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Journal of Skin and Sexually Transmitted Diseases

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Review Article

Vulvar inflammatory disorders: A review

Smitha Prabhu[1], Swathy Krishna[1]

[1]Department of Dermatology and Venereology, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India

***Corresponding author:**
Smitha Prabhu,
Department of Dermatology and Venereology, Kasturba Medical College, Manipal Academy of Higher Education, Manipal, Karnataka, India.

drsmithaprabhu@yahoo.com

Received: 04 February 2021
Accepted: 16 March 2021
EPub Ahead of Print: 30 April 2022
Published: 14 October 2022

DOI
10.25259/JSSTD_11_2021

Quick Response Code:

ABSTRACT

Vulvar inflammatory disorders (VID) are a miscellaneous group of non-infectious conditions of the vulvar area, which can be broadly classified based on etiology and pathology. Here, we discuss a few pertinent VID including vulvar dermatitis, psoriasis, lichen planus, and lichen sclerosus. This review will focus only on common VID, and a few rare conditions with characteristic features such as Crohn's disease, plasma cell vulvitis, acantholytic disorder of genitocrural area, desquamative vulvitis, and atrophic vulvovaginitis. A thorough history taking and relevant investigations including dermoscopy and biopsy are relevant to proper diagnosis and management of VID. Early management of VID is essential since these conditions are prone to interfere with psychosexual functioning of the patient. Potent topical steroids are the first line management in most cases, followed by topical calcineurin inhibitors, emollients, and antihistamines. Vulvar area is resistant to steroid induced atrophy; hence, use of potent topical steroids does not pose a problem. Oral steroids or steroid sparing agents are used in resistant cases.

Keywords: Vulva, Inflammatory, Genital, Psoriasis, Lichen sclerosus

INTRODUCTION

Vulvar inflammatory disorders (VID) are a miscellaneous group of non-infectious conditions affecting the vulvar area that are characterized predominantly by inflammation.

CLASSIFICATION

VID can be broadly classified based on etiology or pathology though there is no universally used single classification. The International Society for the Study of Vulvovaginal Disease (ISSVD) has classified VID based on histological reaction patterns [Table 1].[1]

This review will focus only on common VID, and a few rare conditions with characteristic features. Autoimmune vesiculobullous conditions, drug-induced vulvar dermatoses and many such, which occur as part of generalized disease, are not discussed here.

Commonly encountered VID are vulvar contact dermatitis, vulvar psoriasis, lichen planus, lichen sclerosus (LS), hidradenitis suppurativa, and lichen simplex chronicus (LSC).[2] Vulvar pyoderma gangrenosum, plasma cell vulvitis, and desquamative vaginitis are relatively rare.[3] Vulvar Crohn's disease and acantholytic dermatoses of the vulvocrural area are often misdiagnosed.

VULVAR DERMATITIS

Vulvar area is prone to barrier disruption and dermatitis due to varying factors such as anatomical position, occlusion, frequent contact with bodily secretions, estrogen deficiency, friction, and heat.[4]

Table 1: ISSVD classification of inflammatory vulvar dermatoses.

Lichenoid pattern	Lichen planus, lichen sclerosus
Dermal sclerosis	Lichen sclerosus
Spongiotic pattern	Atopic, irritant, and allergic contact dermatitis
Vesiculobullous pattern	Bullous pemphigoid and its variants, pemphigus vulgaris linear IgA dermatoses bullous drug eruptions
Acanthotic pattern	Psoriasis, lichen simplex chronicus
Acantholytic pattern	Hailey-Hailey disease, Darier's disease, acantholytic dermatoses of the vulvocrural area
Granulomatous pattern	Crohn's disease Melkersson-Rosenthal disease
Vasculopathic pattern	Aphthous ulcers, Behcet's disease, plasma cell vulvitis

ISSVD: The International Society for the Study of Vulvovaginal Disease

Vulvar dermatitis manifests as erythema, epithelial disruption, erosions, and lichenification associated with pruritus. Common endogenous causes include atopic dermatitis, seborrheic dermatitis, and LSC, whereas exogenous dermatitis comprises allergic and irritant contact dermatitis. These two entities may overlap.[5]

Diagnosis is usually clinical; histopathological examination, which reveals prominent spongiosis with lymphohistiocytic infiltrate can differentiate dermatitis from specific causes. Intraepidermal vesiculation is characteristic of acute cases, whereas hyperkeratosis and acanthosis are present in chronic cases.

