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# Contact dermatitis of the vulva

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**ABSTRACT:** Vulvar diseases rarely stand alone. They are often caused or worsened by primary irritant or allergic contact dermatitis, and this should be considered when evaluating any vulvar complaint. All irritants should be avoided in all women, and those with vulvar dermatoses should be patch tested to help define or rule out allergens.

**KEYWORDS:** contact dermatitis, vulva, vulvar diseases.

## Introduction

Vulvar disease is often multifactorial, and contact dermatitis of the vulva is frequently an important contributor. Although it is not usually the primary cause of vulvar symptoms, it is often the compounding factor in patients complaining of persistent vulvar pruritus, irritation, or burning. This is not surprising considering that vulvar patients often self-medicate and have open, irritated skin for very long periods of time. They have commonly seen numerous caregivers and the contact dermatitis has gone unrecognized. Consequently, a long list of prescription and over-the-counter preparations have been used. These products may be irritating, frankly allergenic, or in some cases, both. Unfortunately, the clinical appearance of the vulva is often not helpful in making the diagnosis. There is a spectrum from mild local erythema to thick, weeping, fissured, lichenified plaques. Consider the confusion of etiologies in a patient with vulvar lichen sclerosus who is washing five times a day with a caustic soap and is now allergic to her topical corticosteroid. Vulvar disease can be difficult enough without two or three overlapping conditions all together in one area!

The reported incidence of contact dermatitis in the anogenital area varies. From the vulvar clinic in Oxford, UK, in 2000, 20–30% of cases had dermatitis, and of these, 26% had relevant patch tests (1). From an Australian vulvar clinic, 15% of cases had contact dermatitis (2). There have been several large series evaluating vulvar contact sensitivity, but the overall incidence in the general population is unclear (3–8).

Contact dermatitis is an inflammation of the skin resulting from an external agent that acts as an irritant or as an allergen. The skin reaction may be acute, subacute or chronic. As mentioned above, contact dermatitis of the vulva can be difficult to diagnose since the changes vary from minor to extreme, and are often superimposed on pre-existing, complicated, and chronic conditions like lichen simplex chronicus, lichen planus or lichen sclerosus.

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## Irritant contact dermatitis

Irritant contact dermatitis of the vulva is the commonest condition overall, although the published reports reviewing the etiology of pruritus vulvae (2–8) indicate that irritant contact dermatitis was reported specifically in only one series at 22.8% of a mix of male and female patients (7).

It is not surprising that irritant contact dermatitis is more common than allergic contact dermatitis (9). Vulvar skin reacts more intensely to irritants than other skin areas (10). Vulvar skin was shown to be more reactive to benzalkonium chloride and maleic acid than forearm skin (10), although increased vulvar sensitivity does not extend to all irritants, as shown in a study using sodium lauryl sulfate (11). Measuring vulvar irritant dermatitis

can be difficult and only accepted measuring techniques should be used (12). This increased tendency to irritant dermatitis may cause problems by itself, or act as a risk factor for further development of allergic contact dermatitis.

The barrier function of vulvar skin is substantially weaker than at other anatomical sites, as already shown by the previous studies. Moisture, friction, urine, and vaginal discharge all contribute to vulvar irritation by weakening barrier function. There are three main clinical responses to irritants: acute irritant dermatitis, chronic (cumulative) irritant dermatitis, and sensory irritation (13). The acute type is equivalent to a chemical burn caused by a potent, caustic irritant. The cumulative, chronic type develops after repeated exposure to weak irritants. Sensory irritation is characterized by stinging and burning caused by chemical exposure where there is no detectable skin change. All three affect the vulva, and a single chemical such as propylene glycol can induce all three irritant patterns as well as being an allergen.

The strong, caustic irritants usually burn and sting immediately, so the cause is obvious. Some such as 5-fluorouracil cause delayed pain. Phenol causes anesthesia plus extensive destruction. These types of substances are used purposely by some patients with self-destructive tendencies, and by others obsessed with the need to "cleanse" because of being physically or morally "dirty". The weak irritants are the more common, often insidious offenders. Fear of detectable odor or infection may drive overzealous hygiene habits. Many soaps and detergent cleansers are too harsh for the vulva. Patients do not realize the damage that they are doing. Overzealous hygiene can be a major problem since a cleanser or soap may be followed by powder and, later in the day, by antiseptic wipes. It is no wonder that these ladies are irritated (14).

