

Contact Dermatitis of the Vulva

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KEYWORDS

- Vulva • Female genitalia • Allergic contact dermatitis
- Irritant contact dermatitis • Contact dermatitis

Inflammatory dermatoses and cutaneous infections commonly affect the vulva. Cultural taboos, embarrassment, and fear may prevent women from reporting vulvar pruritus, pain, and cutaneous eruptions to their health care providers. As a result, women often self-medicate using a myriad of over-the-counter and/or herbal remedies. Vulvar disease is often multifactorial. Diagnosis of vulvar diseases can be challenging because of the effect of local factors (moisture, friction), clinical manifestations, which differ from nongenital cutaneous sites, and the frequent concomitant presence of multiple diagnoses. Lack of knowledge by practitioners causes underrecognition and misdiagnosis of vulvar disorders, which leads to repeated failure to improve despite use of multiple medications.

Whether self-induced or iatrogenic, contact dermatitis frequently complicates vulvar disorders. Contact dermatitis results from exposure to exogenous agents, either irritants or allergens. Irritant contact dermatitis (ICD) affects the vulva more often than allergic contact dermatitis (ACD).¹ Acute, subacute, and chronic forms exist. Subacute and chronic contact dermatitis, either allergic or irritant, typically present with eczematous changes ranging from mild erythema to lichenified, thick erythematous plaques with excoriation, fissures, and weeping. Severe, acute contact dermatitis may be bullous, erosive, and extremely painful.

The vulva is particularly susceptible to ICD and ACD.^{2,3} Vulvar skin has been shown to react more intensely to some irritants (eg, benzalkonium chloride, maleic acid) than forearm skin; however, vulvar skin may not be more susceptible than other skin areas to all irritants.^{2,4} The barrier function of

vulvar skin is compromised by moisture (urine, vaginal discharge), enzymes (stool residua), friction, and heat, all of which constitute the normal vulvar environment.⁵ Estrogen is integral in maintaining the strength and integrity of vulvar tissues. Estrogen deficiency, as occurs during premenarche, oral contraceptive use, postpartum, lactation, and menopause, decreases the barrier function of the vulvar epithelium.^{5,6} Persistent contact with urine can alone cause irritant dermatitis, widely recognized as diaper dermatitis in infants. Urinary incontinence, secondary to pelvic floor muscle weakness and laxity, severely affects up to 20% of women aged 80 years and older.⁷ Obesity and limitations in physical mobility, which reduce the ability to touch and visually inspect the vulva, can significantly impede a patient's ability to keep the vulva clean and dry. Additional risk factors for vulvar contact dermatitis include preexisting dermatosis, occlusion (from natural skin folds, sanitary napkins), poor nutritional status, overzealous hygiene practices, and concomitant microbial infections (ie, candidiasis, infectious diarrhea).

ICD of the vulva is more common than ACD.¹ Both types of contact reactions may occur simultaneously and have overlapping features clinically and histologically. Contact dermatitis should be considered early in the evaluation of patients with chronic vulvar symptoms (pruritus, irritation) with or without abnormal findings on examination. In patients with other vulvar dermatoses, contact reactions should be suspected in patients who do not respond appropriately to therapy.⁸ Vulvar contact dermatitis may result from direct application, inadvertent transfer from other body sites, local exposure to

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products excreted in the urine and/or feces after oral consumption, and systematized contact reactions. This article addresses the most common causes of ICD and ACD affecting the vulva and provides practical recommendations for evaluation and management.

IRRITANT CONTACT DERMATITIS

ICD is common, although the exact prevalence remains unclear. ICD results from cutaneous exposure to substances that cause direct cytotoxicity to keratinocytes without prior sensitization. Irritants can remove surface lipids and water-holding substances, damage cell membranes, and denature keratins and other proteins.^{9–11}

Severe, acute ICD is equivalent to a caustic burn and manifests as erythema, edema, and vesicles, which evolve quickly into erosions and superficial ulcerations (**Fig. 1**). Subacute and chronic irritant reactions present with poorly demarcated erythematous patches and plaques with variable scale and excoriations. Patients may complain of burning, rawness, stinging pain, and/or itching. Severe irritant dermatitis can progress to punched-out ulcers with elevated, indurated borders (erosive diaper dermatitis of Jacquet) or erythematous to violaceous pseudoverrucous papules and nodules (granuloma gluteale infantum). These clinical presentations are well recognized in infants with chronic diaper dermatitis but may also occur in adults.¹² Significant pain and dysuria may occur in the context of vulvar erosions. Irritant reactions may also manifest as stinging and burning in the absence of identifiable skin changes.¹³