Atopic dermatitis

Onset is usually in childhood, and family history of atopy may be present. Clinically and histologically, it cannot be distinguished from other eczemas, and occasionally, only xerosis, erythema, scaling, or lichenification is seen.

LSC

Vulva is a common site for LSC and any age-group may be affected. This can occur primarily, especially in atopics, or may be secondary to an initial pruritic vulvar condition.[3] It is extremely pruritic, and itch-scratch cycle plays a major role in perpetuation with scratching, characteristically resulting in intense pleasure, than pain.[3,4] Chronic scratching damages skin barrier, thus making the area susceptible to irritants and infections.

Heat, friction, moisture, menstruation, tight fitting synthetic underwear, and sanitary pads can precipitate or aggravate the condition.

LSC commonly occurs on the hair-bearing surface, but may extend to perianal or mucosal aspect. Striking lichenification, with occasional erythema, edema, erosions, crusting, or oozing is seen. In chronic cases, hair may be scanty or absent.

Histopathology may reveal hyperkeratosis, acanthosis, hypergranulosis, and papillomatosis with variable inflammatory infiltrate. Lamellar thickening of papillary dermis and neural fibrosis is seen.

Contact dermatitis

The fragile and constantly occluded vulvar skin is susceptible to contact dermatitis. Genital hygiene practices, menstrual pads, and hair removal techniques may contribute.

Irritant dermatitis is more common. Irritants disrupt corneocytes and lipid bilayer leading to inflammation that manifests as burning, stinging, and rawness. Common irritants include body secretions such as vaginal discharge, urine, feces, sweat and semen, topical medications, hygiene products, and hair removal creams. Older women with urinary incontinence and women who indulge in excessive genital cleansing are susceptible.[5]

Common allergens include fragrances, topical medicaments, local anesthetics, antiseptics, wet wipes, preservatives, nail polish, sanitary pads, condoms, and nickel.

Erythema, scaling, crusting, vesiculation, and oozing are seen in allergic contact dermatitis, though rawness and excessive weeping are more in favor of irritant dermatitis. Chronicity leads to secondary LSC.

Differentials

In the acute phase, vesicular and erosive conditions such as candidiasis, herpes simplex, fixed eruptions, autoimmune bullous dermatosis, and erosive LP have to be ruled out, whereas chronic contact dermatitis may be confused with seborrhoeic dermatitis, atopic dermatitis, psoriasis, LSC, or Bowen's disease.

Diagnosis

- A thorough and detailed history should be taken to identify the cause
- Patch testing may help in confirming suspected allergic contact dermatitis, though a negative report does not preclude the latter
- Histopathologic confirmation helps in atypical or unresponsive lesions.

Treatment of vulvar dermatitis

Treatment involves reducing the inflammation, breaking the itch-scratch cycle, and repairing the skin barrier. All

known irritants and allergens must be avoided. Topical corticosteroids (TCS) are the first-line management. Start with potent TCS, especially in LSC, apply twice daily for a week, with gradual tapering to once a week over time. Liberal application of emollients helps in barrier repairs and itch control. Sedative antihistamines are required to control itch. Second-line treatment involves topical calineurin inhibitors (tacrolimus 0.03% or 0.1% w/v or pimecrolimus 0.1% w/v). Addressing contributing issues such as stress, tight synthetic underwear, obesity, and excessive cleansing too may help. The patient should be discouraged from excessive genital hygiene measures.

VULVAR PSORIASIS

Up to 5% of women with persistent vulvar symptoms are subsequently diagnosed as vulvar psoriasis.[6,7] In the vulvar area, friction can act as a Koebner phenomenon leading to formation of psoriatic lesions.

Classic plaque-type psoriasis and inverse psoriasis are common, though pustular psoriasis also may occur. The well demarcated, erythematous plaques with silvery white scales of classic psoriasis have a predilection for hair-bearing areas such as mons pubis and labia majora.[8] Inverse psoriasis is seen in intertriginous areas and folds as shiny, erythematous plaques with minimal scaling and at times, as fissuring.[5,6,9] Common symptoms include pruritus and burning (65% cases), pain (45%), and dyspareunia (30%).[10] The presence of typical psoriatic lesions (in other areas) and nail changes provides a clue in up to 30% cases.[10-12] Lichenoid dermatoses, LSC, vulvovaginal candidiasis, or contact dermatitis may be confused with psoriasis.[10]

Vulvar psoriasis reflects the characteristic histopathology of psoriasis. Occasionally superadded dermatitis or infections may necessitate a clinicopathological correlation. Dermoscopy reveals typical, red dot capillaries on an erythematous background.

