Vulvar Dermatoses: A Practical Approach to Evaluation and Management

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ABSTRACT

- *Objective:* To present a practical clinical approach to evaluation of vulvar dermatoses and general treatment principles.
- *Methods:* Review of the literature.
- *Results:* The presentation of skin diseases on modified mucous membranes is often nonspecific, and multifactorial processes are common. Determining the diagnosis and the required treatment can be difficult. The majority of patients presenting with a vulvar skin condition will complain of either pruritus or some degree of pain or irritation. An investigation to determine the diagnosis is best accomplished by obtaining a thorough history, performing a detailed physical examination, utilizing appropriate laboratory studies, considering a broad differential diagnosis, and conducting periodic re-evaluations as required. Treatment principles include restoring the skin barrier, reducing inflammation, symptomatic relief, and preventing and treating secondary infection. Patients with chronic vulvar dermatoses require long-term treatment and follow-up to prevent complications associated with the disease process and treatment.
- *Conclusion:* The symptoms associated with vulvar skin dermatoses are distressful to patients.The culmination of clues from a careful history, physical examination, and laboratory testing ideally provide a clinical diagnosis that responds to appropriate treatment.

The symptoms associated with vulvar skin dermatoses, primarily pruritus, irritation, and pain, are distressful to patients. Although the true prevalence of vulvar dermatoses is unknown, it is well accepted that vulvar symptoms are a common problem for women [1–3]. The social taboos associated with medical conditions affecting the vulva as well as the position of this unique skin surface between multiple medical specialties are general obstacles to research and studies. Moreover, the genital location itself is consequential for both the patient with these symptoms and the medical providers caring for such patients.

Unlike other areas of the skin, the vulva is difficult for the patient to examine herself, and compared to an area like the scalp or back, it is awkward to ask a family member or friend to help. Additionally, genital skin symptoms often trigger concerns of poor hygiene, sexually transmitted infections, or undiagnosed cancer, all of which can elicit embarrassment, fear, and anxiety [4]. Many women delay seeking care from medical providers, as they assume that the symptoms are caused by a yeast infection or an allergic reaction to clothing, a cleansing product, or a personal hygiene product [5]. By the time a woman presents to a medical provider, she has likely already changed her hygiene routine, tried multiple home remedies or over-the-counter treatments, and become frustrated and anxious due to the effect these symptoms have had on her daily activities, exercise, and sexual relationships.

Most women with vulvar symptoms present initially to family physicians or gynecologists. However, the primary etiology may be a skin condition rather than a gynecologic disorder. Trained to identify cutaneous disease, optimize barrier function, treat inflammatory skin conditions, and biopsy all skin surfaces, a dermatologist can be integral to the evaluation and treatment of this special population of patients [5–7]. And yet the dermatologist must understand that common dermatology principles are altered in vulvar skin and that gynecologic conditions can significantly impact diagnosis and treatment (eg, morphology changes in moist, mucosal skin; effects of vaginal discharge on vulval skin; secondary vaginal candidiasis). In complicated and chronic cases, a multidisciplinary team is ideal.

For the medical provider, the evaluation is complicated because the genital area is difficult to examine, requiring

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time and effort to adequately inspect genital skin. The examination of the vulva is challenging, as even the normal appearance of the vulva varies with age, hormonal factors, and skin tone [6–8]. Signs of vulvar skin disease are often subtle and difficult to distinguish from variations of normal surface architecture. Compared with hair-bearing skin, the modified mucous membranes of the vulva tend to exhibit erythema in light-complexioned women or hyperpigmentation in darker skin tones. These variations of normal can be interpreted by the patient and examiner as inflammation. Furthermore, clinically minor abnormalities such as subtle erosions or small, healing fissures may be overlooked and yet are often the cause of significant irritation. Reviewing diagrams and clinical photos in texts and atlases of the normal vulvar anatomy, architectural variants, and examples of vulvar dermatoses is recommended [6–11], as is building one's own collection of clinical photos. Additionally, the International Society for the Study of Vulvovaginal Disease (ISSVD) website (www.issvd.org) is a reliable resource for patients and providers.

The diagnosis of vulvar symptoms is also complicated by the fact that multiple inflammatory skin conditions tend to cause similar clinical findings and that the presentation of vulvar dermatoses differs from that of the same disease appearing on other skin surfaces. The classic cutaneous presentation of scale, which often is utilized to differentiate common dermatoses, is altered in the vulva due to warmth, moisture, and friction as well as due to the transition from hair-bearing cutaneous skin to mucosal skin [6–8,12–14]. For example, the moisture of the modified mucous membranes can make the thickened keratin of eczema, lichen simplex chronicus, lichen sclerosus, human papilloma virus, and squamous cell carcinoma appear white and clinically indistinguishable. Additionally, the evaluation of the underlying vulvar dermatosis is frequently complicated by a secondary infection, contact dermatitis from excessive hygiene practices or previous treatments, or secondary skin changes such as lichenification and excoriation due to rubbing or scratching [6–8,15]. Therefore, a multifactorial process should always be considered.

The majority of patients presenting with a vulvar skin condition will complain of either pruritus or some degree of pain or irritation. Vulvar pruritus describes an itch that produces a desire to scratch or rub and feels good when scratched. Vulvar pain describes a sensation in the affected skin that may be described by patients as soreness, rawness, prickling, or burning and does not evoke a desire to scratch. It is not uncommon for patients to report vulvar pain occurring as a result of rubbing or scratching what was first perceived as pruritus. Defining the original symptom can be helpful in differentiating the underlying disease process. It is essential to note that vulvar pruritus and vulvar pain are symptoms and not diagnoses. An investigation to determine the diagnosis is best accomplished by obtaining a thorough history, performing a detailed physical examination, utilizing appropriate laboratory studies, considering a broad differential diagnosis, and performing periodic re-evaluations as required.

HISTORY TAKING

Obtaining a full history regarding vulvar skin symptoms is critical to making the correct diagnosis. A questionnaire completed by the patient prior to seeing the provider (**Table 1**) can be very helpful in guiding the patient to report pertinent historical points and may be used as a springboard to an even more in-depth interview. The history should first define the symptom. The questionnaire, or interviewer, can direct the patient by offering descriptors such as itch, burn, rawness, soreness, and pain. The patient should grade the severity on a scale of 0 to 10, with 0 indicating no symptoms and 10 indicating most severe symptoms. Establishing such a baseline can be helpful during reassessments. Next, the history should define the timeline of symptoms as well as the temporality, location, triggers, and associations of each symptom.

A careful history of vulvar care regimens and treatment should be elicited. Ask the patient to list prescribed and over-the-counter treatments, length of use, and treatment outcomes. Inquire about personal hygiene routines and products, including soaps, douches, use of washcloths, baby wipes, lubricants, moisturizers, and sanitary products, and determine how and how frequently these products are used. These details can be critical in diagnosing a contact dermatitis. Gather additional details regarding pertinent medical and sexual history, conduct a review of systems, and identify pre-existing conditions [7,12,14].

PHYSICAL EXAMINATION

The physical exam requires that the patient undress for a full mucocutaneous exam, which includes all the skin, conjunctiva, oral mucosa, and genitalia—ideally with a chaperone or assistant. Initially, while the patient is standing up or sitting on the exam table, look for stigmata of skin disease (eg, eczema, psoriasis, derma-

(continued on page 210)

Figure 1. Eczematous dermatitis—symmetrically distributed erythematous patches.

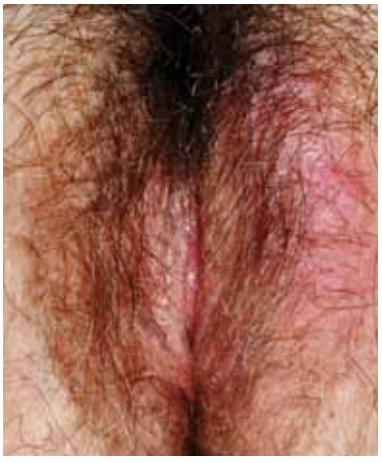

Figure 2. Eczematous dermatitis—unilateral erythematous plaque. Notice the focal decreased hair density overlying the plaque secondary to rubbing or scratching.

