Acta Dermatovenerol APA

Acta Dermatovenerologica
Alpina, Pannonica et Adriatica

2025;34:123-131
doi: 10.15570/actaapa.2025.25

Chronic vulvar itch: diagnostic and therapeutic challenges

Katarina Trčko[1] ✉

[1]*Department of Dermatology, Maribor University Medical Center, Maribor, Slovenia.*

Abstract

Vulvar pruritus refers to itching affecting the skin and mucosal surfaces of the external genital and perineal regions. It is most frequently associated with infections, inflammatory skin disorders, or neoplastic conditions. Due to the distinctive anatomical and physiological features of the anogenital area, clinical manifestations in this region are often subtle or atypical, which can complicate both diagnosis and management. Because vulvar itch can be highly distressing, timely identification and appropriate intervention are crucial for improving patient quality of life. A comprehensive clinical approach is essential when evaluating patients with vulvar pruritus. This includes a detailed medical history, focused physical examination, and relevant diagnostic testing. Management should involve elimination of contributing or exacerbating factors and treatment directed at the underlying cause. This review article discusses the common causes of vulvar pruritus, emphasizing the diagnostic approach and outlining current treatment strategies. The importance of an individualized patient-centered management plan is emphasized.

Keywords: vulvar itch, pruritus, diagnosis, treatment, barrier function

Received: 20 June 2025 | **Returned for modification:** 30 July 2025 | **Accepted:** 31 July 2025

Introduction

Vulvar pruritus is a common symptom that can cause physical discomfort and emotional distress. It is classified as acute or chronic, with the distinction based on a duration of less than or more than 6 weeks, respectively (1). Depending on the underlying cause and the timeliness or effectiveness of treatment, acute vulvovaginal pruritus has the potential to progress to a chronic form (2, 3). Unlike sensations such as burning or stinging, pruritus is typically characterized by an urge to scratch, which can temporarily alleviate discomfort (4). Despite its prevalence, vulvar itch is often overlooked in clinical settings. Although it can significantly interfere with daily life, intimacy, and mental health, data on its overall prevalence and clinical impact are limited (5, 6). Most of the available data come from studies that focus on specific underlying conditions, which makes it difficult to quantify pruritus as a distinct clinical concern (7, 8). In Europe, estimated prevalence rates of chronic vulvar pruritus range from 5% to 10%, although these figures may underestimate the true burden due to social stigma and the sensitive nature of genital symptoms, which often discourages reporting (9). Importantly, vulvar pruritus is a symptom rather than a distinct diagnosis. It can arise from a wide range of causes, including inflammatory dermatoses, infections, neoplastic processes, hormonal changes, and neuropathic disorders (1, 2). Without adequate treatment, persistent scratching can result in secondary skin changes, such as excoriations and lichenification (thickening of the skin), which increase the risk of further irritation or infection (10).

This narrative review provides clinicians with a concise summary of potential etiologies, diagnostic considerations, and therapeutic options, emphasizing the importance of a multidisciplinary and patient-centered approach.

Vulvar physiology

Due to several anatomical and physiological factors, the skin of the vulva is more sensitive than the skin on other parts of the body (11). Located between the urinary and digestive tracts, the vulva serves as a transition point from keratinized skin to non-keratinized mucosal surfaces (12). Its unique location means it is constantly exposed to irritants, such as perspiration, vaginal discharge, urine, and friction from skin-to-skin contact, clothing, and hygiene products (13). These factors can compromise the integrity of the epidermal barrier. In the genital region, skin pH levels are typically higher than in other areas of the body and are comparable to those found in occluded, moisture-prone sites such as the axillae (14). Moreover, vulvar pH varies not only during the menstrual cycle—being more acidic in the estrogen-dominant mid-cycle phase and temporarily elevated during menstruation—but also across different life stages. In prepubertal girls and postmenopausal women, lower estrogen levels are associated with a higher (more alkaline) pH, whereas, during puberty and the reproductive years, increased estrogen supports a more acidic environment that favors mucosal integrity and microbial balance. Elevated vulvar skin pH may activate serine proteases, disrupting the epidermal barrier and contributing to pruritus (15). The vulvar region has higher moisture levels and increased transepidermal water loss, indicating a weakened skin barrier and consequently greater moisture loss (2, 16). All these factors contribute to itchiness by exacerbating abnormal sensory responses in the vulvovaginal region, making it more susceptible to irritant and allergen penetration, and increasing the risk of local inflammation and irritation (17).

The structural and functional health of vulvar tissue is strongly influenced by hormonal status, particularly estrogen levels. Estrogen plays a central role in maintaining vulvar epithelial thickness, an acidic pH, and a healthy microbiota composition (18). Throughout a woman's life, hormonal fluctuations significantly impact the structure and function of the vulvar epithelium, including pH balance and microbial composition. In early childhood, low estrogen levels result in a neutral or alkaline vulvovaginal pH due to the absence of lactobacilli. As estrogen levels rise during puberty, glycogen accumulates in the vulvovaginal epithelium, providing a substrate for lactobacilli. These bacteria

✉ **Corresponding author: katarina.trcko@gmail.com**

ferment glycogen into lactic acid, thereby lowering the vaginal pH and promoting a protective acidic environment. During the menstrual cycle and menopause, estrogen levels fluctuate or decline, leading to an increase in pH (19). This more alkaline environment can enhance protease activity, which can potentially trigger neurogenic inflammation by activating protease-activated receptors on sensory neurons, keratinocytes, and endothelial cells (20). In postmenopausal women, reduced estrogen levels lead to mucosal thinning, reduced elasticity and lubrication, and increased vulnerability to irritants. These changes weaken the skin's protective barrier, making it more susceptible to mechanical injury, irritation, and infection (19).

Classification of pruritus according to etiology

Various classification systems have been developed to categorize pruritus based on its underlying etiology and clinical features (21). Due to the distinct anatomical and physiological properties of vulvar skin, the causes of vulvar pruritus differ from those of other cutaneous regions and often necessitate a tailored diagnostic approach. To facilitate differential diagnosis and guide management, vulvar pruritus can be clinically classified into the following categories: dermatological, systemic, neuropathic, psychogenic, mixed, and of undetermined origin (21–24). This classification system emphasizes the multifactorial nature of vulvar itch and highlights the importance of comprehensive clinical evaluation. An overview of this classification is presented in Table 1.