Treatment: The mainstay is potent TCS, followed by less potent steroids and psoriasis-specific treatments, such as coal tar cream, topical calcipotriol, topical retinoids, and topical tacrolimus.[12] Systemic methotrexate and dapsone are reserved for severe, resistant, or generalized disease.[7,12,13]

VULVOVAGINAL LICHEN PLANUS (VVLP)

VVLP can occur in isolation, or as part of a generalized disease in up to 50% of patients.[14,15] Vulvovaginal-gingival syndrome is seen in up to 65% of patients with generalized disease.[16]

Clinical subtypes include classical papulosquamous, hypertrophic, and erosive types.

Classical papulosquamous variant has papules and plaques with whitish lacy striae on vulvar and periclitoral areas. It is responsive to TCS and may spontaneously resolve.[14]

Hypertrophic variant is extremely itchy and is resistant to treatment.[14]

Erosive VVLP (EVVLP) is the most common form, characteristically affecting the introitus and labia minora in perimenopausal women and may be associated with oral LP.[14]

Pain, burning sensation and irritation (92%) are more predominant than itch (50%).[8] Erosions may be non-specific or glazed/glossy in appearance with reticulated white borders. Complications include vaginal scarring, synechiae, obstruction, bleeding, and copious yellow discharge.[17,18]

In severe cases, strictures with loss of vulvar architecture, vaginal shortening, marked narrowing or agglutination of introitus, and phimosis of the clitoral hood can occur and may often go unnoticed.[19-21] There may be difficulty in micturition, per vaginal examination, and normal intercourse. EVVLP is associated with significant dyspareunia and emotional distress.[22] There is a 2.3% estimated risk of malignant transformation, with sporadic reports of vulvar neoplasia.[23-25]

The Delphi diagnostic criteria for erosive vulvar LP is given in Table 2.[26]

Diagnosis can be confirmed by dermoscopy showing Wickham's striae and histopathology revealing typical lichenoid interface dermatitis.

Hypertrophic LP shows pronounced irregular acanthosis. Erosive LP shows epidermal erosion with underlying inflammation and typical LP changes in the edge of the lesion.[27] It is imperative to include the edge of the lesion in the biopsy.

Differential diagnoses include LS, drug eruption, cicatricial pemphigoid, vaginal intraepithelial neoplasia, and graft versus host disease (conditions that manifest with erosion, sclerotic changes, or whitish discoloration).

Treatment aims at alleviating the suffering and preventing the progression of the disease.

Ultrapotent TCS is the first-line therapy with up to 71% response rate, which may be accordingly tapered once the active lesions resolve.[20]

Table 2: Diagnostic criteria for vulvar erosive lichen planus (Simpson *et al.*).

1	Scarring
2	Hyperkeratotic border or Wickham's striae
3	Other mucosal involvement
4	Well-demarcated erosions at the vaginal introitus
5	Pain/burning symptoms
6	Vaginal inflammation
7	Band-like infiltrate at the dermal-epidermal junction
8	Lymphocyte predominance
9	Evidence of basal degeneration

Any 3 out of the 9 should be present for a definitive diagnosis

Second-line treatment includes topical calcineurin inhibitors.[21,28]

For severe disease, systemic treatment is required.[2,29] Oral prednisolone (0.5 mg/kg body weight) or monthly intralesional infiltration of triamcinolone acetonide (10 mg/ml) is used for rapid control of symptoms. Corticosteroid-sparing agents found useful are azathioprine, cyclophosphamide, cyclosporine, dapsone, griseofulvin, hydroxychloroquine, minocycline combined with nicotinamide, mycophenolate mofetil, methotrexate, systemic retinoids, thalidomide, and biological agents such as adalimumab and etanercept. These are useful in long-term management, though often the results are disappointing, and adverse effects limit their prolonged use.[30,31]

Hydrocortisone cream or suppositories in tapering frequency, and periodic insertion of vaginal dilators smeared with corticosteroid ointment maintains vaginal patency in patients with vaginal strictures.[32] 150 mg fluconazole once a week is used as prophylaxis against candidal superinfection.[33]

Labial fusion, vaginal stenosis, and clitoral phimosis may require surgical intervention after the control of the active disease, for relief of symptoms, and for restoration of urinary and sexual functions.[34,35]