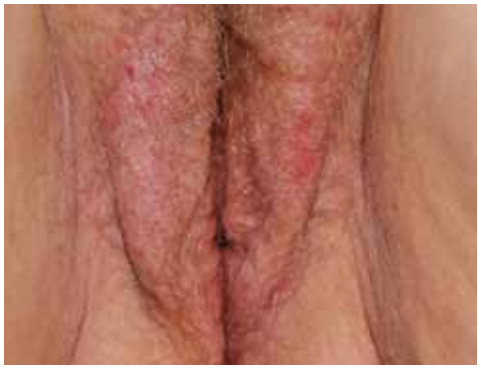

Figure 3. Lichen simplex chronicus—bilateral erythematous, lichenified plaques with excoriation.

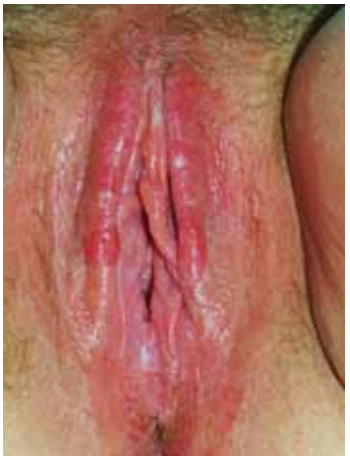

Figure 4. Contact dermatitis—erythematous, edematous plaques with multiple erosions.

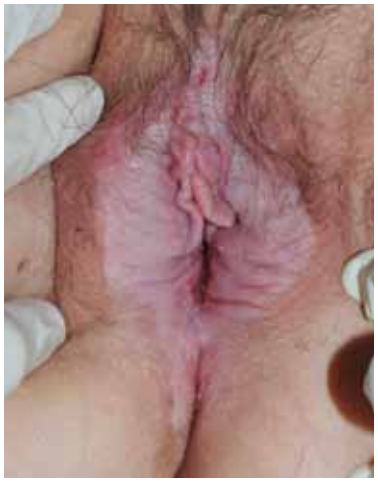

Figure 5. Lichen sclerosus—confluent hypopigimented plaque extending from the vulva to the peri-anal skin (figure eight distribution) notable for shiny, crinkled atrophy with fissure superiorly and secondary thickening from scratching over the labia majora.

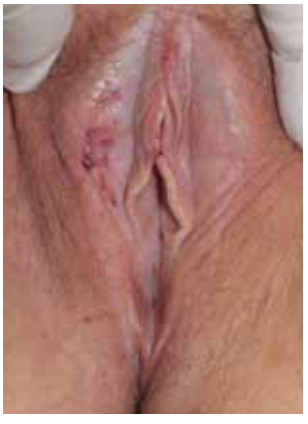

Figure 6. Lichen sclerosus—shiny, atrophic, hypopigmented plaque affecting the clitoral hood, intra-labial sulcus, and posterior vulva with purpura and erosion.

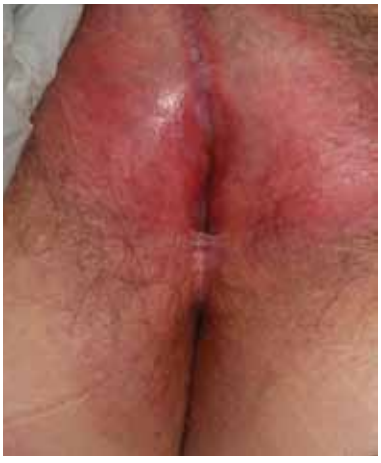

Figure 7. Lichen planus—erythematous, glazed, erosive plaque with agglutination and adhesions that narrow the introitus.

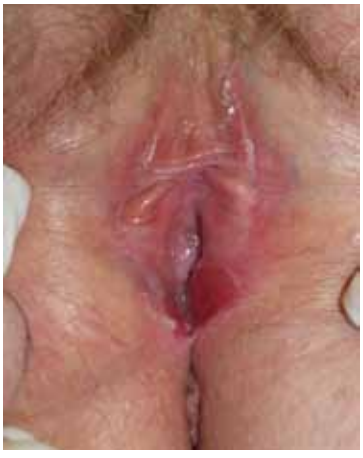

Figure 9. Lichen planus—erythematous plaque with large erosion of the posterior vestibule, resorption of the labia minora, and fusion of the clitoral hood. Note the white peripheral rim surrounding the erosion.

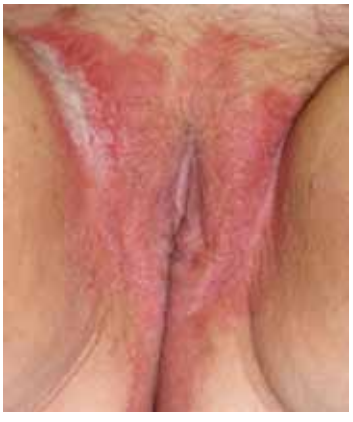

Figure 11. Psoriasis—erythematous plaques with white macerated scale and erosions overlying the inguinal creases and labia majora as well as fine white scale along peripheral rim.

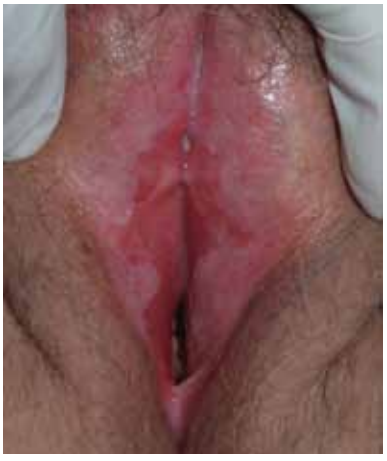

Figure 8. Lichen planus—erythematous plaque with central erosion and peripheral lacy white striae as well as loss of normal architecture due to resorption of labia minora and clitoral hood.

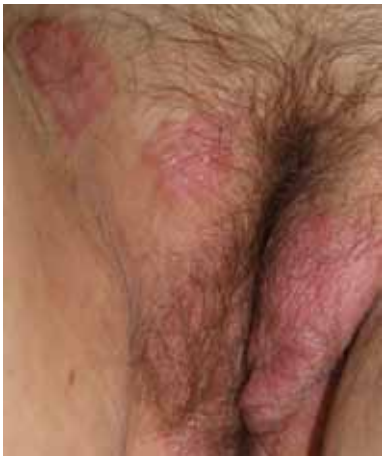

Figure 10. Psoriasis—erythematous plaques with minimal scale.

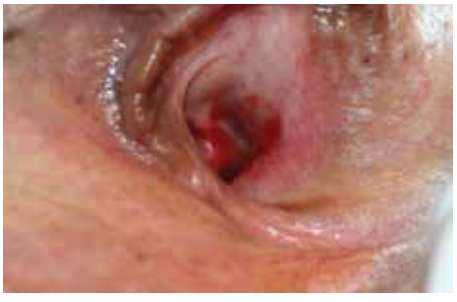

Figure 12. Plasma cell vulvitis—erythematous-brown glistening plaque.

Table 1. Historical Information to Obtain from Patient

Age
Allergies
Medications
Previous surgeries

Describe your discomfort:
    Itch, rawness, soreness, burning, other _____
Do you feel an urge to scratch your skin? Yes/No
When did your symptoms start?
Are your symptoms continuous or do they come and go?
Do any particular triggers make the symptoms worse such as sexual activity, menses, exercise, other? _____
Have you noticed any change in vaginal discharge? Yes/No
    If yes, please describe _____
What have you tried to treat your symptoms? _____

Have you ever had a vulvar biopsy? Yes/No
    If yes, please obtain report and bring to appointment

Last menstrual period _____
Most recent pregnancy _____
What sanitary products do you use during your menses?
Panty liners, pads, tampons; scented vs unscented
Brand(s) _____

Experienced menopause at age _____
Do you take hormone replacement? Yes/No
    If yes, orally, intra-vaginally, transdermal patch?