For healthy vulvar barrier function, there must be moist, pliable tissue. The normal vulvovaginal secretions maintain this and must not be unnecessarily stripped away. Estrogen is important to maintain the structural and functional integrity of the vagina and introitus. Low estrogen levels occur with menopause, breast-feeding, postpartum, medications (e.g., birth control pills or tamoxifen), castration, and to a partial extent, pre-menarche. These estrogen-deficient patients are particularly vulnerable to irritant contact dermatitis (Fig. 1). Recognizing this issue is important for managing all vulvar disorders, particularly contact dermatitis.

When one considers the wide spectrum of vulvar diseases from infancy to old age, not just those seen in vulvar clinics, the commonest rash is an irritant contact dermatitis—diaper or "napkin" dermatitis. The typical "chafing" diaper rash is well recognized as the commonest rash of infancy, affecting up to 35% of infants at a given time (15). In a study from Italy, up to 15.2% of infants wearing super-absorbent diapers experienced rashes (16). These diaper rashes are precipitated by constant moisture, occlusion, and frictional forces. Excess wetting macerates the stratum corneum so that it is fragile and friction causes further trauma. Urine macerates by itself. Urine and feces enzymatically break down to ammonia that "scalds". Fecal enzymes are directly irritating. Microbes like Candida from stool can cause further damage. Less commonly, other bacteria can be an issue. The degree of irritation will depend on host factors such as underlying atopic dermatitis, psoriasis, and nutritional deficiencies. Other contributing factors are poor skin care from over-washing, concurrent use of antibiotics with a resulting secondary yeast infection and/or diarrhea (17). These are all very well recognized and managed in infancy but not so well recognized in adults. Incontinence is a very common problem in women, starting in their fifties or even earlier depending on the integrity of their pelvic floor muscles. Pads are worn, and are often inadequate to assure dryness and the patients hide this problem from their healthcare providers because of embarrassment. Clinically, these patients usually have the same type of rashes which we see in infancy (Fig. 2).

**Fig. 1. Irritant contact dermatitis caused by urinary incontinence in a 66-year-old woman with vulvar atrophy.**

Dermatologists are being consulted ever more frequently to manage exactly the same problem in aged and nursing home patients. Dealing with perineal hygiene for a chronically incontinent,

**Fig. 2. A 55-year-old patient with vulvar and perianal lichen sclerosus with secondary irritant dermatitis from fecal incontinence. Note the characteristic whiteness of lichen sclerosus with secondary excoriations and erythema.**

uncooperative, elderly person with severe diarrhea is difficult. Sometimes the irritant dermatitis can even progress to the severe, papular, and erosive eruption on the vulva called papuloerosive erythema of Sevestre and Jacquet (18).

Many products can cause irritation of the vulva (see Table 1). Some are obvious like the strong caustic products while the weaker irritants are more insidious and often missed.

Clinically, the patients complain of varying degrees of itching, burning, and irritation. The onset may be sudden or gradual depending on the etiology. The patient may be aware of the offending product that precipitated the problem or may be totally mystified. There is often a history of repeated use of the product, especially soaps. The "obsessive cleansers" have already been discussed above. Elderly women may use normally harmless products which, when combined with lack of estrogen, napkins for incontinence, and pantyhose and/or girdles, can produce the typical glazed vulvar erythema seen in "diaper dermatitis" in infants.

Typically, there is a subacute presentation of varying redness, swelling, and scaling. Infrequently, in severe cases, there may be erosions or frank ulcers with marked erythema and swelling. In chronic examples, one sees lichenification with excoriation, dyspigmentation (hyper- and/or hypopigmentation) and scaling. Secondary infection may be manifested by pustules, crusting, and fissuring. In black patients, hyperpigmentation can be prominent. The area of involvement may be localized to the site of a contactant or be more generalized as the product spreads, with moisture, in the perineum. The patients also tend to scratch more widely, extending the area of damage (14,19).

