are the most common vulvar disorders that present among prepubertal girls, representing one-third of all patients in an Australian series with vulvar disease.[2] Identifying these conditions and treating them appropriately can significantly improve a patient's quality of life and appropriately decrease health care expenditures by preventing unnecessary additional referrals or follow-up visits and decreasing pharmaceutical costs. This article provides an approach to the clinical evaluation and management of these 3 eczematous vulvar disorders.

ATOPIC DERMATITIS

Atopic dermatitis (eczema) is a common, chronic inflammatory skin condition characterized by the presence of red itchy patches and thin plaques on the skin. The development

Disclosure Statement: No conflicts of interest.
Department of Dermatology, Wake Forest University School of Medicine, 4618 Country Club Road, Winston-Salem, NC 27104, USA
E-mail address: rpichard@wakehealth.edu

Obstet Gynecol Clin N Am 44 (2017) 371–378
http://dx.doi.org/10.1016/j.ogc.2017.05.003 **obgyn.theclinics.com**

of atopic dermatitis results from an interaction between hereditary (genetic) and environmental factors, including a defect in the skin barrier function and changes in the weather making the skin more susceptible to irritation. Some patients have a genetic predisposition for the development of allergic IgE reactions to common environmental allergens, as demonstrated by a personal or family history of allergic rhinitis (hay fever), asthma, and/or atopic dermatitis, otherwise known as the atopic triad.

At the microscopic level, greater transepidermal water loss and thus lower skin surface hydration levels are some of the characteristics of a disrupted skin barrier. Vulvar skin is particularly prone to barrier dysfunction because of irritation caused by sweat, urine and/or feces, the use of irritating and/or allergenic products including baby and adult wipes, washcloths, lubricants, hygiene products, deodorized pads, tampons, overwashing, condoms, tight pants, G-strings, shaving, waxing, or cycling, and sexual encounters that may worsen the primary disease. In addition, the changes in the skin barrier may induce an immune response related to type 2 T helper lymphocytes (Th2 type). Severe pruritus is the most common characteristic of eczema. It may be worse during the evening and exacerbated by sweating or wool clothing. This is at least one of the ways that the itch–scratch cycle starts.

Diagnostic Evaluation

A diagnosis of eczema is made by the combination of a history of itching and scratching along with typical clinical findings. Biopsy may also play a role if the diagnosis cannot be confirmed via history and physical examination findings. Evaluating for infection, especially candidiasis or dermatophytosis, is important because it can mimic eczema. It is also possible to develop a superinfection on top of eczema, because both topical corticosteroid use and the eczema itself are correlated with an increased risk of infection compared with noneczematous vulvar skin.[3]

Atopic dermatitis presents acutely as poorly demarcated erythematous edematous plaques with vesicles, subacutely as erythematous patches or plaques, and chronically as accentuated skin markings or a thickened lichenified plaque.[4] Excoriations may be present secondary to scratching. Repetitive trauma, scratching, or rubbing may lead to postinflammatory pigmentary alteration, either hyperpigmentation or hypopigmentation (**Fig. 1**).

The histologic findings of eczema depend of the stage of the lesion that is biopsied. In an acute lesion, there is a combination of spongiosis, intraepidermal edema with microvesiculation or macrovesiculation and inflammatory infiltrates composed of lymphocytes, eosinophils, and mast cells in the upper dermis. In an older lesion, spongiosis persists, but vesiculation tends to dissipate. As the lesion progresses in chronicity, the epidermis gets progressively thicker owing to hyperplasia (called acanthosis) and inflammation and spongiosis may become mild or absent.

Management

Patient counseling regarding the chronic nature of atopic dermatitis and its management is critical. Patient education is fundamental to achieve control of this disease. It is important for patients to understand that control, rather than cure, is the goal.

The management of atopic dermatitis can be fundamentally characterized by 4 factors:

- Eliminating irritant and allergen exposure,
- Controlling pruritus,
- Repairing barrier function, and
- Applying topical antiinflammatory agents.