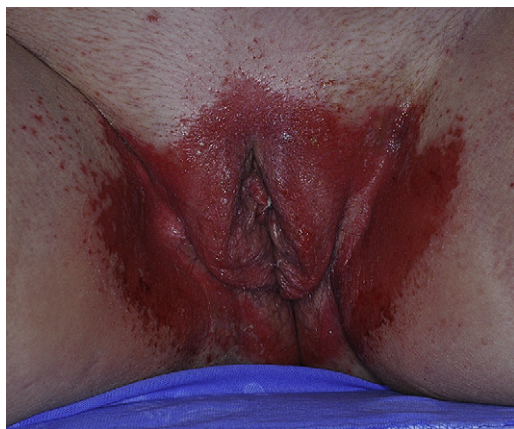


Fig. 1. Severe, erosive ICD of the vulva to witch hazel. Note the relative sparing of the inferior genitoinguinal creases in which natural occlusion of skin folds minimized irritant contact. (Courtesy of Dr Lynette J. Margesson, Manchester, NH.)

Greater concentrations of a given irritant are more likely to cause dermatitis. Common classes of irritants include acids, alkalis, surfactants, solvents, oxidants, and enzymes.¹⁴ More specifically, urine, feces, sweat, topical medications, overzealous cleansing practices, and feminine hygiene products cause irritant contact reactions (**Box 1**).⁶ Strong irritants usually cause immediate symptoms, which facilitate correlation between the culprit product and disease. Weaker irritants cause more subtle changes and are therefore less often suspected.

Douches contain both irritants and sensitizers. Douches that contain acids or alkalis can become irritants if not diluted properly by the patient during preparation. The main irritants in douches are alum, citric acid, and lactic acid. Sodium bicarbonate and sodium borate are alkalis that cause irritant contact reactions.¹⁵

Box 1

Common vulvar irritants

Body Fluids

- Abnormal vaginal discharge
- Feces (enzymes)
- Semen
- Sweat
- Urine (ammonia)

Excessive bathing

Feminine hygiene products

- Depilatories
- Douches
- Feminine hygiene wipes
- Lubricants
- Panty liners
- Sanitary napkins/pads

Heat

- Heating pads
- Hair dryer

Medications

- Alcohol-based creams and gels
- Bichloroacetic acid, trichloroacetic acid
- Cantharidin
- Fluorouracil
- Imiquimod
- Phenol
- Podophyllin
- Propylene glycol
- Spermicides (foaming agents, emulsifiers)

Soaps and detergents (including bubble baths)

ALLERGIC CONTACT DERMATITIS

ACD represents a delayed (type IV) hypersensitivity reaction that occurs in a genetically predisposed individual after sensitization to an allergen. This type of immunologic reaction involves antigen presentation by Langerhans cells, antigen recognition in the context of MHC class II molecules by T cells, and subsequent T-cell activation and proliferation. Allergens are typically low molecular weight, lipid-soluble molecules.¹⁴ Because there is a delay between exposure to the allergen and onset of symptoms or physical abnormalities (typically 48–72 hours), allergens may not be readily recognized. Acute ACD results from exposure to a strong allergen to which the patient was previously sensitized and presents as an acute, vesiculobullous eruption with severe pruritus and/or pain; patients typically seek care immediately. Significant erythema, edema, coalescing vesicles, and ulcerations occur (**Figs. 2 and 3**). Vesicles may assume linear or geometric configurations reflecting areas of allergen contact when transferred by fingers or when touched by plant leaves. Robust allergic reactions may become generalized,



Fig. 2. Acute erosive ACD to benzocaine. Note the background of erythema and lichenification involving the mons and medial thighs. (Courtesy of Dr Lynette J. Margesson, Manchester, NH.)