**Table 1.** Common vulvar irritants (19,33)

| |
|---|
| Strong caustic irritants |
| Bichloroacetic acid and trichloroacetic acid |
| 5-Fluorouracil |
| Lye (in soap) |
| Phenol |
| Podofilox |
| Podophyllin |
| Sodium hypochlorite |
| Solvents |
| Weak cumulative irritants |
| Alcohol |
| Deodorants |
| Detergents |
| Diapers |
| Feces |
| Feminine spray |
| Pads |
| Perfume |
| Povidone iodine |
| Powders |
| Propylene glycol |
| Semen |
| Soap |
| Sweat |
| Urine |
| Vaginal secretions |
| Water |
| Wipes |
| Physically abrasive contactants |
| Face cloths |
| Sponges |
| Thermal irritants |
| Hot water bottles |
| Hair dryers |

The histology of irritant contact dermatitis is nonspecific, showing spongiosis, sometimes acanthosis, parakeratosis, and some dermal inflammatory infiltrate. The picture is similar to atopic and allergic contact dermatitis.

The diagnosis is made on history and clinical pattern. The history must be thorough since patients often do not recognize the association between their problem and the contactant. Review their routines for hygiene, and for personal care during menstrual periods and with coitus. Ask specific questions as to their use of soaps, cleansers, washcloths, detergents, lubricants, and anti-yeast treatments, for example. Be sure to check all prescription and over-the-counter products. Find out what makes them better or worse. The primary

diagnostic and therapeutic step is to stop all products. Just use plain tepid water or Sitz baths daily (no cleansers), and apply plain petrolatum after the soak, while the skin is moist. Patch testing may define the allergens responsible for allergic contact dermatitis. Appropriate cultures are essential. Biopsy may be necessary, especially if there are other associated conditions (e.g., squamous cell carcinoma with contact dermatitis).

The differential diagnosis includes candidiasis, psoriasis, seborrheic dermatitis, allergic contact dermatitis, and lichen simplex chronicus. These conditions may sometimes coexist, yielding, for example, an irritant plus an allergic contact dermatitis with underlying psoriasis and lichen simplex chronicus complicated by secondary candidiasis and a bacterial infection.

The most important treatment is to stop the offending agent and/or practice. Next, correct barrier function using a Sitz bath with plain lukewarm water for 5–10 min twice a day. (If a Sitz bath is not possible, compresses can be used.) After the Sitz bath, gently pat dry then coat the area with a thin film of plain petrolatum to hold in the moisture. Where there is a lack of estrogen, replace it if possible. Control secondary Candida infection with oral fluconazole 150 mg weekly for 1–3 weeks. Oral treatment avoids other potential topical irritants and allergens. Treat secondary bacterial infections with an appropriate oral antibiotic. The fluconazole will prevent secondary yeast infection. Stop scratching with nighttime sedation using hydroxyzine or doxepin 10–75 mg at 6–7 p.m. In the morning, consider cetirizine 5–10 mg bid, or an SSRI like citalopram 20–40 mg or sertraline 50–100 mg. Nonspecific treatment to stop scratching includes cool gel packs (keep in resealable plastic bags in the refrigerator, not the freezer) or even plain, cold yogurt on a sanitary napkin for 5–10 min, as needed.

To reduce inflammation, topical steroids are used. Triamcinolone 0.l% ointment bid can be used for moderately irritated dermatitis. If severe, a super-potent topical steroid like clobetasol or halobetasol 0.05% ointment is prescribed bid for 1–3 weeks. The stronger steroids can then be replaced by milder 1–2.5% hydrocortisone ointment as needed. For very severe irritation, prednisone 0.5–1 mg $kg^{-1}$ $day^{-1}$ decreased over 14–21 days or triamcinolone acetonide 40 mg $mL^{-1}$ (Kenalog 40), 1 mg $kg^{-1}$ intramuscularly as a single dose can be an excellent choice.

Good patient education is needed plus follow-up to adjust treatment. Changing hygiene habits can be a challenge (see Table 2).