LS

This is a chronic inflammatory autoimmune dermatosis, predominantly affecting the perineum, and occasionally other sites, leading to significant pain, pruritus, and sexual dysfunction. Older terminologies are now obsolete.[36]

The estimated prevalence ranges from 1:300–1000 in vulvar clinics, 1.7% in gynecology setting, and 3% in geriatric facility.[37,38] There is a distinct bimodal peak in age. Prepubertal (5–15%) and postmenopausal women (50–60%) are mostly affected.[4,39] Childhood LS may resolve or can persist into adulthood.[39]

In up to 34% cases, a concurrent autoimmune disease (thyroiditis, pernicious anemia, vitiligo, and alopecia areata) may be present.[40]

Symptoms vary depending on the site, extent, and level of sclerosis.

Clinical features include intolerable, nocturnal vulvar pruritus (90%), burning sensation, pain, dyspareunia, and dysuria. It is asymptomatic in 1% cases. Hypo to depigmented macules and plaques with fragile, "cigarette paper" like textured skin are characteristically seen.[33] Fragility leads to petechiae, erosions, and fissuring. Constant scratching leads to lichenification. Advanced sclerosis and scarring lead to resorption of tissue, synechiae or obliteration of labia and clitoris, loss of labia minora, and stenosis of introitus leading to dyspareunia and sexual dysfunction.[33,39,40]

Typical "figure-of-eight pattern" progression encircling vulvar and perianal areas occurs in up to 60% of cases. In perianal LS, constipation, rectal bleeding, and pain on defecation may occur. Extragenital lesions occur in up to 18% women.[41]

LS is often undiagnosed or misdiagnosed as vulvovaginal atrophy or vitiligo.[4,5,39] Early diagnosis and treatment are essential as malignancy has been reported in 1–5% of chronic, untreated cases with a cumulative incidence of 6.7%.[42] Physical signs of sexual abuse in children, vitiligo, LSC, LP, cicatricial pemphigoid, and psoriasis may mimic LS lesions or vice versa. Vagina and cervix are typically spared in LS, in contrast with vulvar LP.[43]

A biopsy is recommended when the clinical diagnosis is uncertain, in treatment failure or if there is a suspicion of malignancy.[41] The following changes are observed in LS: Basal cell vacuolization, lichenoid infiltrate in the upper dermis, epidermal atrophy, papillary dermal edema and sclerosis, and hyalinization and homogenization of collagen.[39]

Early and adequate TCS treatment, till the color and texture of skin is normalized, significantly lowers the risk of malignant transformation.[41]

Patient counseling is important for treatment compliance and for the prevention of malignancy. Treatment is mandatory even in asymptomatic cases, as the risk of malignancy increases with age and chronicity.

First-line treatment consists of potent or ultrapotent TCS daily for 1 month, followed by alternate day application for a month and thereafter bi-weekly application for another month leading to considerable improvement in 60–95% cases.[18,39] The labia minora and peri-clitoral areas are resistant to steroid induced atrophy and telangiectasia, making prolonged maintenance regimens safe.[44]

Frequent and prolonged application of emollient cream is an important part of maintenance regime.[45] Less potent TCS such as mometasone furoate are also efficacious.[3,40]

Second-line treatment includes topical calcineurin inhibitors. Clobetasol 0.05% is found to be significantly more effective than tacrolimus 0.1% in reducing inflammation.[46] Pimecrolimus is equally efficient in reducing pruritus, burning, or pain.[47]

In recalcitrant disease, intralesional injection of 0.1 mL triamcinolone, repeated monthly for 3 months, not more than 4 doses a year can be tried.[41]

In resistant cases, systemic immunosuppressives (cyclosporine and methotrexate) and oral acitretin (25–50 mg/day) may be used.[4] Surgery is indicated in malignant disease. Cicatrices or strictures and stenosis, when present, require surgical intervention after the resolution of inflammation. Ongoing treatment after surgery prevents recurrence.[39] Adjuvant therapy includes treatment of associated candidiasis or bacterial infection, antipruritic agents, and soothing emollients.