Common differential diagnoses of vulvar pruritus and its diagnosis

Precise data on the prevalence of specific causes of chronic or recurrent vulvar pruritus are lacking; however, according to the available literature, dermatitis is the most common cause, accounting for more than half of cases (ranging from 54% to 64%). Other notable causes include lichen sclerosus et atrophicus (7%–13%), chronic vulvovaginal candidiasis (approximately 10%), dysesthetic vulvodynia (approximately 9%), and psoriasis (approximately 5%) (25). The differential diagnosis of vulvar pruritus varies significantly across different life stages. In prepubertal girls, common triggers include group A streptococcal infections, poor hygiene, irritant contact dermatitis, atopic dermatitis, psoriasis, and lichen sclerosus. Among women of reproductive age, the condition is often associated with irritant or allergic contact dermatitis, recurrent vulvovaginal candidiasis, lichen simplex chronicus, psoriasis, lichen sclerosus, and sexually transmitted infections (STIs). In postmenopausal women, declining estrogen levels lead to atrophic vulvovaginitis and recurrent lichen sclerosus, both of which are often accompanied by persistent itching. Recognizing the age-specific prevalence of these conditions is critical for achieving timely diagnosis and effective treatment (2, 26).

When evaluating a patient presenting with vulvar pruritus, it is essential to obtain a thorough medical history, including potential risk factors for STIs, exposure to irritants or allergens, personal hygiene practices, and psychosocial stressors. The use of hormonal contraceptives—in particular, progestogen-only pills—should also be carefully considered because they can suppress endogenous estrogen levels, leading to vulvovaginal dryness and mucosal thinning and secondary itching. It is also necessary to assess the patient for specific clinical signs that may inform the diagnostic process. The clinical approach should be tailored according to symptom duration and any accompanying findings. Diagnosis may be complicated by environmental factors unique to the vulvar region, such as increased warmth, humidity, and partial occlusion, which can alter the clinical presentation of common dermatoses and obscure characteristic features such as scaling (3, 9, 13, 27, 28).

In cases of acute pruritus (lasting less than 6 weeks), the primary focus should be on ruling out STI and other infectious causes. Treatment should be based on the results of these tests. If there is no improvement following the initial treatment, or if the test results are negative, clinicians should consider noninfectious causes or dermatoses. In such cases, further investigation into underlying dermatological conditions is indicated (3, 9). For chronic

Table 1 | **Proposed etiological classification of vulvar pruritus, adapted from Ständer et al (21).**

Category	Associated disease
Dermatological	Inflammatory dermatoses: atopic dermatitis, irritant contact dermatitis, allergic contact dermatitis, lichen sclerosus, lichen planus, lichen simplex chronicus, psoriasis, seborrheic dermatitis, plasma cell vulvitis, dermatographism, autoimmune bullous disorders (pemphigoid, pemphigus, linear IgA disease), acantholytic dermatosis (Darier disease, Hailey–Hailey disease, papular acantholytic dyskeratosis), Fox–Fordyce disease, aphthae (idiopathic or secondary to systemic disorders such as Behçet, inflammatory bowel disease, etc.). Infectious dermatoses: fungal (*Candida albicans*, *C. glabrata*, dermatophytes), bacterial (streptococcal and staphylococcal infections, *Escherichia coli*, *Neisseria gonorrhoeae*, *Chlamydia trachomatis*), viral (herpes simplex virus, human papillomavirus, molluscum contagiosum, varicella zoster virus), parasitic infection (*Trichomonas vaginalis*), parasitic infestations: scabies, pediculosis, enterobiasis. Neoplasms: vulvar intraepithelial neoplasia (VIN), Paget's disease, squamous cell carcinoma, melanoma.
Systemic	Diabetes mellitus, hepatic/renal diseases, drugs, estrogen deficiency, HIV infection, chronic hepatitis C virus infection, hyper/hypothyroidism, malignancies (leukemia, Hodkin's disease), hematologic (iron deficiency, polycythemia rubra vera).
Neuropathic	Result of nerve dysfunction or damage: • Small fiber polyneuropathy (SFPN), often secondary to systemic conditions such as diabetes mellitus, vitamin B12 deficiency, amyloidosis, sarcoidosis, drugs (chemotherapy, alcohol use, etc.); • Myelopathy and central nervous system lesions; • Postherpetic itch as manifestation of postherpetic neuralgia; • Degenerative and inflammatory spine diseases (e.g., compression of nerves or nerve roots).
Psychogenic/psychosomatic	Delusional parasitosis, tactile hallucinations, obsessive compulsive disorders, anxiety, depression, somatoform and dissociative disorders.
Mixed	Overlapping and coexistence of several diseases.
Of undetermined origin	No clear underlying cause identified despite thorough evaluation: idiopathic vulvar pruritus (sensitive skin).

pruritus lasting more than 6 weeks, infectious causes are less likely and underlying dermatoses are a more probable cause. Table 2 summarizes the main features of common vulvar dermatoses, including their clinical presentation and treatment approaches.

Figures 1–3 present the typical morphological presentation of lichen sclerosus, highlighting its key diagnostic characteristics. Figure 4 illustrates the typical appearance of vulvar lichen planus.

A biopsy is indicated when a diagnosis remains uncertain despite noninvasive testing and clinical examination. This is the case when neoplasia is suspected, treatment response is poor, or diagnostic uncertainty continues after therapy is completed (29).

Further investigations may be required based on clinical suspicion. Patch testing can confirm contact allergies, and skin prick tests can determine underlying atopy in patients with a history of allergic disease (30). Laboratory testing, including blood glucose and iron studies, as well as relevant immunological tests, can also support the diagnosis of systemic or metabolic conditions that may be contributing to vulvar symptoms (9). If a neuropathic origin is suspected, appropriate imaging studies should be performed (31). In cases in which a psychogenic component is considered, referral for a psychiatric evaluation may be necessary (2, 32).

When evaluating a patient presenting with vulvar pruritus and pain, lichen planus is one of the key differential diagnoses to consider (9, 31). Vulvar pain may occur with or without itching, and so identifying any accompanying symptoms is crucial for guiding further evaluation. If pruritus is absent, attention should be directed toward identifying the underlying cause based on the specific symptoms present. One common cause in postmenopausal women is genitourinary syndrome of menopause, previously known as atrophic vaginitis (32).

Figure 2 | Figure-eight involvement of vulvar lichen sclerosus.