Fig. 3. Severe ulcerative ACD to benzocaine. Note severe ulceration of the labia majora with urinary catheter in place. The proximal medial thighs are frequently affected because of inadvertent contact and spreading of vulvar allergens. (Courtesy of Dr Lynette J. Margesson, Manchester, NH.)

especially affecting sites of previous contact dermatitis to the same allergen (recall phenomenon). Chronic ACD may result from intermittent or prolonged exposure to a relatively weak allergen and presents as subacute inflammation with variable degrees of pruritus; there is often a pattern of exacerbation and remission, which parallels allergen exposure.¹⁶ Chronic ACD manifests as erythematous to hyperpigmented, lichenified plaques with variable scale and excoriation (**Fig. 4**). In dark-skinned patients, hyperpigmentation may be prominent and can mask erythema.

Vulvar ACD was first reported in the literature in 1990.¹⁷ ACD has been assessed in numerous studies of patients with pruritus vulvae or other vulvar dermatoses, and numerous allergens have been identified (**Box 2**).^{8,12,18–20} Patients with pruritus vulvae and pruritic vulvar dermatoses are at higher risk for developing contact dermatitis because of their frequent, and potentially excessive, use of topical anesthetics (eg, benzocaine) and other products in the quest for symptom relief.¹⁹ The most frequently identified allergens in large studies include neomycin,^{8,17,19} “caine” anesthetics (benzocaine, dibucaine hydrochloride),^{19,21} fragrance,^{19,21} balsam of Peru,⁸ and nickel.^{8,20,21}

Medicaments are agents used to treat, prevent, or alleviate the symptoms of disease. Culprit allergens in medicaments reflect local prescribing practices and vary geographically.²² Frequently cited medicaments in vulvar ACD include topical anesthetics, antibiotics, corticosteroids, antiseptics, and preservatives.^{20,22} Of 135 patients with chronic vulvar pruritus and discomfort who underwent patch testing, 63 (47%) had at least 1 positive patch reaction, and 39 (29%) were considered



Fig. 4. Chronic ACD with mild erythema, scaling, lichenification, and areas of hyperpigmentation. (Courtesy of Dr Bethanee J. Schlosser, Chicago, IL.)

relevant to the patient's clinical condition. Medicaments and their components accounted for most of the positive reactions.⁸ Lewis and colleagues¹⁹ showed similar results when 121 women with pruritus vulvae underwent patch testing; 57 (47%) had at least 1 relevant positive patch reaction, of which medicaments or their components were the most common allergens. Utas and colleagues²³ performed patch testing on 50 women with pruritus vulvae including women treated for primary vulvar dermatoses; 26 (52%) of the women had at least 1 positive patch reaction, and 8 (16%) were deemed relevant. The most frequent relevant allergens were fragrance, preservatives, and medicaments. Brenan and colleagues²¹ obtained positive patch test results in 42% of 50 patients with chronic vulvar symptoms; medicaments (including "caine" mix and ethylenediamine) and fragrance were common allergens identified.

Neomycin is the most common allergen among topical antibiotic preparations. Positive patch test reactions are observed in 11.6% of adults and 8% of children.²⁴ Drugs that cross-react with neomycin include bacitracin, streptomycin, kanamycin, gentamicin, and tobramycin.²⁵

The "caine" anesthetics are widely recognized causes of ACD. Ester anesthetics (benzocaine, procaine, tetracaine) are the best-known allergens

and cross-react with para-aminobenzoic acid, paraphenylenediamine, sulfa drugs, thiazide diuretics, and azo and aniline dyes.²⁵ Unfortunately, benzocaine continues to be widely available in over-the-counter topical anti-itch preparations (ie, Vagisil). A history of sulfa allergy or adverse reaction to hair dye can provide diagnostic clues to the culprit allergen. Amide anesthetics (bupivacaine, lidocaine, dibucaine, mepivacaine, and prilocaine) are less potent and less common sensitizers. Patients may have simultaneous ACD to both classes of topical anesthetics.²⁶

Topical corticosteroids may be sensitizers, due to the steroid molecule itself or its vehicle components. Patch testing to tixocortol pivalate (Class A), budesonide (Class B and D), and hydrocortisone-17-butyrate (Class D2) detects most cases of topical corticosteroid-induced ACD.¹⁵