In LS-associated clitoral phimosis, adhesion lysis by lacrimal duct probe resulted in patient satisfaction and sexual function.[48] Vulvectomy is reserved only for malignancy.[40]

Emerging potential therapies include fractional CO_2 laser, platelet-rich plasma treatment, and high-intensity focused ultrasound; however, current evidence does not support any role for intervention in the prevention of squamous cell carcinoma.[49-54]

Treatment of pediatric LS is challenging due to long-term compliance issues. Most therapeutic failures in LS are due to inadequate or intermittent treatment.[4]

Periodic follow-up with biopsy of suspicious lesions is necessary to exclude neoplasia.[33]

PLASMA CELL VULVITIS

Also called as vulvitis circumscripta plasmacellularis or Zoon's vulvitis, this is a distinctive, chronic, idiopathic condition exhibiting well-demarcated shiny, erythematous, orange-red plaques, occasionally with punctate petechiae, in the vestibule and around the meatus, which may extend up to labia minora, causing itching, and burning sensation.[2]

It must be differentiated from erosive vulvar LP and vulvar intraepithelial neoplasia (VIN). Histopathology shows atrophic epidermis with flattened, "lozenge shaped" epithelial cells, and a dermal infiltrate predominantly composed of plasma cells (>20%).

Treatment involves use of potent TCS such as clobetasol propionate 0.05% twice daily, imiquimod 5%, topical cyclosporine, topical tacrolimus 0.1%, and topical pimecrolimus 1%. For resistant lesions, intralesional infiltration with triamcinolone acetonide 10 mg/ml and ablative CO_2 laser may be tried.[55]

DESQUAMATIVE INFLAMMATORY VAGINITIS

It is a lesser known, idiopathic, inflammatory mucositis pertaining to vagina, manifesting as excessive purulent discharge associated with itching, burning sensation, and dyspareunia. Erythema and edema of introitus and labia minora may be seen. It is a diagnosis of exclusion.[2]

Differentials include all causes of vaginitis/cervicitis such as candidiasis, trichomoniasis, bacterial vaginosis, gonococcal, non-gonococcal, and atrophic vulvovaginitis and erosive conditions such as EVVLP, Zoon's vulvitis, and atrophic vulvitis.

Diagnosis: Wet mount shows increased polymorphonuclear lymphocytes and squamous cells with absence of lactobacilli. Vaginal pH is more than 5.

Infectious causes should be ruled out by appropriate tests. In menopausal women, a trial with estrogen cream should be done.

Treatment includes vulvovaginal low potent steroid such as hydrocortisone and clindamycin vaginal pessaries for 1 month, later to be tapered to the least frequency required.[2]

Low dose doxycycline, low dose naltrexone, and even oral immunosuppressives are tried in resistant cases.

PAPULAR ACANTHOLYTIC DERMATOSES OF THE VULVOCRURAL AREA

This is a rare, focal acantholytic disorder. Some consider this as a variant of Hailey-Hailey disease or Darier's disease localized to the genitalia.[56] Painful or pruritic, hypo-pigmented to slightly erythematous, papules, and plaques appear over the vulva which may extend to the perineum and the medial aspect of thighs.[57] Clinical differentials include Hailey-Hailey disease, Darier's disease, warty dyskeratoma, pemphigus vegetans, and condyloma acuminata.

Histopathology reveals hyperkeratosis, focal parakeratosis, full thickness acantholysis, and dyskeratosis visible as "corps ronds" and grains.

Treatment: Unlike Hailey-Hailey and Darier's disease, lesions are very resistant to any form of therapy including topical steroids, tacrolimus, and tretinoin. CO_2 laser, radiofrequency ablation as well as cryotherapy have been tried. Solitary or a few nodules can be surgically excised.

VULVAR CROHN'S DISEASE

Mucocutaneous involvement is seen in approximately 15% cases of Crohn's disease, and skin involvement precedes intestinal disease in one-fourth cases. Contiguous disease from the colon produces perianal fistulas, whereas metastatic Crohn's has varying presentations.

Distinctive clinical features include non-pitting vulvar edema and knife cut ulcers at skin creases. Other features include perianal fistula, violaceous nodules, and firm or verrucoid tags. Chronicity leads to lymphedema, lymphangiectasia, verrucosa nostras, and vulvar ulcers.[58] Young girls typically present with indurated labial edema.[59]

Diagnosis is by correlation with other organ involvement; granulomatous inflammation is characteristic on histopathology.

Elevated fecal calprotectin level (>250 microgram/mg) is 87% specific and 99% sensitive for diagnosis.[60]

Systemic treatment of Crohn's disease will improve the vulvar lesions too.