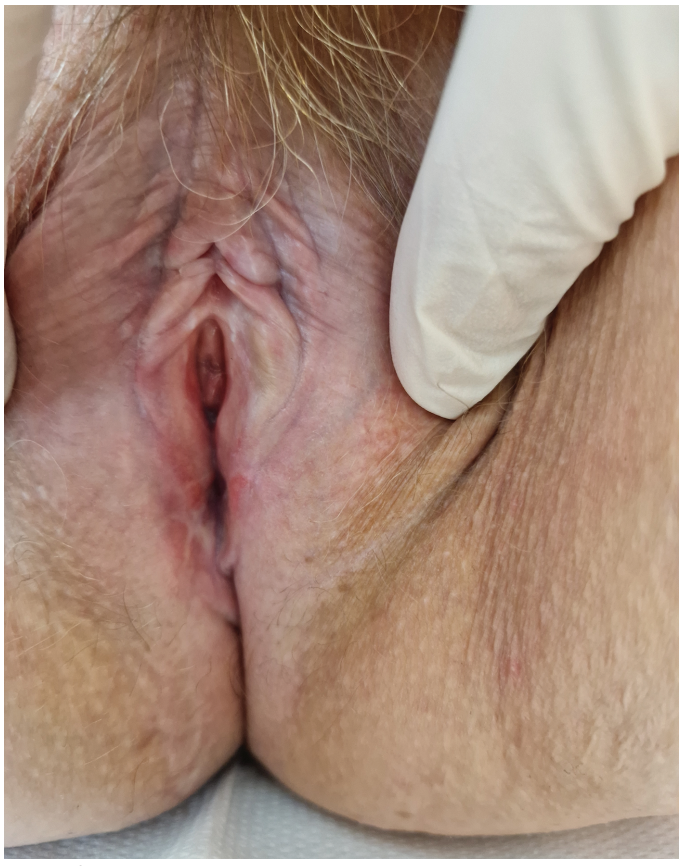

Figure 1 | Lichen sclerosus typically presents as whitish plaques, change in the texture of the mucosa, altered vulvar architecture with the fusion of the labia minora with the interlabial sulci, clitoral phimosis, and erosions.

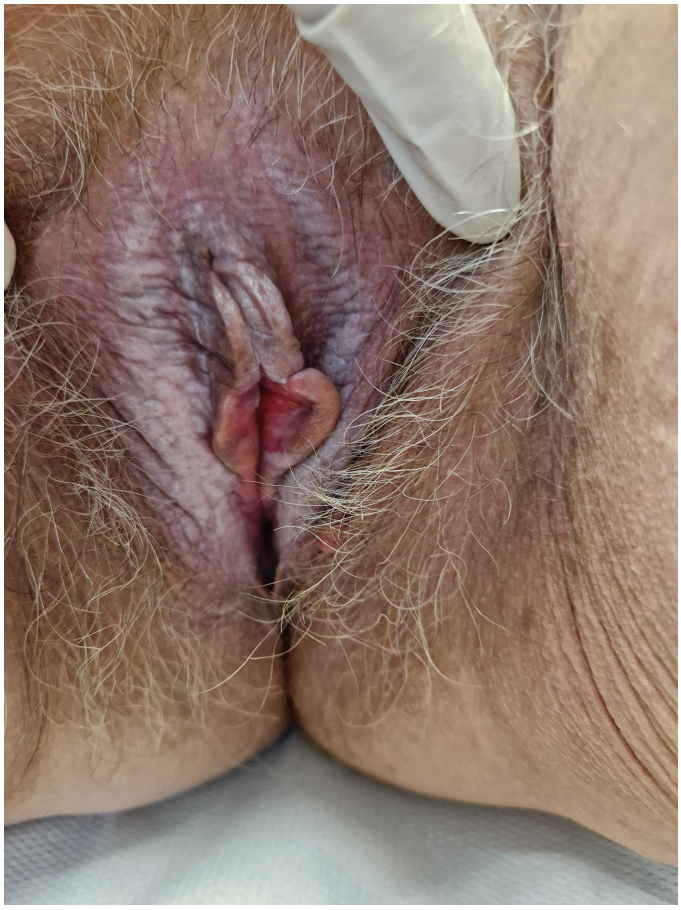

Figure 3 | Hyperkeratotic lichen sclerosus.

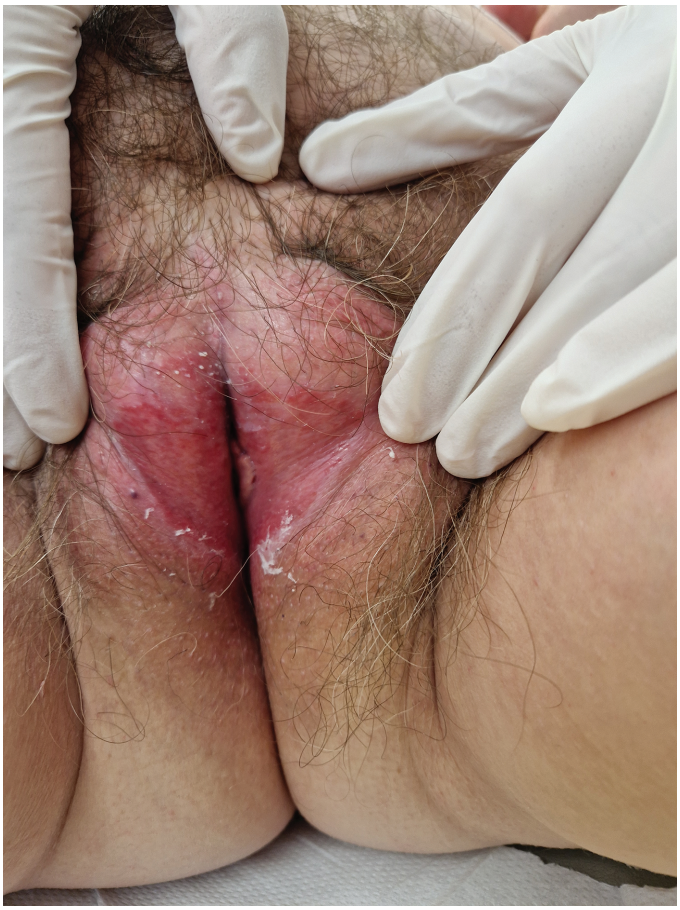

Figure 4 | Lichen planus: sharply demarcated, intensely erythematous erosions accompanied by surrounding white epithelium and significant loss of normal vulvar architecture.