More than 5000 fragrance materials are used at present.¹⁵ In general, fragrance allergy is common; the North American Contact Dermatitis Group has demonstrated sensitivity to fragrance mix I or balsam of Peru in 9.1% and 10.6% of patients, respectively.²⁷ Balsam of Peru (*Myroxylon pereirae*) and fragrance mix I (a mix of 8 fragrances) have conventionally been used to screen patients for fragrance allergy. Fragrance mix II, which contains lylal, citral, farnesol, citronellol, hexyl cinnamic aldehyde, and coumarin, began to be used in 2007. Balsam of Peru is a naturally occurring compound of benzyl acetate, benzyl alcohol, cinnamic acid, cinnamic alcohol, cinnamic aldehyde, eugenol, and isoeugenol, derived from Central and South American fir trees. Balsam of Peru is a useful marker for fragrance allergy, with a positive patch reaction in more than 50% of fragrance-allergic patients.¹⁵ Sensitivity to balsam of Peru and/or fragrance has been implicated in pruritus vulvae and chronic vulvar dermatoses.^{8,19–21,28}

Ethylenediamine hydrochloride is a stabilizer that became notorious for ACD to the original formulation of Mycolog cream.¹⁵ Ethylenediamine hydrochloride is also the parent compound for the class of antihistamines that includes hydroxyzine and is a component of the asthma medication, aminophylline. Although the present formulation of Mycolog does not contain ethylenediamine hydrochloride, this sensitizer continues to be present in various topical preparations.

Sensitivity to nickel has been documented in several studies of vulvar patients. Crone and colleagues²⁹ evaluated 35 vulvar dermatitis patients with patch testing; positive reactions were documented in 26% with nickel being the most common. Similarly, Lucke and colleagues²⁸ demonstrated nickel sensitivity in 31% of 55

Box 2**Common vulvar allergens****Anesthetics**

- Amides (dibucaine, lidocaine)
- Crotamiton
- Diphenhydramine
- Esters (benzocaine, tetracaine)

Antibiotics

- Bacitracin
- Neomycin
- Polymyxin
- Sulfonamides

Antifungals

- Imidazoles (clotrimazole, miconazole)
- Nystatin

Antiseptics

- Chlorhexidine
- Gentian violet
- Mercuric chloride
- Phenylmercuric salts
- Povidone iodine
- Thimerosal

Corticosteroids**Douches**

- Benzethonium chloride
- Fragrance/perfumes
- Methyl salicylate
- Oil of eucalyptus
- Oxyquinoline
- Phenylmercuric acetate
- Thymol

Emollients

- Glycerin
- Jojoba oil
- Lanolin
- Propylene glycol

Fragrance

- Balsam of Peru
- Cinnamic alcohol, cinnamic aldehyde
- Eugenol
- Isoeugenol
- Hydroxycitronellal

Nail Polish

- Toluene-sulfonamide formaldehyde resin

Nickel**Preservatives**

- Bronopol
- Diazolidinyl urea
- Formaldehyde

- Imidazolidinyl urea
- Kathon
- Quaternium 15

Rubber

- Latex
- Mercaptobenzothiazole
- Thiurams

Sanitary Napkins

- Acetyl acetone
- Formaldehyde
- Fragrance
- Methacrylates

Spermicides

- Hexylresorcinol
- Nonoxynol
- Oxyquinoline sulfate
- Phenylmercuric acetate and butyrate
- Quinine hydrochloride

patients with vulvar dermatoses, and approximately half of the patients experienced reduction in symptoms with avoidance of direct contact with nickel and/or adherence to a nickel-free diet. In the absence of direct nickel contact with the vulva, the role of nickel sensitivity in vulvar pruritus and dermatitis requires further evaluation.

Various spices and flavorings have also been linked to vulvar pruritus and dermatitis.^{30,31} Fifty-three patients with chronic anogenital dermatitis underwent patch testing, and 35 (66%) showed at least 1 positive reaction.³⁰ In addition to noting sensitivity to nickel (18 patients), fragrance (9 patients), and preservatives (6 patients), positive reactions to spices, including curry mix, nutmeg, coriander, peppermint oil, and onion powder, were noted in 15 patients. These reactions were deemed relevant in 4 patients, with symptomatic and clinical improvement noted with avoidance of the allergens.