**Table 2.** Treatment of vulvar contact dermatitis

- Identify and stop offending agent and practices
- Correct barrier function
  - Sitz baths, plain tepid water bid
  - Pat dry—gently. Do not rub
  - Thin film of plain petrolatum
  - Add estrogen if indicated
  - Treat infection
    - Oral fluconazole 150 mg weekly
    - Oral antibiotics
- Stop scratching
  - Nonspecific
    - Cool packs
    - Cool Sitz baths
    - Cool yogurt
  - Specific
    - Sedation
      - (p.m.) Hydroxyzine or doxepin 10–75 mg at 6–7 p.m.
      - (a.m.) Cetirizine 5–10 mg bid
        Citalopram 20–40 mg
- Reduce inflammation
  - Topical steroids for 2–3 weeks
    - Triamcinolone ointment 0.l% bid
    - Halobetasol or clobetasol ointment 0.05% bid
    - Reduce potency and frequency to 1–2.5% hydrocortisone ointment for maintenance
  - Systemic steroids
    - Prednisone 0.5–1 mg $kg^{-1}$ $day^{-1}$ decreased over 14–21 days
    - Intramuscular triamcinolone acetonide 1 mg $kg^{-1}$

## Allergic contact dermatitis

Allergic contact dermatitis is an immunologically mediated inflammatory cutaneous reaction to an allergen in a sensitized individual (20). Differentiating an allergic from an irritant contact dermatitis on the vulva can be confusing since they often overlap. An important key to the diagnosis of allergic contact dermatitis is the intermittent nature of the symptoms (21). As an example, a patient with a sanitary napkin allergy will flare only when using the offending pads. Vulvar allergic contact dermatitis has been studied only in the past 12 years, with the first report in 1990 (22) and with the first significant study of 135 women in 1992 (4). There have been eight relevant reports, of which seven were limited to women (2–8). Most of these studies were investigating causes of vulvar pruritus or dermatoses (see Table 3). In the study by Lewis et al. (6), it is important to note that 47% of patients with lichen sclerosus had positive patch tests. Lichen simplex chronicus can be triggered by various factors. Irritation is a

**Table 3.** Reports of allergic contact dermatitis of the vulva: (N/A) not applicable

| Condition studied | Number of cases | Percentage with one or more positive tests | Percentage with relevant positive Tests | Most important allergens | References |
|---|---|---|---|---|---|
| Pruritus vulvae | 50 women | 78 | N/A | Nickel, perfume, local anesthetic, neomycin | Doherty et al. (3) |
| Vulvar dermatoses (diagnostically difficult vulvar rashes) | 135 women | 47 | 29 | Nickel, balsam of Peru, ethylenediamine, neomycin | Marren et al. (4) |
| Chronic vulvar symptoms (pruritus and vulvodynia) | 50 women | 42 | N/A | Nickel, fragrance, ethylenediamine | Brennan et al. (2) |
| Anogenital dermatoses | 201 women | 39 | 28 overall 19 vulvar cases | Antibiotics, anesthetics, corticosteroids, perfume | Goldsmith et al. (5) |
| Vulvar pruritus | 121 women | 58.7 | 49 | Caines, fragrance, neomycin | Lewis et al. (6) |
| Anogenital complaints | 1008 men and women (51.8% male, 48.2% female) | 47 | 34.8 | Nickel, balsam of Peru, fragrance, and dibucaine hydrochloride | Bauer et al. (7) |
| Vulvar dermatoses | 55 women | 65 | 73 (see study) | Nickel, fragrance, medicaments, dyes | Lucke et al. (8) |
| Vulvar lichen simplex chronicus | 61 women | 47.5 | 26 | Medicaments, preservatives | Virgili et al. (18) |

major cause, but so is allergic contact dermatitis. A recent study showed patch testing positive to relevant allergens in 26% of the 51 women studied (23). Nickel is a common allergen. In 55 women with vulvar pruritus, nickel sensitivity was found in 31% (8). Of these subjects, 50% had their symptoms improve with nickel avoidance. Allergic contact dermatitis has not been found to be relevant in patients with vulvodynia (2,6,23,24).

There are many important allergens reported in vulvar patients (Table 4). Each of these allergens elicits a type IV delayed hypersensitivity reaction. Antigen-presenting Langerhans cells recognize and process the antigen, and the production of T-lymphocytes and numerous cytokines starts the inflammatory process. Finding the allergen can be a challenge. It may be a seemingly harmless item that the patient is using, like her incontinence pad (25) or even a fragrance her partner uses as an aftershave. One must be a real detective for these cases.