If no itching or menopausal changes are present, clinicians must consider other vulvodynia-related or structural causes (9). Vulvodynia is a chronic pain condition of unknown origin, which is often associated with neuropathic mechanisms. Rather than being considered a distinct disease entity, it is classified as a pain syndrome characterized by ongoing vulvar discomfort, such as burning, pain, stinging, or itching. These symptoms are often triggered by minimal stimuli, and they persist for at least 3 months without a known cause. Proposed etiological factors include peripheral nerve damage or irritation that alters pain signaling pathways from the vulva to the spinal cord, as well as increased density and hypersensitivity of vulvar nociceptive fibers. Elevated concentrations of proinflammatory mediators such as cytokines, abnormal sensory responses to environmental stimuli, possible genetic predisposition, and dysfunction of the pelvic floor musculature manifesting as weakness, hypertonicity, or instability have also been implicated in its pathogenesis (33, 34). In addition, vulvodynia may, in certain instances, have a psychogenic origin involving a different pathophysiological mechanism to that of neuropathic pain (30). When appropriate, an interdisciplinary approach involving dermatologists, gynecologists, pain management specialists, and other relevant specialists is recommended.

Disease progression and associated morbidities

The risk of vulvar squamous cell carcinoma is notably elevated in women diagnosed with lichen sclerosus, with incidence estimates ranging from 1.16 to 13.67 per 1,000 person-years and reported absolute risks reaching up to 21.88%. In comparison, lichen planus is associated with a considerably lower and less clearly established risk, with available data suggesting an absolute risk of approximately 1.16% (35). Due to the increased risk of malignancy, timely diagnosis, appropriate treatment, and regular long-term follow-up are necessary for all patients with these conditions.

Vulvar pruritus can have a significant impact on quality of life. Psychological burdens include anxiety, depression, and disrupted sleep. It can also affect sexual health, often resulting in discomfort during intercourse and reduced sexual satisfaction, which affects intimate relationships (36–38). Women with vulvar symptoms often initially self-treat with over-the-counter (OTC) antifungal products, often due to embarrassment or misinterpreting their symptoms as a fungal infection. However, these products are often ineffective, delaying accurate diagnosis and management, and potentially worsening the underlying vulvar condition. Inappropriate use of OTC agents can irritate the delicate vulvar skin barrier, and it can even exacerbate inflammation and itchiness (3).

Therapeutic principles in vulvar pruritus

Successful management of vulvovaginal pruritus depends on more than just accurate diagnosis and disease-specific therapy; several other factors also play a role. These include educating patients about the chronic nature of the condition, identifying and eliminating irritants, and using well-tolerated topical formulations. Realistic expectations must be set because most vulvovaginal conditions are chronic and treatment usually aims to control symptoms rather than provide a definitive cure.

Basic supportive care involves avoiding known irritants and triggers. These include fragranced hygiene products, cleansers containing emulsifiers, antimicrobial agents such as parabens, certain lubricants, and latex condoms. It is recommended that the vulva be washed once or twice a day with lukewarm water, without soap, cleansers, or detergents (2). Regular application of lipid-rich emollients or petrolatum can help restore moisture and support barrier function. Additional measures, such as wearing loose-fitting clothing, choosing silk or cotton underwear, and avoiding pubic hair removal can further reduce mechanical irritation and support skin recovery (39). Activities such as cycling should be avoided because these may worsen symptoms, and treatment for incontinence should be sought if necessary (40).

Acute symptom flare-ups can be managed with cold compresses, followed by the application of emollients to hydrate and protect the skin. Disruption to sleep due to itching can be managed with sedating antihistamines (e.g., diphenhydramine or hydroxyzine) or tricyclic antidepressants, such as amitriptyline or doxepin (13).

If vulvovaginal symptoms persist despite treatment, clinicians should consider poor adherence, contact dermatitis, resistance to topical corticosteroids, or other factors, such as physical limitations in applying treatment, especially in elderly or obese patients. Clinicians should consider the possibility of misdiagnosis, associated candidiasis, or an emerging systemic disease, and perform a biopsy where appropriate (13).

The most commonly used topical agents according to the identified etiology are outlined in Table 2.

Systemic treatment is generally only considered for severe or refractory cases of vulvar pruritus, particularly when standard topical therapies have been ineffective. Systemic corticosteroids may be indicated for certain inflammatory dermatoses, such as lichen sclerosus or lichen planus, for which more aggressive im-

munosuppressive treatment is required to control disease activity and alleviate pruritus. Short courses of oral corticosteroids, such as prednisolone, can provide rapid symptom relief in such cases, but they must be administered with caution because long-term use can lead to adverse effects. Appropriate tapering and monitoring are required (40, 41).

Immunosuppressive and immunomodulatory agents including methotrexate, azathioprine, cyclosporine, mycophenolate mofetil, and hydroxychloroquine may have potential roles in selected inflammatory vulvar conditions (44, 45). Acitretin has been shown to be effective in treating lichen sclerosus (46). Evidence-based guidelines are essential for optimizing patient outcomes and supporting clinical decision-making.

In the treatment of genital psoriasis, interleukin (IL)-17 and IL-23 inhibitors have demonstrated significant clinical efficacy (47–49). Apremilast, a phosphodiesterase-4 inhibitor, has also shown promise, particularly in reducing pruritus and improving skin clearance (49). Dupilumab, which blocks IL-4 and IL-13 signaling, has shown consistent effectiveness in treating atopic dermatitis, including cases with genital involvement (50). Furthermore, oral Janus kinase (JAK) inhibitors, such as upadacitinib and abrocitinib, both of which have been approved by the Food and Drug Administration for treating moderate-to-severe atopic dermatitis, have been shown to be beneficial in controlling dermatitis and alleviating lichen simplex chronicus and the associated itchiness (51, 52). Most of the systemic therapies currently used to manage vulvar dermatoses are used off-label, which reflects the limited availability of approved treatment options for this condition. This underlines the need for high-quality randomized controlled trials in the future to evaluate the efficacy and safety of these interventions more effectively. Neuropathic or psychogenic vulvar pruritus may benefit from neuromodulating therapies. Anticonvulsants, such as gabapentin and pregabalin, have shown efficacy in treating neuropathic itch (53, 54). Tricyclic antidepressants (e.g., amitriptyline or mirtazapine 10 to 100 mg 2 hours before bedtime) and selective serotonin reuptake inhibitors (e.g., paroxetine) possess antipruritic effects and may be considered in cases linked to central sensitization or mood disorders (13). Duloxetine, a serotonin-norepinephrine reuptake inhibitor used for neuropathic pain and anxiety, may also reduce itch intensity in psychogenic presentations (54).