What do you apply your genital skin? (Circle all that apply)
    Water only, cleanser, soap, washcloth, powders, moisturizers, sprays, creams, ointments, other _____
List previous treatments including start date, end date, why stopped, effect on symptoms

Are you sexually active? Yes/No
    If yes
        How often do you have intercourse? _____
        Do you have pain with intercourse? Yes/No
        If yes, when? during penetration, during/after intercourse?
        Do you use any lubrication products? Yes/No
        Which brand, why? _____
        Do you use any type of contraception? Yes/No
        If yes – condoms, lubricants, diaphragm, other _____
        Which brand? How often? _____

Have you ever been told that you have or had:
    Abnormal Pap smear? Yes/No
    Genital warts? Yes/No
    Genital herpes simplex virus infection? Yes/No
    Herpes zoster? Yes/No

Do you have a history of any of the following? Please circle.
    Allergic rhinitis, eczema, asthma, psoriasis
    Diabetes, irritable bowel syndrome
    Fibromyalgia, interstitial cystitis, chronic fatigue
    Family history of psoriasis, eczema, or genital skin problems?

Do you experience a significant problem with any of the following? Please circle.
    Sleep disturbance, headaches, low energy, fatigue,
    Anxiety, depression
    Irritation or dryness of eyes or mouth, mouth sores
    Diarrhea, constipation, reflux symptoms
    Pain with urination, urinary frequency, incontinence
    Joint pain, back pain

What do you think is causing your symptoms?
How are your symptoms affecting you?

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tophytosis, and lichen planus) that may play a role in vulvar symptoms.

Position the patient to allow adequate exposure and lighting to examine the entire anogenital area. Use the physical exam to further pinpoint symptom location and to teach the patient anatomical terms. Ask the patient to point to or touch the area of her discomfort and, if applicable, show where and how she has applied treatments. Consider taking clinical photos for the medical record. Photos can be used to objectively assess treatment during follow-up evaluations, and printouts can be used to teach anatomy and to instruct patients on how and where to apply medications.

Inspect all surfaces of the vulva for subtle erythema, swelling, lichenification, hyper- or hypopigmentation, fissures, excoriation, erosions, tumors, atrophy, and the presence of scarring. Vulvar agglutination describes a form of scarring associated with loss of tissue mass or normal architecture. Any inflammatory condition can cause vulvar agglutination, which may be recognized clinically as resorption of the labia minora, fusion of the clitoral hood, or adhesions that narrow the introitus [7,14]. A skin potassium hydroxide (KOH) prep or culture should be performed on any erythematous, scaling plaque or any intertriginous plaque to diagnose or exclude dermatophytosis. Dermatophyte infections of anogenital skin, however, are uncommon in women and are often accompanied by dermatophyte infection on the feet and toenails.

Utilize a speculum to visualize the vaginal mucosa for erythema, erosions, and synechiae. To minimize patient discomfort, the use of a small or pediatric speculum is recommended. Alterations to the normal physiologic vulvar discharge due to any type of vaginitis or erosive vaginal skin diseases can cause or worsen a vulvar condition. A sample of the vaginal secretions should be studied microscopically via saline wet mount and with KOH to characterize the epithelial cells and to identify the presence of white blood cells, clue cells, lactobacilli, hyphae, or budding yeast. As needed, vaginal cultures should be ordered. Fungal cultures can be critical to confirming the diagnosis of candidiasis and speciating yeast. This is especially relevant in the setting of chronic symptoms that may be caused by resistant, non-*albicans Candida,* which requires nontraditional antiyeast treatment [16,17].

A biopsy is warranted for any abnormal findings that cannot be defined clinically, do not respond to treatment as expected, or are suspicious for malignancy. The biopsy specimen should be of a specific lesion, such as a white or hypopigmented plaque, or the edge of an erosion, to include a sample of normal skin. In most cases a shave biopsy yields an adequate specimen. It can, however, be technically difficult to perform a shave biopsy on the vulva due to the moist, pliant skin. A preferred method described by Edwards uses a suture to tent the skin followed by scissor snip under local anesthesia [8]. This technique allows the provider to control the size and depth of the specimen and avoids crushing the tissue with forceps.

Biopsy results from genital skin can be nonspecific. If there are multiple morphologies, taking biopsies from multiple sites may increase the likelihood of a diagnostic biopsy. The discovery of a concerning pigmented lesion, or any lesions suspicious for malignancy, should be biopsied via punch technique or removed by excision to allow full-thickness histologic evaluation. In cases of a blistering or erosive condition with a nonspecific initial biopsy, a second biopsy from adjacent normal-appearing (perilesional) skin can be sent in Michel's solution for direct immunofluorescence to aid in diagnosing vesiculobullous disease [18]. All specimens of suspected vulvar dermatoses should be sent along with an informative history and differential diagnosis to a dermatopathologist.

APPROACH TO DIFFERENTIAL DIAGNOSIS

As noted previously, diagnosing vulvar skin dermatoses is complicated. The differential diagnosis associated with vulvar pruritus and vulvar pain includes myriad skin conditions, neoplasms, infections, infestations, and systemic diseases (**Table 2**) [16,19–36]. Certainly, most benign and malignant neoplasms are expected to be biopsied and identified by histology. Additionally, infections and infestations can typically be diagnosed clinically with the aid of cultures and, occasionally, histology. Unfortunately, however, reducing the differential diagnosis to non-neoplastic, noninfectious skin conditions still leaves the clinician with a diagnostic challenge.

Ideally, the culmination of clues from a careful history, physical exam, and lab testing provide a clinical diagnosis that responds to appropriate treatment. Often, however, despite an adequate evaluation, the cause of vulvar symptoms cannot be clearly identified due to nonspecific clinical findings, mixed signs and symptoms that allude to multifactorial processes, or simple lack of objective disease. In such a case, a trial of treatment aimed at restoring the skin barrier, minimizing inflammation, and

addressing infection as warranted is a reasonable initial approach and is discussed in detail below.

If, however, there are objective clinical findings, a biopsy may confirm a specific diagnosis or aid in narrowing the differential by rendering a pattern of inflammation. A recent advance in the diagnosis of vulvar dermatoses is the standardization of nomenclature and classification by pathohistologic features presented by the ISSVD [37]. The ISSVD classification of pathologic subsets correlates patterns of inflammation with clinical diagnoses, which the clinician can use to determine the most likely diagnosis (**Table 3**). A thorough review of the histology of vulvar inflammatory dermatoses is beyond the scope of this article and readers are referred to a recent review by Selim et al for additional information [18].

This article examines the most common of the vulvar dermatoses noted in the ISSVD classification scheme, including atopic dermatitis, lichen simplex chronicus, contact dermatitis (allergic and irritant), lichen sclerosus, lichen planus, and psoriasis. It also examines those rare conditions that present almost exclusively on the vulva: plasma cell vulvitis and papular genitocrural acanthosis. The goal of this section is to present key concepts in the clinical evaluation and management of patients with these vulvar dermatoses. The pathogenesis, clinical presentation, and diagnostic features of each condition are first surveyed, then management and treatment recommendations are discussed.

The skin conditions of the ISSVD classification scheme that present as genital ulcers, apthae, and Behcet's disease are not included in this review as the evaluation of a genital ulcer as a primary lesion has a distinct differential diagnosis and evaluation that is well described elsewhere [38,39]. Additionally, although systemic diseases such as Crohn's disease and Melkersson-Rosenthal syndrome may rarely present initially with only vulvar symptoms, clues from the history, presentation, and disease progression should prompt appropriate diagnostic suspicion and evaluation and these conditions are not reviewed here. Finally, cicatricial pemphigoid, linear IgA disease, Hailey-Hailey disease, and Darier's disease are not included because diagnostic lesions outside the location of the vulva are expected.