The greatest culprits in allergic contact dermatitis of the vulva are the topical anesthetics. Benzocaine, which is found in hundreds of over-the-counter products for warts to hemorrhoids, is in Vagisil, which is used by women to soothe irritations and as a lubricant (26). Benzocaine cross-reacts with “sulfa” drugs, para-aminobenzoic acid and paraphenylenediamine. Thus, a history of a sulfa or a hair dye allergy can be a useful clue for vulvar patients. Dibucaine hydrochloride in Nupercainal can be a problem since it cross-reacts with lidocaine. Ethylenediamine, another common sensitizer, has been removed from Mycolog cream in the USA, but not in from the generic products or Kenacomb cream in Canada (27). Neomycin, also found in these mixtures and a variety of other topical antibiotics, can be a significant problem. Perfume products are common sensitizers in all toiletries, and should be identified and avoided. The commonest cause of vulvovaginitis is Candida and the therapeutic agents Nystatin and the imidazoles are sensitizers (28). Topical corticosteroids may also be sensitizers because of either the steroid molecule itself or allergens in the

**Table 4.** Allergens in allergic contact dermatitis (34)

| Product | Allergens |
|---|---|
| Additive to medications, hygiene products and cosmetics | |
| Fragrances | Balsam of Peru, cinnamic alcohol and aldehyde, eugenol and isoeugenol, hydroxycitronellal |
| Preservatives | Bronopol, Kathon, stearyl alcohol formaldehyde and formaldehyde releasers—Quaternium 15, imidazolidinylurea, diazolidinylurea |
| Emollients | Lanolin, propylene glycol, jojoba oil, glycerin |
| Medications | |
| Anesthetics | Benzocaine, crotamiton, dibucaine, diphenhydramine, tetracaine, lidocaine |
| Antibiotics | Neomycin, bacitracin, sulfonamides, polymyxin |
| Anti-candidals | Imidazoles—clotrimazole, miconazole, etc., Nystatin |
| Antiseptics | Povidone iodine, thimerosal, phenylmercuric salts, mercuric chloride, hexachlorophene, gentian violet |
| Corticosteroids | All |
| Hormones | Estrogen, progesterone |
| Spermacides | Hexyl resorcinol, nonoxyl, oxyquinoline sulfate, phenylmercuric acetate and butyrate, quinine hydrochloride |
| Miscellaneous | |
| Douches | Fragrances, thymol, oxyquinoline, methylsalicylate, phenylmercuric acetate |
| Rubber products<br>Pessaries<br>Condoms<br>Diaphragms<br>Gloves | Rubber—latex, mercaptobenzothiazole, thiurams, etc. |
| Body fluids | Semen, saliva |
| Clothing | Dye, formaldehyde, synthetic resin |
| Sanitary napkins | Formaldehyde, fragrance, acetyl acetone, methacrylates |
| Nail polish | Formaldehyde resin, toluene, sulfonamide |
| Poison ivy, oak and sumac | Urushiol |

vehicles (29). Sensitization to topical corticosteroids occurs in 4–5% of cases, and although this is uncommon, it must be considered in patients with vulvar dermatoses responding poorly to the usually appropriate corticosteroid. A screen for topical steroid sensitivity using tixocortol, budesonide, and hydrocortisone-17-butyrate will detect most of the main offenders. Pantyhose, clothing with azo dyes, and formaldehyde-treated permanent press fabrics should be suspect.

The clinical presentation can be acute, subacute, or chronic. The symptoms include varying degrees of pruritus and burning, depending on the severity of the reaction. The onset is about 10–14 days following exposure to a newly sensitizing antigen, but it may be less than 24 h if there has been prior sensitization. The degree of reaction will depend on genetic factors, concentration, and duration of skin contact, and the allergenic potential of the chemical itself (30).

The hallmarks of allergic contact dermatitis in the acute phase are vesiculation, severe pruritus and a tendency to spread beyond the site of the contactant (e.g., out to the upper thighs and to the suprapubic area).