Opioid antagonists such as naltrexone, targeting endogenous opioid pathways, have demonstrated positive outcomes in small studies (55). Topical lidocaine has been used with some success for localized neuropathic itch (13). In hypoestrogenic states, topical estrogen therapy may alleviate symptoms of dryness, atrophy, and pruritus by improving mucosal integrity (56).

Future perspective

Targeted therapies are emerging as a potential treatment option for refractory vulvar dermatoses, particularly in cases that do not respond to conventional topical or systemic agents. Although these therapies are frequently employed off-label for vulvar disease, mounting clinical experience validates their potential efficacy, necessitating additional research to clarify their position within treatment protocols.

Adalimumab, a tumor necrosis factor alpha inhibitor, has demonstrated efficacy in treating treatment-resistant lichen sclerosus and lichen planus, with reports indicating improvements in inflammation and symptom control (57). Tildrakizumab, an IL-23 inhibitor, showed clinical effectiveness in a case series of 24 patients diagnosed with vulvar lichen planus (58). Oral JAK inhibitors such as tofacitinib have demonstrated therapeutic success in the management of vulvar lichen planus (59).

The efficacy of apremilast in the treatment of erosive lichen planus has been evaluated, and clinical trials are ongoing to study the use of deucravacitinib (a tyrosine kinase 2 inhibitor) and topical ruxolitinib in treating vulvar lichen planus (60–62).

Studies have demonstrated the efficacy of baricitinib and abrocitinib in the treatment of lichen sclerosus (63–65).

Conclusions

Vulvar pruritus is a common, sometimes disabling symptom, with a broad and multifactorial etiology. The diagnostic process can be complex due to the anatomical and neurophysiological sensitivity of the vulvar region, as well as the overlap of symptoms. After infectious, neoplastic, and systemic causes have been addressed or excluded, management should focus on gentle vulvar care, eliminating potential irritants and reinforcing skin barrier protection. As with other chronic pruritic conditions, it is crucial to set realistic expectations early during treatment because symptom resolution is often gradual and may require long-term follow-up. Despite growing interest in vulvar dermatoses, vulvar pruritus remains an under-researched area with much therapeutic uncertainty. Continued investigation is needed to improve diagnostic accuracy and identify more effective targeted antipruritic therapies with favorable safety profiles.

Table 1 | **Common vulvar dermatoses.**

Disease	Clinical manifestations	Signs/symptoms	Additional diagnostic clues	Treatment
Irritant contact dermatitis (ICD)	• Acute:* erythema, edema, vesicles, erosions, usually confined to the contact area; • Chronic:** erythema, dryness, scaling, and fissuring.	• Acute: burning, stinging, pain, and skin soreness; • Chronic: pruritus is often a less prominent symptom than burning and pain.	• Exposure to irritants; • Onset of symptoms within minutes to hours of exposure; • Chronic cumulative ICD develops gradually after repeated exposure to weak irritants;** • May improve quickly after removal of the irritant.	• Avoidance of irritants; • Topical steroid, until lesions regress; • Emollients.
Allergic contact dermatitis	• Acute: erythema, edema, vesicles/blisters; it may extend beyond the initial site of contact, occasionally presenting in a diffuse or spreading pattern; • Chronic: lichenification, scaling, and hyperpigmentation.	• Intense pruritus.	• Typically occurs 24–48 hours after exposure to allergens in sensitized individuals; • Persists or recurs upon re-exposure to the allergen; may become chronic if undiagnosed; • Patch testing is diagnostic and helps identify specific allergens.	• Allergen*** identification and strict avoidance; • Topical steroid until lesions regress; • Emollients.
Atopic dermatitis	• Acute: erythema, edema, vesicles, erosions; • Chronic: lichenified erythematous to hyperpigmented plaques with variable scale.	• Pruritus.	• Early age of onset; • Chronic/relapsing history; • Personal/family history; • Typical age-specific pattern of eczema; • Atopy; • Xerosis cutis; • Elevated IgE.	• Topical steroid; • Topical calcineurin inhibitors; • Emollients; • Antihistamines.
Lichen sclerosus	• Atrophic, white scaly patches/plaques with wrinkled or thinned appearance; • Symmetric distribution; affects labia minora, clitoris, perineal region, vestibule; • "Figure-of-eight" anogenital distribution; • Erythema, ecchymosis occasionally, hyperkeratosis is prominent; • Erosions/fissures/ecchymoses; • Advanced disease: labial resorption, burying of the clitoris, and narrowing of the introitus.	• Irritation, pruritus, burning, painful sexual intercourse, dysuria, constipation in girls; • In 1% asymptomatic.	• In patients of all age groups; • Most common in postmenopausal/ perimenopausal women and prepubertal girls; • Other body parts can be involved; • May have a family or personal history of another autoimmune disease (thyroid disease, alopecia areata).	• High-potency topical steroid ointment once per day; after one to three months less frequently; • Life-long topical steroid maintenance dose in reduced dose (once or twice per week); • Emollients; • Second-line therapy: calcineurin inhibitors (off-label); • Continued follow-up is recommended.
Lichen planus	• Intense erythema involving the introitus and vagina, whitish striae, and whitish epithelium; • Well-defined and intensely erythematous erosions; • Scarring and adhesions may develop, leading to clitoral burial and potential narrowing of the introitus, with possible extension to the vaginal mucosa.	• Early form: pain, severe pruritus, burning; • Late form: dyspareunia, postcoital bleeding, burning; • Rarely asymptomatic.	• Oral mucosa: reticulated white patches on buccal mucosa are common; • Other mucous membranes (e.g., vaginal, anal, esophageal) may also be affected; • Vaginal involvement must be assessed using a speculum, wet mount, or biopsy; • Nail dystrophy or scarring in chronic forms; • Cutaneous lesions: flat, polygonal, purple papules (less common in vulvar-only disease); • Biopsy.	• High-potency topical steroid ointment once per day, after one to three months less frequently; • Life-long topical steroid maintenance dose in reduced dose (once or twice per week); • Emollients; • Second-line therapy: calcineurin inhibitors; • Systemic therapy in therapy-resistant cases;**** • Continued follow-up is recommended.