Table 2. Differential Diagnosis of Vulvar Pruritus and Vulvar Irritation or Pain

Cutaneous disease
Atopic or eczematous dermatitis
Lichen simplex chronicus
Allergic contact dermatitis
Irritant contact dermatitis
Lichen sclerosis
Lichen planus
Psoriasis
Plasma cell vulvitis
Papular genitocrural acanthosis
Vaginitis, vaginosis
Atrophic vulvovaginitis
Candida vulvovaginosis
Bacterial vaginitis
Desquamative inflammatory vaginosis
Infectious causes
Fungal – Candidiasis, tinea cruris
Bacterial – Group A streptococcus, *Staphylococcus aureus*, *Trichomonas vaginalis, Neisseria gonorrhea*, *Chlamydia trachomatis*
Viral – Herpes simplex virus, human papilloma virus, herpes zoster virus, molluscum contagiosum
Infestations – Scabies, lice, enterobiasis, threadworm
Neoplasms
Vulvar intraepithelial neoplasia
Squamous cell carcinoma
Extramammary Paget's disease
Syringomas
Mammary-like gland adenomas (Hidradenoma papilliferum)
Langerhan's cell histiocytosis
Basal cell carcinoma
Systemic diseases
Pemphigoid, cicatricial typle
Behcet's disease
Crohn's disease
Systemic lupus erythematosus
Acrodermatitis enteropathica
Idiopathic
Vulvodynia – generalized, localized
Dermatographism
Drug eruption

Eczematic Dermatitis

Atopic, or eczematous, dermatitis refers broadly to a spectrum of very common, acute and chronic, pruritic skin conditions. Patients with an atopic diathesis have a tendency to have allergies, asthma, and eczema and are more inclined to perceive a sensation of pruritus and scratch the skin in response. The classic atopic dermatitis patient presents in childhood. Although eczematous dermatitides occur more frequently in patients with a per-

Table 3. Modified Version of the 2006 ISSVD Classification of Vulvar Dermatoses: Pathologic Subsets and Their Clinical Correlates

Histologic Pattern	Diagnosis
Spongiotic	Atopic dermatitis
	Allergic contact dermatitis
	Irritant contact dermatitis
Acanthotic	Lichen simplex chronicus (primary and secondary)
	Psoriasis
Lichenoid	Lichen sclerosus
	Lichen planus
Dermal homoginization/ sclerosis	Lichen sclerosus
Vasculopathic	Plasma cell vulvitis
	Aphthous ulcers
	Behcet's disease
Acantholytic	Papular genitocrural acantholysis
	Hailey-Hailey disease
	Darier's disease
Vesiculobullous	Pemphigoid, cicatricial type
	Linear immunoglobulin A disease
Granulomatous	Crohn's disease
	Melkersson-Rosenthal syndrome

Data from reference 37.

sonal or family history of atopy, such eruptions also are common without an atopic history. Heat, friction, sweat, and mild trauma are common triggers for pruritus in all skin surfaces and are especially significant in the vulva.

An eczematous dermatitis can present acutely as red, edematous plaques with vesicles; subacutely as erythematous patches or plaques; or chronically as subtle accentuated skin markings or a thickened, lichenified plaque (**Figure 1, Figure 2**). Eczematous dermatitis occurring outside the vulva is most commonly diagnosed clinically by the typical history and physical exam findings [40]. As discussed previously though, due to the moist skin and friction of the vulva, the physical findings in this area tend to be less diagnostic. A biopsy of eczematous dermatitis may show a spongiotic inflammatory pattern supporting a diagnosis of eczematous dermatitis, but this pattern also is seen in contact dermatitis. Eczematous dermatitis generally is treated with topical corticosteroids and changes to skin care that repair and maintain the barrier function. Specific treatment principles, with an emphasis on variations specific for vulvar skin, are discussed in the next section.

Lichen Simplex Chronicus

Lichen simplex chronicus (LSC) is a localized plaque of chronic eczematous inflammation created by repeated rubbing or scratching of the skin in response to a sensation of pruritus. While this rubbing and scratching yields relief and feels pleasurable, the act of rubbing and scratching produces more irritation and more itching, giving rise to a phenomenon often referred to as the itch/scratch cycle. It is because of this cycle that the scratching becomes habitual and recurrence is common. While LSC, as a focal, chronic subtype of eczematous dermatitis, is a common cause of primary vulvar pruritus, it may also be secondary to any cause of vulvar irritation that triggers rubbing and scratching [41–43].

The hallmark of vulvar LSC is focal, intractable pruritus that is not expected to involve mucosal membranes. Clinically, the provider may observe little objective change to the skin, but more commonly chronic irritation from the rubbing and scratching produces inflamed, thickened, excoriated plaques (**Figure 3**). At times, the skin becomes so inflamed and excoriated that the patient's presenting complaint may be of vulvar pain rather than the initial triggering pruritus. If the characteristic thickening of the skin, referred to as lichenification, is present, the diagnosis can be made clinically. If, however, the clinical findings are nonspecific, the differential diagnosis of any thickened plaque must include eczema, lichen sclerosus, lichen planus (hypertrophic type), human papilloma virus, and squamous cell carcinoma and may be differentiated histologically. LSC is treated in the same manner as eczematous dermatitis with an additional emphasis on extinguishing habitual scratching and nighttime sedation as needed [44].

Contact Dermatitis

Contact dermatitis presents clinically like an eczematous dermatitis and can have the same acute, subacute, and lichenified forms, but arises as a result of contact with an irritant or allergen (**Figure 4**). Any skin with

disrupted skin barrier function is vulnerable to irritants and allergens, but the vulva is specifically more sensitive to irritants and allergens than skin elsewhere on the body because of its occlusion from natural skin folds, sanitary products, and clothing; hydration secondary to perspiration and vaginal discharge; and susceptibility to friction from clothing and activity [45–47]. Like LSC, contact dermatitis can be a primary diagnosis, as well as a secondary, complicating factor.

Allergic contact dermatitis (ACD) is a delayed response that requires prior sensitization. Acute ACD is suspected on the basis of sudden onset of agonizing itching and vesiculation or erosion with exudation. The classic example of acute ACD is caused by poison ivy or poison oak. The diagnosis of chronic ACD, however, is more difficult, as both the history and clinical findings can be indistinguishable from, and overlap with, chronic irritant contact dermatitis (see discussion below) and LSC. A diagnosis of ACD should be suspected in patients who apply multiple agents to their skin and whose pruritus does not respond to usual therapy [42].

ACD is diagnosed by patch testing and should be ruled out in any recalcitrant chronic or recurrent vulvar dermatosis. In cases where a particular product is suspected to be the trigger, provocative use testing can be helpful. In provocative use testing, a tiny amount of suspected trigger is rubbed on the ventral forearm multiple times daily for 5 days. If erythema develops, the test is positive. Note, however, that a false negative provocative use test may occur in cases where the unique vulvar environment is integral to the reaction. Potential sensitizers most relevant to the vulva include topical anesthetics (particularly benzocaine, which is found in Vagisil), topical antibiotics (particularly neomycin, bacitracin, polymyxin), topical antifungal imidazole creams, topical corticosteroids, fragrances, preservatives (pervasive in personal care products, topical medications, and moistened towelettes), rubber (such as latex in condoms and elastic or spandex in underclothing), and nickel (via direct contact or transferred by hand from snaps, buttons, jewelry, etc.) [47–50]. The primary treatment of ACD is avoidance of the triggering allergen and restoration of the skin barrier as detailed in the next section.

Irritant contact dermatitis (ICD) is a nonspecific reaction that can occur minutes to hours after exposure to a strong irritant such as urine, feces, or topical medications, or as a result of repeated exposure to milder irritants, such as soaps, which may damage the skin barrier [45–47]. Of course, sensitivity to any irritant is increased once the skin barrier is compromised. Overwashing, shaving, application of irritating hygiene products (especially alcohol-based creams and gels), lubricants, spermicides, douches, and urinary and fecal incontinence can cause vulvar inflammation that presents as pruritus or irritation [42,47,51]. ICD should be suspected in any patient who has an extensive hygiene routine. Physical irritation from the friction of scrubbing, tight and occlusive clothing, and intercourse also can cause an irritant dermatitis. The cornerstone to treating ICD consists of avoiding all possible irritants and restoring the integrity of the skin's barrier function [45–47]. To do this, it is important to inquire about incontinence, hygiene products, and cleansing habits and to give patients educational instructions on how to clean the vulvar area without causing irritation as outlined below.

Vulvar ICD due to alterations in vaginal secretions associated with inflammation or erosive disease of the vagina, estrogen deficiency, candidiasis, or bacterial vaginitis is common. Thus, examining the vaginal secretions via wet mount and KOH prep is critical to a comprehensive evaluation. A complete discussion of these conditions is beyond the scope of this paper, but readers are referred to the following citations for more information [16,22,23]. While vulvovaginal candidiasis is the most common cause of acute onset vulvar pruritus, presumptive diagnosis and empiric treatment by clinicians must be avoided, especially in women with chronic vulvar pruritus, as many genital skin problems are mistakenly attributed to candidiasis. Furthermore, providers should educate patients that a yeast infection is only one of a number of causes of vulvar pruritus and should recommend in-office evaluation of all vulvar symptoms to avoid missed and delayed diagnoses.