An acute reaction to a strong antigen like poison ivy results in severe swelling, vesiculation, oozing, and erosion. There can be extensive secondary changes with excoriations, serosanguineous crusting, and even bruising. The open, eroded areas will burn. The morbidity can be severe. The reaction starts at the site of exposure, but can spread with linear vesicular streaking and can become generalized to any other part of the body. If one reacts to an antigen that previously caused a contact dermatitis, then the reaction will be not only at the site of antigen exposure, but also at any other sites on the body which were previously involved (e.g., the earlobes or wrists).

In subacute or chronic allergic contact dermatitis, the symptoms and signs will be quieter. Pruritus and burning will be subdued. The patient

**Fig. 3. Subacute contact dermatitis with erythema, and swelling of labia majora and minora caused by benzocaine.**

**Fig. 4. Subacute to chronic allergic contact dermatitis with diffuse erythema and swelling superimposed upon lichen sclerosus. Note the loss of the clitoris and labia minora caused by scarring, but no typical whitish changes are visible because of the erythema from the formaldehyde contact dermatitis.**

may wake up at night scratching. The vulva will show the changes as described in the irritant dermatitis section with redness, excoriation, scaling, and altered pigmentation with variable lichenification. Secondary infection can be seen with pustules, fissures, and seropurulent oozing and crusting. As mentioned above, vulvar allergic contact dermatitis may be the primary problem or a secondary one in a patient with another vulvar condition. The clinical picture will then be a combination of several conditions, testing one's diagnostic abilities (Figs 3 and 4).

The histology of allergic contact dermatitis is the same as for the irritant type, except there can be more dramatic spongiosis in acute cases.

The diagnosis is made from the history and the clinical picture. A biopsy can be helpful to rule out other conditions. All efforts are directed at finding the offending allergen. This can be time-consuming. One must be very thorough, following the same questioning as for the irritant contact dermatitis. Good detective work requires the patient and the physician to work very closely together. Lists of all possible contactants are needed, including products used by partners. It often takes more than one or two visits to compile the potential allergen list. Patients are usually adamant that they have used "nothing new". They will often list multiple possible products, but will consistently miss the important one. Review and re-review what they use to clean and lubricate, for example. Patch testing is vital. The North American Contact Dermatitis Group series of 50 allergens is recommended, plus the corticosteroid series, and if indicated, a medication series. Extra series of patch tests may be useful for specific medicaments, perfumes, preservatives, and even emulsifiers (31). Tests are best read at 48, 72 and 96 h. All this may require the services of a dermatologist specializing in contact dermatitis.

The differential diagnosis is the same for irritant vulvar contact dermatitis with the addition of extensive extra-mammary Paget's disease.

Treatment is the same as for vulvar irritant contact dermatitis (see above plus Table 2).

## Summary

Contact dermatitis of the vulva is a common problem. Both irritants and allergens play a role, but often overlap. The appearance of the vulva is often of no help. There may be an underlying dermatosis with or without secondary infection to further complicate the picture. The inflamed vulva appears to be at risk of sensitization in much the same way as the lower leg in stasis dermatitis (32).

For all eczematous vulvar conditions, it is best to assume that an irritant (with or without allergens) is at work. Based on this as a general principle for managing vulvar patients, all irritants must be eliminated from the outset and patch testing must be organized so that allergens are identified and avoided. New reactions to "old" allergens may occur at any time, so when following vulvar patients, be vigilant in reassessing the role of contact dermatitis. It can recur, and an outbreak caused by a new allergen seriously complicates management (see Table 5). Contact dermatitis is the component of vulvar dermatoses that is most

**Table 5.** Tips on vulvar contact dermatitis

- The history of the contactant may be difficult to elicit
- Irritant contact dermatitis is the most common form, especially in those exposed to moisture
- Excess soap and water strip the skin's protective barrier
- Urine and feces burn, enzymatically and/or chemically
- Patients with inadequate estrogen have a less-effective barrier and are at more risk
- Patients with an existing dermatosis (e.g., lichen sclerosus) are at higher risk of sensitization
- Suspect allergic contact dermatitis with sudden onset of intense itching, and/or vesiculation and weeping
- Always stop all vulvar irritants
- Always patch testing to rule out or define role of allergens
- Continue to reassess vulvar patients for contact dermatitis

often overlooked. Detecting and eliminating the offending substance is rewarding for both patient and physician.

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