Table 1 | **Continued.**

Disease	Clinical manifestations	Signs/symptoms	Additional diagnostic clues	Treatment
Lichen simplex chronicus (LSC)	• Thickened and lichenified plaques with excoriations, erosions, hyperpigmentation, or whitish hue due to the presence of scale.	• Pruritus; • Rubbing or scratching results in intense pleasure.	• Primary LSC: in individuals with an atopic predisposition; • Secondary LSC develops in the context of underlying itchy vulvar pathology; • Pruritus is frequently exacerbated by heat, perspiration, physical activity, mechanical friction, and psychological stress.	• Potent topical corticosteroids; • Nighttime antihistamines; • Emollients.
Psoriasis	• Well-demarcated erythematous plaques with/without scaling on the labia majora, may extend to the inguinal folds; • The labia minora are usually spared.	• Pruritus; • Burning, irritation.	• Usually accompanied by lesions in other areas (elbows, knees, scalp, nails); • Family and personal history is often present; • May be the only manifestation in inverse or genital psoriasis.	• Topical corticosteroids (low- to mid-potency); • Calcineurin inhibitors (off-label); • Vitamin D analogs (calcipotriol); • Systemic therapy (moderate to severe or refractory cases): methotrexate, cyclosporine, acitretin, biologics.
Plasma cell vulvitis	• Well-demarcated, erythematous to orange-red macules/patches, involving the introitus, labia minora, or periurethral area; • Lesions may exhibit a glazed, shiny surface with punctate petechiae or telangiectasias; • Typically nonulcerative, even though erosions may occur.	• Pruritus, burning, soreness, dyspareunia.	• Rare dermatosis; • Fifth to eighth decades.	• Topical steroids; • Topical calcineurin inhibitors (off-label); • Imiquimod (off-label); • Surgical excision; • Cryotherapy; • Carbon dioxide laser ablation.
Vulvar intraepithelial neoplasia (VIN)	• White, red, brown, or skin-colored papules, plaques, macules, nodules, or thickened areas; • Solitary or multifocal; • Areas turn white after application of 5% acetic acid (helps in lesion detection); • Erosions more common in differentiated VIN •	• Pruritus (most frequent); • Burning, pain, or tenderness; • Dyspareunia; • Irritation or discomfort; • Some cases are asymptomatic, discovered incidentally during routine examination.	• Usual-type VIN (uVIN): typically multifocal; associated with human papillomavirus (HPV) infection; often occurs in younger women; lesions may be warty or basaloid; • Differentiated VIN (dVIN): usually unifocal; occurs in older women, often in the background of lichen sclerosus; lesions are often subtle, erythematous, or eroded plaques; higher risk of progression to invasive carcinoma; • Biopsy is essential to confirm diagnosis.	• Usual-type VIN (HPV-related): imiquimod; laser ablation; surgical excision; • Differentiated VIN (HPV-unrelated): surgical excision is mandatory due to high malignant potential.
Extramammary Paget's disease	• Sharply defined erythematous plaques with whitish scaling and a moist or exudative surface; • The labia majora are most frequently involved, with possible extension to adjacent areas, such as the perineum, perianal region, or inner thighs.	• Persistent pruritus; • Burning, pain; • Potentially asymptomatic.	• Postmenopausal women; • Slow growing, often misdiagnosed as chronic eczema; • Biopsy is essential to confirm diagnosis.	• Extensive workup to rule out underlying adenocarcinoma; • Wide local excision or Mohs micrographic surgery; • Radiation, laser therapy, and topical therapies (imiquimod); • Follow-up.

Table 1 | **Continued.**

Disease	Clinical manifestations	Signs/symptoms	Additional diagnostic clues	Treatment
Candidiasis	• Vulvar edema and erythema; • Vaginal discharge may be absent or present as thick, white, and clumpy with minimal odor—but in some cases it can appear thin, watery, and nonspecific.	• Pruritus (most frequent); • Burning, soreness, irritation; • Dysuria, dyspareunia; • Infections with *Candida glabrata* or other non-albicans species usually resent with mild or minimal signs.	• Symptoms frequently worsening before menstruation; • Predisposing factors: diabetes mellitus; treatment with sodium glucose cotransporter 2 inhibitors; use of broad-spectrum antibiotics; increased estrogen levels (e.g., during pregnancy or postmenopausal estrogen therapy); immunosuppression.	• Acute: oral fluconazole 150 mg orally given once or twice (72 hours after the first dose) or topical clotrimazole preparations; • Chronic: oral fluconazole 150 mg for two to three sequential doses, 72 hours apart, followed by maintenance fluconazole 150 mg orally once a week for at least 6 months; • *C. glabrata*: intravaginal boric acid 600 mg daily for 14 days; • *C. krusei*: intravaginal clotrimazole, miconazole for 7 to 14 days; • All other non-albicans *Candida*: conventional dose fluconazole; • Pregnancy: topical clotrimazole or miconazole for 7 days.

*Strong irritants: solvents, drugs (5-fluorouracil, podophyllotoxin, trichloroacetic acid).
**Cumulative irritants: sweat, urine, feces, semen, vaginal secretions, diapers, perfumes, washcloths, sponges, deodorants, detergents, soaps, cleansers, powders, douches, perfumes, bubble baths, bath oils or salts, depilatory creams, adult or baby wipes, topical antibacterial and antifungal medications, over-the-counter creams, condoms, spermicides, diaphragms, lubricants.
***Common contact allergens: topical anesthetics, (e.g., benzocaine), fragrances (e.g., fragrance mix-I and II, Balsam of Peru, cinnamic alcohol), preservatives (in creams, prescription creams, hygiene products), topical medications (corticosteroids, antibiotics, antimycotics, antiseptics), clothing with azo dyes.
****Methotrexate, mycophenolate mofetil, systemic corticosteroids, hydroxychloroquine, acitretin, minocycline, cyclosporine.