Lichen Sclerosus

Lichen sclerosus (LS) is a chronic inflammatory condition, generally accepted to be an autoimmune disorder, with a complex pathogenesis that may be influenced by additional genetic, hormonal, and infectious factors [52–55]. LS affects both sexes and all areas of the body. It is, however, markedly more common in women, manifesting most commonly on the vulva. A minority of patients with genital LS also have extragenital cutaneous lesions that resemble morphea [55]. Yet LS only rarely affects the oral mucosa, and involvement of the vagina is not expected. Onset can occur at any age, but notable bimodal peaks are seen at times of low estrogen

in prepubertal girls and menopausal women. Most patients present complaining of vulvar pruritus or irritation. LS can be asymptomatic, however, and should not be overlooked on routine exam. Physical examination reveals atrophic, hypopigmented-to-white, crinkled, fragile plaques classically distributed in a figure 8 pattern around the vulva, perineal body, and perianal skin (**Figure 5**). Scratching may lead to thickening of the skin and secondary lichenification, which can mask the classic atrophic appearance. As the disease progresses, the symptoms may shift from pruritus to pain, including dyspareunia, dysuria, and pain with bowel movements, which can be associated with a late physical finding of purpura, erosions, resorption of the labia minora, and fusion of the clitoral hood (**Figure 6**) [14,52–55].

Although classic presentations of vulvar LS may appear to be clinically evident, a biopsy is generally recommended to confirm the diagnosis. Clinical presentation may be misleading, as white plaques, erosions and scarring arising from any inflammatory vulvar condition can be clinically indistinguishable from LS. Moreover, because the chronic nature of the LS diagnosis commits the patient to long-term treatment, pathologic correlation is prudent [55,56]. In contrast, confirmational biopsy in children is not typically recommended, as the classic presentation in children is likely to be more reliable. In the pediatric population there is a tendency toward earlier presentation for medical care, less frequent secondary complications, and a narrowed differential diagnosis. Readers are referred to the following citation for more information on pediatric LS [57].

Patients with LS have an increased incidence of other autoimmune diseases, particularly thyroid disease, vitiligo, pernicious anemia, and alopecia areata [58]. As such, a directed review of systems is appropriate, as is consideration of screening labs. Approximately 2% to 5% of women with vulvar LS will develop vulvar squamous cell carcinoma, which may be prevented with appropriate long-term treatment with topical steroids or diagnosed early with routine, scheduled surveillance [59,60]. The essential components of managing vulvar LS include controlling symptoms, minimizing scarring, and preventing, or detecting early, malignancy.

Lichen Planus

Lichen planus (LP) is another chronic inflammatory dermatosis considered to be an autoimmune condition. LP can affect the mucous membranes of the vagina, conjunctiva, esophagus, urethra, and anus as well as cutaneous skin, scalp, and nails. Vulvovaginal LP most commonly presents in postmenopausal woman but can occur earlier in adult women and on rare occasions in children. A typical presentation is a menopausal woman reporting vulvovaginal pain or pruritus, dyspareunia, and an irritating vaginal discharge. Physical exam may reveal 1 of 3 morphologies or a combination of the 3: erosive, glazed, or glossy erythematous plaques (**Figure 7**) (most common morphology occurring in the vulva); classic erythematous-to-violaceous papules with an overlying lacy, white, reticulate striae (**Figure 8**); or uniformly white, hyperkeratotic plaques (**Figure 9**) [53,61]. The inflammation associated with LP often causes resorption of the labia minora and fusion of clitoral hood. Gentle speculum exam of the vagina may reveal erythema, erosions, scarring, and an inflammatory discharge. Vaginal LP is most commonly seen in woman with the erosive-type vulvar LP. Examination of oral mucosa can yield significant diagnostic clues, as many women with vulvar LP also have evidence of oral LP on examination. Oral LP, which can be painful or asymptomatic, may manifest as erosions, reticulate striae on the buccal mucosa, or gingival inflammation [53,61,62].

Vaginal involvement can distinguish vulvar LP from vulvar LS and LSC since the latter two are not expected to affect the vagina [53]. The differential diagnosis of erosive vulvovaginal disease also includes mucosal vesicular diseases (eg, cicatricial pemphigoid, pemphigus vulgaris, fixed drug eruptions, toxic epidermal necrosis, and herpes simplex virus), and hyperkeratotic lesions must be differentiated from human papilloma virus and neoplasia. These diagnoses usually can be discerned by the history and presence of extragenital lesions as well as histopathology and staining on direct immunofluorescence.

Patients with vulvar LP have a comparable association with other autoimmune conditions [58] and risk of developing vulvar squamous cell carcinoma as those with LS. The development of malignancy and scarring associated with either LS or LP can be asymptomatic, necessitating long-term follow-up. LP is treated primarily with superpotent topical steroids in a regimen similar to that described above for LS. Vaginal inflammation and the significant potential for sexual dysfunction, however, must be addressed and is discussed in the next section.

Psoriasis

Psoriasis is a chronic, immunologically mediated skin condition of rapid epithelial turnover resulting in

characteristic, well-demarcated, erythematous plaques with silvery scale. Identifying classic lesions of psoriasis elsewhere on a full-body skin examination can focus the differential diagnosis. Look for psoriatic plaques in the typical locations, including the elbows, knees, and scalp, and for psoriatic nail changes, including pitting, oil spots, and onycholysis. Alternatively, and less common, an inverse distribution pattern of psoriasis may affect the skin folds (eg, the gluteal cleft, umbilicus, axilla, and groin). Due to the moisture and friction of skin folds, the classic psoriatic lesion is replaced with a poorly demarcated, erythematous plaque with minimal scale and shiny texture (**Figure 10, Figure 11**). Again attributed to moisture and friction, psoriatic lesions located in skin folds are more likely to be associated with pruritus [63]. If there are no diagnostic clues on exam, a biopsy may be required to confirm the diagnosis of suspected vulvar psoriasis. The treatment algorithm for vulvar psoriasis is the same as that for psoriasis on other parts of the body with modification of topical treatments for the vulvar environment as discussed below.

Other Dermatoses

Plasma cell vulvitis and papular genitocrural acanthosis are rare vulvar dermatoses. Plasma cell vulvitis (Zoon's vulvitis, vulvitis circumscripta plasmacellularis) is a benign, chronic disease of unknown etiology. It most frequently presents clinically as multiple, fixed, red-to-orange or brown glistening plaques on the vestibule that may be asymptomatic, pruritic, or painful (**Figure 12**) [20,64]. A biopsy may yield a characteristic dense plasma cell infiltrate. Superpotent topical steroids can improve symptoms. Papular genitocrural acanthosis, which also is known as papular acantholytic dyskeratosis, presents clinically as pruritic, discrete skin-colored, white, or dully erythematous verrucous papules coalescing into plaques that are localized to the anogenital region [21]. Although clinically these lesions may mimic genital warts, diagnosis can be made by histologically [65].

MANAGEMENT

A general treatment approach, including restoring the skin barrier, reducing inflammation, symptomatic relief, and preventing and treating secondary infection has the potential to address vulvar symptoms caused by eczematous dermatitis, lichen simplex chronicus, and allergic or irritant contact dermatitis [66,67]. This approach also may significantly improve symptoms of lichen sclerosus, lichen planus, and psoriasis [68]. These latter conditions are chronic, however, and therefore likely to recur when topical steroid treatment is discontinued. Ongoing topical steroid treatment and follow-up is required to manage the chronicity associated with these diseases.

Restore Skin Barrier Function

Treatment of all vulvar dermatoses begins with restoring the integrity of the skin barrier. The epidermis functions as the primary defense layer to the external environment. Irritants, allergens, inflammation, and physical trauma, such as from scratching, disrupt the epidermis and the function of the skin as a barrier. Barrier dysfunction further permits invasion of microorganisms and penetration of allergens and irritants into the skin, increasing the risk of secondary infection and secondary contact dermatitis. Clinical signs of skin barrier disruption include erythema, linear or angular erosions due to excoriation, fissures, a wet, weeping surface, crust, or scab [69].