CLINICAL DIFFERENTIAL DIAGNOSIS

Acute vesiculo-erosive forms of contact dermatitis can mimic other bullous and ulcerative disorders (**Box 3**). The differential diagnosis of acute contact dermatitis includes herpes simplex virus infection, candidiasis, fixed-drug eruption, immunobullous disorders (pemphigus vulgaris, mucous membrane pemphigoid, bullous pemphigoid), and erosive lichen planus. The acute onset of the eruption favors contact dermatitis; immunobullous

Box 3**Clinical differential diagnosis of ICD and ACD****Acute vesiculoerosive contact dermatitis**

- Bullous pemphigoid
- Candidiasis
- Erythema multiforme
- Fixed drug eruption
- Hailey-Hailey disease (benign familial pemphigus)
- Herpes simplex virus infection
- Lichen planus (erosive, bullous)
- Mucous membrane pemphigoid
- Pemphigus vulgaris

Chronic eczematous contact dermatitis

- Atopic dermatitis
- Candidiasis
- Erythrasma
- Extramammary Paget disease
- Lichen simplex chronicus
- Psoriasis
- Seborrheic dermatitis
- Squamous cell carcinoma in situ
- Tinea cruris

disorders and erosive lichen planus typically are more gradual in onset.

Subacute and chronic forms of ICD and ACD can be similar clinically to several eczematous and papulosquamous dermatoses (see **Box 3**). Atopic dermatitis, lichen simplex chronicus, seborrheic dermatitis, inverse psoriasis, and tinea cruris should be considered in the differential diagnosis. The less common but more serious diagnoses of squamous cell carcinoma in situ and extramammary Paget disease should also be considered for localized lesions.

EVALUATION

Contact dermatitis, both irritant and allergic, should be considered early in the evaluation of women with vulvar complaints and compatible clinical findings. In addition, contact dermatitis often complicates the management of other vulvar dermatoses. Patients with vulvar diseases that do not respond to appropriate treatment should be interviewed and evaluated for possible contact reactions.

Accurate diagnosis of ICD or ACD requires a high degree of suspicion and thorough investigation to correlate symptoms and clinical findings with the patient's exposure history. A thorough history of all vulvar products, medications (prescription, over-the-counter, and complementary), contraceptives, sexual products, and hygiene practices

must be obtained to identify potential contactants. Questioning may reveal extreme hygiene practices including the use of bleach, lye, and antiseptic mouthwashes to cleanse the vulva, minimize odor, and/or prevent infection. In addition, cosmetics and products used elsewhere on the body may be culprits after inadvertent transfer to the genitalia from the hands (ie, toluene sulfonamide formaldehyde resin in nail polish as a cause of vulvar ACD). Products used by intimate partners may play a role, as close contact (via sexual activity, ie, connubial transfer, and shared living spaces) can result in irritant and allergen transfer. The interview should also address potential occupational and hobby exposures; a floral worker with vulvar dermatitis had a relevant positive patch reaction to primin.³⁰ The temporal relationship between the onset of recurrent episodes of dermatitis and food consumption should also be investigated; oral products may cause sensitization and dermatitis after excretion in the urine and/or feces.³¹ There are myriad types of products about which patients should be questioned (see **Boxes 1** and **2**). Additional areas of cutaneous involvement should be investigated; concomitant eyelid dermatitis in patients with vulvar contact dermatitis is not uncommon and reinforces the importance of considering allergen transfer by touching.

The clinical features of ACD and ICD may be indistinguishable from each other as well as other vulvar dermatoses. Irritant and allergic contact reactions may occur simultaneously, further complicating the diagnosis. Histopathologic features may be nonspecific, with overlapping features noted between ICD and ACD. Histopathologic changes of ICD can vary according to the specific irritant and its concentration.¹¹ Low irritant concentrations typically cause epidermal spongiosis, mild superficial dermal edema, and a superficial perivascular lymphocytic inflammatory infiltrate.³² High concentrations of irritant can result in ballooning of keratinocytes in the upper epidermis with variable degrees of cell necrosis and neutrophilic inflammation.³³ ACD demonstrates epidermal spongiosis, intraepidermal spongiotic vesicles, and a superficial dermal inflammatory infiltrate composed of lymphocytes, macrophages, and Langerhans cells. Eosinophils are present in variable numbers. Spongiosis becomes less prominent as lesions become more chronic.³⁴ Psoriasiform hyperplasia, typified by acanthosis, hyperkeratosis, and parakeratosis, may occur in chronic forms of both ACD and ICD.³⁴ Biopsy, for both routine histopathology and direct immunofluorescence, may be most

useful in the setting of erosive lesions to exclude immunobullous disorders, erosive lichen planus, and other entities.