References

1. Ständer S, Weisshaar E, Mettang T, Szepietowski JC, Carstens E, Ikoma A, et al. Clinical classification of itch: a position paper of the International Forum for the Study of Itch. Acta Derm Venereol. 2007;87:291–4.
2. Raef HS, Elmariah SB. Vulvar pruritus: a review of clinical associations, pathophysiology and therapeutic management. Front Med (Lausanne). 2021;8:649402.
3. Mashoudy KD, Tomlinson AF, Kim S, Shivashankar V, Yosipovitch G, Fletcher M. Scratching the surface: a comprehensive guide to understanding and managing vulvovaginal itching. Am J Clin Dermatol. 2025;26:361–78.
4. Savin JA. How should we define itching? J Am Acad Dermatol. 1998;39:268–9.
5. Hickey S, Bell H. Quality of life in the vulvar clinic: a pilot study. J Low Genit Tract Dis. 2010;14:225–9.
6. Haefner HK, Aldrich NZ, Dalton VK, Gagné HM, Marcus SB, Patel DA, et al. The impact of vulvar lichen sclerosus on sexual dysfunction. J Womens Health. 2014;23:765–70.
7. Zamirska A, Reich A, Berny Moreno J, Salomon J, Szepietowski JC. Vulvar pruritus and burning sensation in women with psoriasis. Acta Derm Venereol. 2008;88:132–5.
8. Conte S, Mohamed SD, Cohen Y, Yacovelli A, Starkey S, Johnston L, et al. Clinical presentations and complications of lichen sclerosus: a systematic review. J Dtsch Dermatol Ges. 2025;23:143–9.
9. Woelber L, Prieske K, Mendling W, Schmalfeldt B, Tietz HJ, Jaeger A. Vulvar pruritus—causes, diagnosis and therapeutic approach. Dtsch Arztebl Int. 2020;116:126–33.
10. Weedon D. Weedon's skin pathology. 3rd ed. London: Churchill Livingstone Elsevier; 2010. Section 2, Tissue reaction patterns; p. 72–91.
11. Farage MA. Sensitive skin in the genital area. Front Med (Lausanne). 2019;6:96.
12. Turner ML, Marinoff SC. General principles in the diagnosis and treatment of vulvar diseases. Dermatol Clin. 1992;10:275–81.
13. Stewart KM. Clinical care of vulvar pruritus, with emphasis on one common cause, lichen simplex chronicus. Dermatol Clin. 2010;28:669–80.
14. Burry JS, Coulson HF, Esser I, Marti V, Melling SJ, Rawlings AV, et al. Erroneous gender differences in axillary skin surface/sweat pH. Int J Cosmet Sci. 2001;23:99–107.
15. Ali SM, Yosipovitch G. Skin pH: from basic science to basic skin care. Acta Derm Venereol. 2013;93:261–7.
16. Britz MB, Maibach HI. Human labia majora skin: transepidermal water loss in vivo. Acta Derm Venereol Suppl (Stockh). 1979;59:23–5.
17. Britz MB, Maibach HI. Human cutaneous vulvar reactivity to irritants. Contact Dermat. 1979;5:375–7.
18. Farage M, Maibach H. Lifetime changes in the vulva and vagina. Arch Gynecol Obstet. 2006;273:195–202.
19. Hammerschlag MR, Alpert S, Rosner I, Thurston P, Semine D, McComb D, et al. Microbiology of the vagina in children: normal and potentially pathogenic organisms. Pediatrics. 1978;62:57–62.
20. Lee SE, Jeong SK, Lee SH. Protease and protease-activated receptor-2 signaling in the pathogenesis of atopic dermatitis. Yonsei Med J. 2010;51:808–22.
21. Bernhard JD. Itch and pruritus: what are they, and how should itches be classified? Dermatol Ther. 2005;18:288–91.
22. Stumpf A, Ständer S. Neuropathic itch: diagnosis and management. Dermatol Ther. 2013;26:104–9.
23. Cohen AD, Vander T, Medvendovsky E, Biton A, Naimer S, Shalev R, et al. Neuropathic scrotal pruritus: anogenital pruritus is a symptom of lumbosacral radiculopathy. J Am Acad Dermatol. 2005;52:61–6.
24. Fischer GO. The commonest causes of symptomatic vulvar disease: a dermatologist's perspective. Australas J Dermatol. 1996;37:12–8.
25. Rimoin LP, Kwatra SG, Yosipovitch G. Female-specific pruritus from childhood to postmenopause: clinical features, hormonal factors, and treatment considerations. Dermatol Ther. 2013;26:157–67.
26. Margesson LJ. Vulvar disease pearls. Dermatol Clin. 2006;24:145–55.
27. Tan A, Bieber AK, Stein JA, Pomeranz MK. Diagnosis and management of vulvar cancer: a review. J Am Acad Dermatol. 2019;81:1387–96.
28. Pichardo Geisinger R. Atopic and contact dermatitis of the vulva. Obstet Gynecol Clin North Am. 2017;44:371–8.
29. Pereira MP, Wiegmann H, Agelopoulos K, Ständer S. Neuropathic itch: routes to clinical diagnosis. Front Med (Lausanne). 2021;8:641746.
30. Misery L. Pruriplastic itch—a novel pathogenic concept in chronic pruritus. Front Med (Lausanne). 2021;7:615118.
31. Soper DE, Patterson JW, Hurt WG, Fantl JA, Blaylock WK. Lichen planus of the vulva. Obstet Gynecol. 1988;72:74–6.
32. Cucinella L, Tiranini L, Cassani C, Martini E, Cumetti A, Memoli S, et al. Insights into the vulvar component of the genitourinary syndrome of menopause (GSM). Maturitas. 2024;186:108006.
33. Vieira Baptista P, Lima Silva J, Pérez López FR, Preti M, Bornstein J. Vulvodynia: a disease commonly hidden in plain sight. Case Rep Womens Health. 2018;20:e00079.