Giving both written and verbal instructions regarding gentle skin care, avoidance of irritants, and the proper use of medications, as well as supportive and informational resources is critical to successful treatment. An example of a patient instruction sheet is provided in **Table 4**. Accordingly, instruct the patient to gently clean vulvar skin with her fingertips using only minimal mild cleanser or water alone. There is no need to rub or scrub the genital skin. After bathing, gently pat the skin dry. If a moisturizer is needed, a bland emollient such as a thin film of petrolatum jelly is recommended. Eliminate all potential irritants, including overwashing, personal hygiene products, douches, lubricants, home remedies, and over-the-counter medications. Treat patients who are estrogen-deficient with local estrogen-replacement therapy as atrophic vaginitis is a common primary cause of symptoms as well as an exacerbating problem.

If a particular allergic contact dermatitis trigger is identified by patch testing, direct the patient to the American Contact Dermatitis Society website (www.contactderm.org), which reviews products to avoid as well as safe alternatives. It has been this author's experience that some patients struggle to give up the use of moistened towelettes (baby wipes). There are multiple reports of the preservatives in these products causing allergic contact dermatitis [70–75]. Two of the 5 preservatives implicated are included in the T.R.U.E patch test kit, and the others may be identified via provocative use

Table 4. Example Patient Instructions

1. Restore skin barrier and avoid further irritation

A very important part of the treatment is gentle skin care to avoid irritation. Washing is the most common irritation for skin.

- Wash with warm water using only gentle fingertips rather than rough washcloths. Gently pat skin dry after bathing, and apply a tiny amount of petrolatum jelly/ointment as moisturizer as needed.
- Avoid: hot water, soaps, bubble baths, washcloths, moisturizing creams and lotions
- Avoid: wet wipes, baby wipes, and medicated wipes
- Avoid: medications other than those prescribed above
- Avoid: rough fabric, tight clothing that cause sweating and overheating
- Avoid: panty liners, sanitary pads, douches, powders, perfumes, deodorants
- Avoid: KY and Vagisil brand products due to possible irritant effect
- Before sex, protect your vulvar skin from the irritation of friction by using a thin coating of petrolatum ointment, vegetable oil, Astroglide or Slippery Stuff for lubrication as needed

2. Decrease skin inflammation

- Apply a tiny amount of clobetasol ointment to affected area twice daily. If symptoms resolve, continue to apply the medication, but reduce to once daily for an additional 7 days. If symptoms remain resolved, stop applying medication. Even if symptoms are resolved, it is critical to keep your scheduled follow-up appointment.

3. Treat symptoms of irritation and itching

- Apply a liberal amount of lidocaine 2% jelly as often as needed for discomfort. You also can use cool gel packs and a cool washcloth for comfort as needed.

4. Stop scratching

- Cut your nails. Watch for your triggers and observe for habits.
- Try not to rub or scratch, even with washcloths or towels
- Minimize nighttime scratching. Take amitriptyline 10 mg by mouth 1 to 2 hours prior to bedtime. Start by taking 1 tablet nightly and increase by 1 tablet (maximum 10) each night until sleep is restful and without scratching. If you feel too sedated in the morning, reduce dosage. If your symptoms resolve, this medication can be stopped.

5. Treat and prevent infection

- To treat or prevent bacterial infection, take cephalexin 500 mg twice daily for 10 days
- To prevent a yeast infection while using a topical corticosteroid ointment, take fluconazole 150 mg once weekly

6. Schedule a follow-up appointment in 4 weeks

testing [75]. This author is not aware of any moistened towelettes or medicated wipes commercially available in the United States today that do not contain preservatives, although the preservatives vary among commercial brands. A trial of a fragrance-free product with a different preservative may be worthwhile. The most desirable alternatives, such as sitz baths or gentle blotting with a cloth moistened with water, are time-consuming and cumbersome but may be required for some patients. On raw, tender, irritated skin, even plain water can sting, and homemade normal saline (1 teaspoon of salt dissolved in 4 cups of water) can minimize this [66].

Decrease Skin Inflammation

Concurrent with repair of the skin barrier, reducing inflammation is the second factor in treating vulvar dermatoses—most commonly accomplished by use of topical steroids. Topical steroids in ointment vehicles are preferred to those in cream vehicles, as the latter have a tendency to cause burning on application, especially when the skin barrier is disrupted. The mucosal skin of the vulvar vestibule is relatively resistant to topical steroids and requires superpotent strength for longer treatment periods than nonmucosal skin. In contrast, the hair-bearing surfaces of the vulva, inguinal creases, and medial thighs are more susceptible to atrophy associated with long-term use of topical steroids. Consequently, the use of topical steroids on the hair-bearing surfaces of the vulva must be closely followed. Consider starting with a course of superpotent topical steroid ointment twice daily with specific instructions and a scheduled follow-up appointment in 4 weeks. Explain and demonstrate how to apply a tiny amount of the topical steroid to the specific affected area and emphasize that care should be taken to minimize exposure to the adjacent surfaces. If the patient's symptoms resolve before her follow-up appointment, the frequency of application should be decreased to once daily for a few additional days. If the symptoms remain resolved, instruct the patient to discontinue the topical steroid but to keep the follow-up appointment for re-evaluation. Giving the patient detailed written instructions, prescribing an adequate, but small, 15-g supply, and having a short interval follow-up period can avoid misuse of topical steroids and minimize side effects.

In cases of an acute vesicular eruption or significant erosions, oral steroids 40 to 60 mg each morning for 5 to 10 days or intramuscular kenalog at approximately

1 mg/kg may be preferable to topical treatment to minimize additional maceration of the skin. There are many reports of topical calcineuren inhibitors (TCIs), tacrolimus ointment and pimecrolimus cream, successfully treating vulvar dermatoses [76–78]. Compared to topical steroids as anti-inflammatory agents, TCIs carry the advantage of lack of risk of atrophy, striae, and steroid dermatitis. Commonly, however, these products cause burning and stinging with application. In addition, TCIs cost more, have a slower onset than potent and superpotent topical steroids, and carry black-boxed warning for association with squamous cell carcinoma and lymphoma. Therefore, TCIs should be considered second-line treatment and used when an alternative to topical steroids is required.

While atopic dermatitis and contact dermatitis are expected to resolve relatively quickly with topical corticosteroids, these conditions can be recurrent if the skin barrier is not continually maintained. The thicker, lichenified skin of LSC often requires longer treatment intervals. Additionally, when LSC or contact dermatitis is diagnosed and treated, the patient should be reexamined post treatment, even in the absence of symptoms, to rule out the presence and persistence of a primary underlying primary dermatosis.

Vulvar LS is commonly treated with a superpotent topical steroid, such as clobetasol ointment, once to twice daily followed by reassessment in approximately 4-week intervals. Note that the symptoms of LS usually resolve before all the skin changes clear. The steroid potency and frequency of steroid application should be tapered to the minimal amount needed to control the disease. Patients are typically followed monthly while using daily superpotent topical steroids and then every 3 to 6 months as the treatment is tapered. The goal of treatment is clearance of both the symptoms and the signs of disease. Recalcitrant lesions should be biopsied to rule out squamous cell carcinoma. While some patients experience complete resolution, relapses or flares of symptoms are not uncommon and necessitate return to increased steroid strength and frequency. Long-term maintenance treatment of topical steroid application 1 to 3 times weekly and ongoing follow-up is recommended to minimize recurrence, sexual dysfunction from scarring, and malignancy transformation [60]. Patients may be referred to the National Lichen Sclerosus Support Group (www.lichensclerosus.org). Additional information can be found in the published literature [52,53,55,56,60].

The first-line treatment of vulvar LP is superpotent topical steroids with a long-term focus, as described above for vulvar LS. Erosive mucosal LP tends to be more difficult to control. Vaginal LP requires intravaginal steroid application. Clobetasol ointment 1–2 g can be inserted vaginally using an applicator, intravaginal suppositories can be compounded, or patients can be instructed to insert commercially available 25-mg rectal suppositories intravaginally. Scarring and vaginal stenosis tend not to be reversible, but the use of graduated vaginal dilators coated in topical steroids may improve sexual function. Detailed treatment recommendations and alternatives regarding vulvovaginal LP can be found in the literature [53,61,62]. Any hypertrophic lesion that does not respond to treatment should be biopsied to exclude neoplasia.