Patch testing is essential for accurate identification of allergens. For patch testing evaluation of vulvar dermatitis, the North American Contact Dermatitis Group standard and corticosteroid series are recommended. Additional series for medications, perfumes, preservatives, and other allergens may be performed, if indicated.³⁵ Patch tests are best interpreted at 48 and 72 hours with an additional reading obtained between 72 and 120 hours.¹⁵ The relevance of positive patch reactions, either as a primary cause or an aggravating factor, must be considered. If a positive patch test reaction is identified, correlation with the clinical examination and history of exposures should be made.³⁶ Patients may be able to tolerate a medication containing a preservative to which they have a positive patch reaction because the concentration of the preservative in the preparation used is often less than the threshold needed to produce a clinical reaction.³⁷ Patch testing has not been shown to be helpful in patients who presented with vulvodynia (vulvar pain in the absence of any clinical abnormality).²⁸

Patients should be provided with detailed written instructions regarding allergens to avoid and a list of safe alternatives. The Contact Allergen Replacement Database, available from the American Contact Dermatitis Society, is an invaluable tool (www.contactderm.org). Of note, fragrance-allergic patients should be educated that “unscented” products may actually contain low concentrations of masking fragrance (used to mask unpleasant chemical or fatty odors but not intended to convey their own scent). Patients should avoid such unscented products and instead seek “fragrance-free” products that possess no fragrance chemicals including masking fragrance.¹⁵ When the safety of a product is unclear, sensitized patients may be advised to perform repeated open application testing (ROAT, twice-daily application to nondermatitic skin of the antecubital fossa). Because vulvar skin reacts differently to irritants and allergens than nongenital skin, and by definition the vulva is not an open area, ROAT has limited applicability in vulvar contact dermatitis.

The patient's estrogen status should be assessed. Normal saline wet mount preparations of vaginal discharge and pH measurement are essential. Signs of estrogen insufficiency include pH greater than 4.5, reduced or absent lactobacilli, and increased numbers of immature, parabasal keratinocytes, which appear as rounded cells with relatively high nuclear to cytoplasmic ratio.

Patients should be evaluated clinically for secondary infection by bacteria, fungi, and viruses. Clinical clues include fissuring, crusting, and pustules. Bacterial exudate cultures can be obtained from crusted erosions or unroofed pustules. Potassium hydroxide microscopy and fungal culture of pustule contents can assist in the diagnosis of vulvovaginal candidiasis. Herpes simplex virus cultures, Tzanck smears, and/or direct fluorescent antigen preparations should ideally be obtained from unroofed, intact pustules or vesicles. Clinical examination findings in conjunction with the results of laboratory testing should direct antimicrobial therapy.

TREATMENT

All potential irritants and allergens must be discontinued. Patients should be advised to cleanse the vulva no more than twice daily using only lukewarm (not hot) water without soap, cleanser, or detergent. Sitz baths, in which the patient fills the tub with only lukewarm water to cover the hips and buttocks, may be used for cleansing the vulva and for relief of pain or itching. For those patients that are not able to maneuver into a tub, a handheld shower spray can be used on a gentle setting for directed vulvar hygiene. Patients should be instructed to use only their hands to clean the vulva and to discontinue the use of all washcloths, loofahs and sponges. Patients should pat the vulva dry, avoiding rubbing and additional friction. Plain white petrolatum should be applied as an emollient and barrier and is especially important for fissured or eroded areas.

Erosions cause severe dysuria. Patients should be advised to apply a copious layer of plain white petrolatum to all eroded areas before and after each bathroom use. Patients may reduce dysuria further, either by urinating while seated in a shallow tube of lukewarm water or by pouring lukewarm water over the vulva during micturition.

Vesicles and weeping erosions produce additional moisture that can macerate vulvar tissues and cause further irritation. Sitz baths (5–10 minutes twice daily) or compresses are helpful in removing serous crusts and providing symptomatic relief. Dilute aluminum acetate solutions can be applied for short periods as compresses to facilitate desiccation.