34. Bornstein J, Goldstein AT, Stockdale CK, Bergeron S, Pukall C, Zolnoun D, et al. 2015 ISSVD, ISSWSH, and IPPS consensus terminology and classification of persistent vulvar pain and vulvodynia. J Sex Med. 2016;13:607–12.
35. Leis M, Singh A, Li C, Ahluwalia R, Fleming P, Lynde CW. Risk of vulvar squamous cell carcinoma in lichen sclerosus and lichen planus: a systematic review. J Obstet Gynaecol Can. 2022;44:182–92.
36. Van de Nieuwenhof HP, Meeuwis KA, Nieboer TE, Vergeer MC, Massuger LF, De Hullu JA. The effect of vulvar lichen sclerosus on quality of life and sexual functioning. J Psychosom Obstet Gynaecol. 2010;31:279–84.
37. Ranum A, Pearson DR. The impact of genital lichen sclerosus and lichen planus on quality of life: a review. Int J Womens Dermatol. 2022;8:e042.
38. Messele F, Hinchee-Rodriguez K, Kraus CN. Vulvar dermatoses and depression: a systematic review of vulvar lichen sclerosus, lichen planus, and lichen simplex chronicus. JAAD Int. 2024;15:15–20.
39. Beecker J. Therapeutic principles in vulvovaginal dermatology. Dermatol Clin. 2010;28:639–48.
40. van der Meijden WI, Boffa MJ, Ter Harmsel WA, Kirtschig G, Lewis FM, Moyal-Barracco M, et al. 2016 European guideline for the management of vulval conditions. J Eur Acad Dermatol Venereol. 2017;31:925–41.
41. Foster DC. Vulvar disease. Obstet Gynecol. 2002;100:145–63.
42. Savas JA, Pichardo RO. Female genital itch. Dermatol Clin. 2018;36:225–43.
43. Hargis A, Ngo M, Kraus CN, Mauskar M. Systemic therapy for lichen sclerosus: a systematic review. J Low Genit Tract Dis. 2024;28:84–90.
44. Edwards L. Erosive and vesiculobullous diseases. In: Edwards L, Lynch PJ, editors. Genital dermatology atlas. 2nd ed. Philadelphia: Wolters Kluwer; 2011. p. 125.
45. Martin A, Nguyen BT, Kraus CN. Vulvar inflammatory dermatoses: new approaches for diagnosis and treatment. Cutis. 2024;114:37–40.
46. Bousema MT, Romppanen U, Geiger JM, Baudin M, Vähä-Eskeli K, Vartiainen J, et al. Acitretin in the treatment of severe lichen sclerosus et atrophicus of the vulva: a double-blind, placebo-controlled study. J Am Acad Dermatol. 1994;30:225–31.
47. Bonifati C, Lembo S, Richetta AG, Romanelli M, Satolli F, Corazza M, et al. Effectiveness of guselkumab in patients with facial and/or genital psoriasis: interim analysis results at week 12 from the GULLIVER study. J Eur Acad Dermatol Venereol. 2025;39:7–14.
48. Orsini D, Frascione P, Assorgi C, Pacifico A, Sperduti I, Gargiulo L, et al. Risankizumab for the treatment of genital psoriasis: a 1 year, real world experience. J Eur Acad Dermatol Venereol. 2024;38:e256–8.
49. Lai CC, Shao SC, Tsai ETK, Chi CC. Efficacy and safety of biologics and small molecule inhibitors in treating genital psoriasis: a systematic review of randomized controlled trials. Br J Dermatol. 2025;192:357–9.
50. Gosch M, Cash S, Pichardo R. Vulvar pruritus improved with dupilumab. JSM Sexual Med. 2023;7:1104.
51. Simpson EL, Sinclair R, Forman S, Wollenberg A, Aschoff R, Cork M, et al. Efficacy and safety of abrocitinib in adults and adolescents with moderate to severe atopic dermatitis (JADE MONO 1): a multicentre, double blind, randomized, placebo controlled, phase 3 trial. Lancet. 2020;396:255–66.
52. Simpson EL, Papp KA, Blauvelt A, Chu CY, Hong HC, Katoh N, et al. Efficacy and safety of upadacitinib in patients with moderate to severe atopic dermatitis: follow up data from the Measure Up 1 and 2 randomized clinical trials. JAMA Dermatol. 2022;158:404–13.
53. Oaklander AL, Siegel SM. Gabapentin for the treatment of chronic vulvar pain. Pain Res Treat. 2013;2013:1–5.
54. Yosipovitch G, Samuel LS. Neuropathic and psychogenic itch. Dermatol Ther. 2008;21:32–41.
55. Berger TG, Steinhoff M. Pruritus and opioid signaling. Acta Derm Venereol. 2011; 91:469–77.
56. MacBride MB, Rhodes DJ, Shuster LT. Vulvovaginal atrophy. Mayo Clin Proc. 2010;85:87–94.
57. Courtney A, Adamson SR, Veysey E. Adalimumab use in severe recalcitrant vulval lichen sclerosus and vulval lichen planus. J Low Genit Tract Dis. 2025;29:190–4.
58. Kherlopian A, Fischer G. Successful treatment of vulvovaginal lichen planus with tildrakizumab: a case series of 24 patients. Australas J Dermatol. 2022;63:251–5.
59. Mansouri P, Jafari MA, Chalangari R, Roohaninasab M, Goodarzi A. Successful treatment of erosive lichen planus with tofacitinib: a case series and review of the literature. Clin Med Insights Case Rep. 2024;17:11795476241237350.
60. Skullerud KH, Gjersvik P, Pripp AH, Qvigstad E, Helgesen ALO. Apremilast for genital erosive lichen planus in women (the AP-GELP Study): study protocol for a randomised placebo-controlled clinical trial. Trials. 202;22:469.
61. Wijaya M, Fischer G, Saunderson RB. The efficacy and safety of deucravacitinib compared to methotrexate, in patients with vulvar lichen planus who have failed topical therapy with potent corticosteroids: a study protocol for a single-centre double-blinded randomised controlled trial. Trials. 2024;25:181.
62. Brumfiel CM, Patel MH, Severson KJ, Zhang N, Li X, Quillen JK, et al. Ruxolitinib cream in the treatment of cutaneous lichen planus: a prospective, open-label study. J Invest Dermatol. 2022;142:2109–16.e4.
63. Zundell MP, Al-Dehneem R, Song T, Yousif J, Gottlieb AB. Novel clinical applications of topical ruxolitinib: a case series. J Drugs Dermatol. 2024;23:188–90.
64. Bao C, Zhao Y, Luo R, Xu Q, Tong Z, Xiao Z, et al. Application and comparison of dermoscopy and reflectance confocal microscopy in the target treatment of genital lichen sclerosus: a single-arm prospective study. Dermatol Ther (Heidelb). 2023;13:3071–84.
65. Shen CH, Wang TY, Chi CC. Janus kinase inhibitors for the treatment of lichen sclerosus: a systematic review. Br J Clin Pharmacol. 2025;91:1322–9.