Symptom Relief

The third focus of management is supportive measures that address patient symptoms and comfort. Teach the patient about the itch/scratch cycle, and emphasize the importance of refraining from scratching. Recommend cutting fingernails and using lidocaine 2% jelly, cool washcloths, and gel packs to minimize pruritus, irritation, and pain. Nighttime sedation is warranted for those who note that pruritus symptoms are worse in the evening or that they awaken from sleep scratching subconsciously. Tricyclic antidepressants (eg, amitriptyline, doxepine) are favored over sedating antihistamines because they not only produce more deep sleep but may also alleviate the anxiety and depression that frequently manifests in those patients with chronic conditions [79,80]. Patients are usually started on a low dose of 5 to 10 mg amitriptyline taken at night to minimize side effects. This is gradually increased to a daily dose of 50 to 100 mg, depending on the response and tolerability of side effects by the patient. Doxepin can be started at a dose of 25 mg nightly and increased to 75 mg as needed. Patients need to be warned that pain/itch relief is not immediate as it may take several weeks for the drug to become fully effective. Do not underestimate the improvement in quality of life a patient may experience with any degree of symptom relief.

Prevent and Treat Secondary Infection

The final aspect of the general management regimen is prevention or treatment of secondary infection of the irritated or excoriated skin [81]. If the skin is weepy

or crusted, culture the exudate and suspect secondary impetiginization. Cephalexin 500 mg twice daily for 7 to 10 days can be used empirically to treat and prevent secondary bacterial infection. Secondary yeast infections are common during treatment, especially in prepubertal girls and woman with estrogen deficiency. Treat concomitant candidiasis orally to avoid contact dermatitis and maceration of this already moist skin area that might arise from the use of antifungal imidazole creams. Candidiasis is not merely a primary problem but also occurs as a super-infection when topical corticosteroids and oral antibiotics are used to treat underlying conditions [4]. Prescribing fluconazole 100 to 150 mg once to twice weekly during the first few weeks of treatment can prevent this. Especially during acute flares, consider concomitant herpes simplex virus infection and treat with oral antivirals accordingly.

Stress and sexual dysfunction are common in patients who suffer from chronic vulvar itching or irritation. Depression and anxiety may be side effects of the chronic symptom or may be triggers that perpetuate the symptoms. To provide comprehensive care for the patient, the associated stress, sexual dysfunction, and depression must be addressed, including as appropriate acknowledgment of her discomfort, committing to work with her until some amount of relief is found, prescribing antidepressants, including partners in discussions, and referral for professional counseling. Scarring, and dyspareunia caused by scarring, is not expected to resolve with treatment of the primary condition. However, the symptoms can be treated with lubricants, dilators, and surgical repair as warranted. Further, the relevance of other treatable generalized conditions, such as incontinence, diabetes, immunosuppression, and obesity, which may contribute to symptomatology, must be addressed in the treatment plan.

Vulvar dermatoses are often multifactorial. The elements should be unraveled until the diagnoses are as defined as possible and the symptoms resolved or well controlled. Schedule follow-up appointments to re-evaluate for interval changes and effectiveness of treatment. Be willing to biopsy and re-biopsy if symptoms progress or fail to improve. Note that a nonspecific biopsy result does not rule out a diagnosis. While there is currently limited evidence in the literature as to the optimal treatment and follow-up regimens for vulvar dermatoses, chronic conditions—even in the absence of symptoms—require monitoring for complications of disease progression as well as complications from treatment.

CONCLUSION

Vulvar pruritus and irritation associated with vulvar dermatoses are common and distressful symptoms. These nonspecific symptoms can be caused by multiple etiologies and are often multifactorial. The cutaneous changes of vulvar dermatoses require experience to appreciate and easily can be missed or misdiagnosed. Clinical appearance alone cannot consistently be used to distinguish between the numerous skin disorders that affect the vulva. The clinical approach to patients presenting with vulvar skin dermatoses requires a detailed history and physical exam as well as laboratory studies, often including biopsy, in order to identify the cause—or at least narrow the broad differential diagnosis—and allow initiation of directed treatment. Treatment principles include restoring the skin barrier, reducing inflammation, symptomatic relief, and preventing and treating secondary infection. Patients with chronic vulvar dermatoses require long-term treatment and follow up to prevent complications associated with the disease process and treatment.

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REFERENCES

1. Pathak D, Agrawal S, Dhali TK. Prevalences of and risk factors for the vulvar diseases in Nepal: a hospital based study. Int J Dermatol 2011;50:161–7.
2. Hansen A, Carr K, Jensen JT. Characteristics and initial diagnosis of women presenting to a referral center for vulvovaginal disease in 1996-2000. J Reprod Med 2002;47:854–60.
3. Harlow BL, Wise LA, Stewart EG. Prevalence and predictors of chronic lower genital tract discomfort. Am J Obstet Gynecol 2001;185:545–50.
4. Ponte M, Klemperer E, Sahay A, Chren M. Effects of vulvodynia on quality of life. J Am Acad Dermatol 2009;60:70–6.

5. Edwards L. Vulvovaginal dermatology. Preface. Dermatol Clin 2010;28(4):xi–xii.
6. Margesson LJ. Vulvar disease pearls. Dermatol Clin 2006;24:145–55.
7. Schlosser B, Mirowski G. Approach to the patient with vulvovaginal complaints. Dermatol Ther 2010;23:438–48.
8. Edwards L. Evaluation of vulvovaginal disease. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002:361–7.
9. Haefner H. Vulvar anatomy. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002: 123–31.
10. Margesson LJ. Normal anatomy of the vulva. In: Fisher BK, Margesson LJ, editors. Genital skin disorders and treatment. St. Louis: Mosby; 1998:99–107.
11. Mirowski GW, Edwards L. Genital anatomy. In: Edwards L, editor. Genital dermatology atlas. Philadelphia: Lippincott Williams & Wilkins; 2004:1–8.
12. Turner MLC, Marinoff SC. General principles of diagnosis and treatment of vulvar disease. Dermatol Clin 1992;10:275–81.
13. Farage MA, Miller KW, Ledger WJ. Determining the cause of vulvovaginal symptoms. Obstet Gynecol Surv 2008;63:445–64.
14. Lynch PJ, Edwards L. Red plaques with eczematous features. In: Lynch PJ, Edwards L, editors. Genital dermatology. New York: WB Saunders;1994:27–34.
15. Beecker J. Therapeutic principles in vulvovaginal dermatology. Dermatol Clin 2010;28:639–48.
16. Edwards L. Vaginitis. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002:302–16.
17. ACOG Practice Bulletin. No. 93. Diagnosis and management of vulvar skin disorders. Obstet Gynecol 2008;111:1243–53.
18. Selim M, Hoang M. A histologic review of vulvar inflammatory dermatoses and intraepithelial neoplasm. Dermatol Clin 2010;28:649–67.
19. Danby C, Margesson L. Approach to the diagnosis and treatment of vulvar pain. Dermatol Ther 2010;23:485–504.
20. Goldstein AT, Christopher K, Burrows LJ. Plasma cell vulvitis: a rare cause of intractable vulvar pruritus. Arch Dermatol 2005;141:789–90.
21. Wang L, Yang XC, Hao F, et al. Papular acantholytic dyskeratosis of the vulva. Eur J Dermatol 2009;19:402–3.
22. Edwards L. Dermatologic causes of vaginitis: a clinical review. Dermatol Clin 2010;28:727–36.
23. Stika C. Atrophic vaginitis. Dermatol Ther 2010;23:514–22.
24. Forcier M, Musacchio N. An overview of human papilloma virus infection for the dermatologist: disease, diagnosis, management, and prevention. Dermatol Ther 2010;23:458–76.
25. Day S. Threadworm: an infrequent clinical finding in a genitourinary medicine clinic attendee presenting with anogenital irritation. Int J STD AIDS 2009;20:362–3.
26. Lai K, Mercurio M. Medical and surgical approaches to vulvar intraepithelial neoplasia. Dermatol Ther 2010;23:477–84.
27. Pascual JC, Perez-Ramos M, Devesa JP, et al. Extramammary Paget's disease of the groin with underlying carcinoma and fatal outcome. Clin Exp Dermatol 2008;33:595–8.
28. Lam C, Funaro D. Extramammary Paget's disease: summary of current knowledge. Dermatol Clin 2010;28:807–26.
29. Kavala M, Can B, Zindanci I, et al. Vulvar pruritus caused by syringoma of the vulva. Int J Dermatol 2008;47:831–2.
30. Scurry J, van der Putte SC, Pyman J, et al. Mammary-like gland adenoma of the vulva: review of 46 cases. Pathology 2009;41:372–8.
31. Elas D, Benda JA, Galask RP. Langerhans' cell histiocytosis of the vulva: the Iowa experience. J Reprod Med 2008;53:417–9.
32. Saini R, Sarnoff DS. Basal cell carcinoma of the vulva presenting as unilateral pruritus. J Drugs Dermatol 2008;7:288–90.
33. Turner M. Vulvar manifestations of systemic disease. Dermatol Clin 1992;10:445–58.
34. Pipkin C. Erosive disorders of the vulva. Dermatol Clin 2010;28:737–52.
35. Groysman V. Vulvodynia: new concepts and review of the literature. Dermatol Clin 2010;28:681–96.
36. Sherertz EF. Clinical pearl: Symptomatic dermatographism as a cause of genital pruritus. J Am Acad Dermatol 1994;31:1040–1.
37. Lynch PJ, Moyal-Barracco M, Bogliatto F, et al. 2006 ISSVD classification of vulvar dermatoses: pathologic subsets and their clinical correlates. J Reprod Med 2007;52:3–9.
38. Bandow G. Diagnosis and management of vulvar ulcers. Dermatol Clin 2010:28:753–64.
39. Hupper J. Lipschutz ulcers: evaluation and management of acute genital ulcers in women. Dermatol Ther 2010;23: 533–40.
40. Pincus SJ. Vulvar dermatoses and pruritus vulvae. Dermatol Clin 1992;10:297–308.
41. Stewart KM. Clinical care of vulvar pruritus, with emphasis on one common cause, lichen simplex chronicus. Dermatol Clin 2010;28:669–80.
42. Lynch PJ. Vulvar pruritus and lichen simplex chronicus. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002:157–66.
43. Lynch PJ. Lichen simplex chronicus (atopic/neurodermatitis) for the anogenital region. Dermatol Ther 2004;17(1):8–19.
44. Virgili A, Bacilleri S, Corazza M. Managing vulvar lichen simplex chronicus. J Reprod Med 2001; 46:343–6.
45. Schlosser B. Contact dermatitis of the vulva. Dermatol Clin 2010;28:697–706.
46. Margesson L. Contact dermatitis of the vulva. Dermatol Ther 2004;17:20–7.
47. Bauer A, Rodiger C, Grief C, et al. Vulvar dermatoses—irritant and allergic contact dermatitis of the vulva. Dermatology 2005;210:143–9.
48. Utaş S, Ferahbaş A, Yildiz S. Patients with vulval pruritus: patch test reports. Contact Dermatitis 2008;58:296–8.
49. Haverhoek E, Reid C, Gordon L, et al. Prospective study of patch testing in patients with vulval pruritus. Australas J Dermatol 2008;49:80–5.
50. Lucke TW, Fleming CJ, McHenry P, et al. Patch testing in