Depending on the severity and chronicity of the vulvar dermatitis, a low- (hydrocortisone 2.5%), medium- (triamcinolone acetonide 0.1%), or high-potency (clobetasol propionate 0.05%, halobetasol 0.05%) topical corticosteroid ointment should initially be applied twice daily until lesions have healed completely (**Fig. 5**). Corticosteroid potency

and frequency of dosing can be tapered as the dermatitis improves. For severe cases of contact dermatitis, including those that present with vesicular and/or erosive disease, systemic corticosteroids are indicated. Prednisone (40–60 mg daily in the morning or alternatively 0.5–1.0mg/kg/d based on ideal body weight) with gradual tapering over 14 to 21 days quickly provides dramatic relief of symptoms and clinical improvement, more so potentially for allergic rather than irritant reactions. Systemic corticosteroids may be given through intramuscular injection (triamcinolone acetonide, 1 mg/kg as a single dose); however, intramuscular injection precludes individualized tapering of medication dose and does not allow discontinuation of medication if a patient experiences adverse effects (ie, steroid psychosis). As symptoms improve, patients should be transitioned to the use of a topical corticosteroid ointment as noted above. Systemic prophylaxis for candidiasis (fluconazole 150 mg by mouth once weekly) should be considered in all patients treated with topical or systemic corticosteroids.⁵

Pruritus and associated scratching can perpetuate and promote inflammation, the so-called scratch-itch cycle. A combination of nonsedating and sedating antihistamines can be used for maximal symptom relief. Loratadine (10 mg), fexofenadine (60 mg), or cetirizine (10 mg) can be administered in the morning for relief of daytime pruritus; dosing may be increased to twice daily as needed. Hydroxyzine (25–100 mg) and doxepin (10–75mg) are the preferred sedating antihistamines for bedtime use daily to minimize scratching during sleep. Topical antipruritic medications (eg, diphenhydramine, lidocaine) should be avoided. Patients should be advised to avoid heat, which

may increase histamine release and worsen pruritus. Instead, cool compresses, cool gel packs, or frozen vegetables wrapped in thin fabric can provide significant relief of pruritus; frozen products and ice should not be applied directly to the skin.

If secondary infection by bacteria, fungi, or viruses is suspected, appropriate diagnostic testing should be performed, as discussed earlier. Clinical examination findings in conjunction with the results of laboratory testing should direct antimicrobial therapy. Systemic antimicrobial therapy should be used, when possible, rather than topical medications to avoid further irritant or allergic reactions and to minimize physical manipulation of the affected areas. In patients treated with systemic antibiotics, prophylaxis for secondary vulvovaginal candidiasis using fluconazole 150 mg by mouth once weekly should be considered.

Estrogen deficiency should also be corrected. Although estrogen deficiency may not be an issue of acute importance during the initial evaluation, failure to address estrogen deficiency may predispose the patient to recurrent contact dermatitis or other vulvar dystrophy in the future. Consideration should be given to the risks, benefits, and modes of administration in the context of the individual patient.

Comprehensive patient education, both verbal and written, is essential for successful resolution of the dermatitis and, more importantly, to prevent future irritant or allergic contact reactions. Patients should be followed closely during the initial phase of treatment to ensure patient adherence and to monitor for complications of therapy (ie, corticosteroid atrophy, secondary infection, and development of ACD or ICD to prescribed medications).

SUMMARY

Contact dermatitis, both irritant and allergic, should be considered early in the evaluation of women with vulvar complaints and compatible clinical findings. Such considerations are especially important with increased chronicity of symptoms and for those women who have seen multiple providers, as both scenarios increase the number of medicaments and other remedies used by patients. Vulvar contact dermatitis is more often caused by irritants than allergens. The most frequently noted allergens in patients with vulvar pruritus and/or dermatitis include medicaments, preservatives, fragrance, and nickel. Contact dermatitis may be the primary or secondary problem in patients with vulvar symptoms, and vulvar disease is often multifactorial.



Fig. 5. Dramatic improvement in severe ICD to witch hazel (see Fig. 1) after discontinuation of product and treatment with topical corticosteroids. Sparing of the inferior genitoinguinal creases is more easily appreciated after resolution of acute edema and erosions. (Courtesy of Dr Lynette J. Margesson, Manchester, NH.)

An organized, stepwise approach to the evaluation and treatment of patients with vulvar complaints is required to successfully address each abnormality identified toward the end goal of a healthy vulva and happy patient.

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