vulval dermatoses: how relevant is nickel? Contact Dermatitis 1998;38:111-2.
51. Farage MA, Miller KW, Berardesca E, et al. Incontinence in the aged: contact dermatitis and other cutaneous consequences. Contact Dermatitis 2007;57:211–7.
52. Murphy R. Lichen sclerosus. Dermatol Clin 2010:28:707–16.
53. McPherson T, et al. Vulval lichen sclerosus and lichen planus. Dermatol Ther 2010;23:523–32.
54. Ball SB, Wojnarowski F. Vulvar dermatoses: lichen sclerosus, lichen planus, and vulval dermatitis/lichen simplex chronicus. Semin Cutan Med Surg 1998;17:182–8.
55. Edwards L. Lichen sclerosus. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002: 133–45.
56. Neill SM, Tatnall FM, Cox NH. Guidelines for the management of lichen sclerosus. Br J Dermatol 2002;147:640–9.
57. Powell J, Wojnarowska F. Childhood vulvar lichen sclerosus: an increasingly common problem. J Am Acad Dermatol 2001;44:803–6.
58. Cooper SM, Ali I, Baldo M, Wojnarowska F. The association of lichen sclerosus and erosive lichen planus of the vulva with autoimmune disease: a case-controlled study. Arch Dermatol 2008;144:1432–5.
59. Carli P, Cataneo A, DeMagnis, et al. Squamous cell carcinoma arising in lichen sclerosus: a longitudinal cohort study. Eur J Cancer Prev 1995;4:491–5.
60. Renaud-Vilmer C, Cavelier-Balloy B, Porcher R, et al. Vulvar lichen sclerosus: effect of long-term application of a potent steroid on the course of disease. Arch Dermatol 2004;140:709–12.
61. Mirowski G, Goddard A. Treatment of vulvovaginal lichen planus. Dermatol Clin 2010;28:717–26.
62. Cooper SM, Haefner HK, Abrahams-Gessel S, Margesson LJ. Vulvovaginal lichen planus treatment: a survey of current practices. Arch Dermatol 2008;144:1520–1.
63. Zamirska A, Reich A, Berny-Moreno J, et al. Vulvar pruritus and burning sensation in woman with psoriasis. Acta Derm Venereol 2008;88:132–5.
64. Neri I, Patrizi A, Marzaduri S, et al. Vulvitis plasmacellularis: two new cases. Genitourin Med 1995;71:311–13.
65. Browne F, Keane H, Walsh M, Maw R. Papular acantholytic dyskeratosis presenting as genital warts. Int J STD AIDS 2007;18:867–8.
66. Margesson L. Overview of treatment of vulvovaginal disease. Skin Therapy Lett 2011;16:5–7.
67. Weichert G. An approach to the treatment of anogenital pruritus. Dermatol Ther 2004;17:129–33.
68. Lagro-Janssen AL, Sluis S. Effectiveness of treating nonspecific pruritus vulvae with topical steroids: a randomized controlled trial. Eur J Gen Pract 2009;15:29–33.
69. Lynch PJ. Vulvar dermatoses: the eczematous diseases. In: Black M, editor. Obstetrical and gynecologic dermatology. St. Louis: Mosby; 2002:181–94.
70. Rietschel RL, Fowler JF. Allergy to preservatives and vehicles in cosmetics and toiletries. In: Rietschel RL, Fowler JF, editors. Fisher's contact dermatitis. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2001:211–59.
71. Guin JD, Kincannon J, Church FL. Baby-wipe dermatitis preservative-induced hand eczema in parents and persons using moist toilettes. Am J Contact Dermat 2001;12:189–92.
72. Minet A, Eggers S, Wilcox D, et al. Allergic contact dermatitis from Kanthon CG in moist toilet paper. Contact Dermatitis 1989;21:107–8.
73. Guimaraens D, Conde Salazar L, Gonzalez MA. Allergic contact dermatitis on the hands from cholormethylisothiazolinone in moist toilet paper. Contact Dermatitis 1996;35:254.
74. Schollnast K, Kranke B, Aberer W. Anal and palmar contact dermatitis caused by iodopropynyl butycarbamate in moist sanitary wipes. Hautarzt 2003;54:970–4.
75. Fields K, Nelson D, Power T. Contact dermatitis caused by baby wipes. J Am Acad Dermatol 2006;54:S230–2.
76. Weisshaar E. Successful treatment of genital pruritus using topical immunomodulators as a single therapy in multimorbid patients. Acta Derm Venereol 2008;88:195–6.
77. Sarifakioglu E, Gumus II. Efficacy of topical pimecrolimus in the treatment of chronic vulvar pruritus: a prospective case series –a non-controlled, open labeled study. J Dermatolog Treat 2006;17:276–8.
78. Goldstein AT, Parneix-Spake A, McCormick CL, Burrows LJ. Pimecroliums cream 1% for treatment of vulvar lichen simplex chronicus: an open-label, preliminary trial. Gynecol Obstet Invest 2007;64:180–6.
79. Aoki T, Kushimoto H, Hishikawa Y, Savin JA. Nocturnal scratching and its relationship to the disturbed sleep of itchy subjects. Clin Exper Dermatol 1991;16:268–72.
80. Bhatia MS, Gautam RK, Bedi GK. Psychiatric profile of patients with neurodermatitis. J Indian Med Assoc 1996;94:445-6.
81. Abeck D, Mempel M. Staphylococcus aureus colonization in atopic dermatitis and its therapeutic implications. Br J Dermatol 1998;139 Supp 53:13–6.