ATROPHIC VULVOVAGINITIS

The current term is "genitourinary syndrome of menopause" (GSM).[61] It occurs in an estrogen depleted (after natural or surgical menopause, or even during prolonged breast feeding) vulva and vagina. There is atrophy, decreased moisture and elasticity predisposing to barrier dysfunction, erythema, erosions, and mucopurulent discharge. Symptoms include dry, itchy irritated skin with burning sensation, and dyspareunia.

Occasionally urinary symptoms such as increased frequency, dysuria, and recurrent urinary tract infections are seen.[2] Diagnosis is confirmed by microscopy which shows immature parabasal squamous cells with normal or increased polymorphonuclear leukocytes on saline wet mount.

Management: First-line treatment is topical estrogen cream 0.5–4 g at night for 2 weeks, alternate nights for 2 weeks, then maintenance therapy of 1–2 times/week. Adjuvants include moisturizers and lubricants, and short term or intermittent use of low potent steroid such as hydrocortisone 1% cream. Oral ospemifene, an estrogen agonist has been tried, but is not widely used. Counseling the patient (regarding the condition, use of topical estrogen and its side effects and the need for sustained treatment) is of paramount importance. Superadded candidiasis should be treated.[2]

GRANULOMATOUS VULVITIS

This is a lesser known entity also known as hypertrophic vulvitis, chronic edema of the vulva and Melkersson-Rosenthal vulvitis.[39] It is considered to be a counterpart of orofacial granulomatosis, though some consider it to be an early spectrum of Crohn's disease. It may be associated with granulomatous cheilitis. Progressive fibrosis and lymphedema may complicate the course.

Histopathologically, it is characterized by dermal edema, lymphocytic infiltrate, and non-necrotizing epitheloid cell granulomas in the deep dermis. Crohn's disease and sarcoidosis, which also present with vulvar edema and granulomatosis, must be ruled out.[39]

Treatment involves intralesional or systemic steroids for acute and severe cases. Oral metronidazole, clofazimine, hydroxychloroquine, and danazol can be used as maintenance treatment.[39]

DERMATITIS ARTEFACTA

This has to be considered in any vulvar condition which appears atypical and induced. The means of production and appearance of lesions vary considerably, though common methods include application of irritants, chemicals, marking nut and intravaginal insertion of foreign bodies which can lead to erythema, edema, blistering, erosion, and vaginal discharge. A high index of suspicion is needed to make the diagnosis. Symptomatic treatment with psychiatric and clinical psychology consultations forms the crux of the management.

MANAGEMENT OF VULVAR DERMATOSES

All vulvar dermatoses present almost alike with erythema, edema, itching, burning, and dyspareunia. Erosions or ulcers may be primary to the pathology or induced by scratching. Infection induced and neoplastic vulvar dermatoses may mimic inflammatory conditions. Hence, a thorough physical and laboratory examination with a careful eye to any subtle clue should be the norm.

History taking should be thorough and focus on the chronicity, precipitating factors, menstrual history, type and amount of discharge or lesions, and any associated systemic complaints.

A detailed, careful inspection of the vulva including mons, crural folds, and perianal region, and then focusing centripetally should be performed. A general examination and dermatological examination should complement the genital examination.

Other causes for vulvar edema and erosions should be ruled out, a detailed description of which is beyond the realm of this article [Table 3].[39]

Corroborative investigations include wet mount or Gram stain of the vaginal discharge, potassium hydroxide (KOH)

Table 3: Differentials for vulvar edema and erosions.

Vulvar edema		Vulvar erosions/ulcerations	
Infections	Candidiasis, cellulitis, primary herpes genitalis, filariasis, tuberculosis, lymphogranuloma venereum	Vesiculobullous disorders	Pemphigus, bullous pemphigoid, fixed drug eruption, linear IgA disease
Inflammation	Dermatitis, sarcoidosis, vulvar Chron's disease	Infections	Bullous impetigo, crusted impetigo, sexually transmitted infections
Subcutaneous tumors	Lymphangioma, lipoma, Bartholin's cyst	Inflammatory	Behcet's disease, pyoderma gangrenosum, major aphthosis, plasma cell vulvitis
Medical and surgical causes	Following radiotherapy or vulvar surgery, nephrotic syndrome, congestive cardiac failure, hypoalbuminemia	Nutritional	Acrodermatitis enteropathica, iron deficiency, folate deficiency, riboflavin deficiency
Miscellaneous	Pregnancy, vulvar varicosities, sexual trauma, hematoma, angioedema	Malignancies	Vulvar intraepithelial neoplasia, squamous cell carcinoma, malignant melanoma
		Injuries	Cryotherapy, trauma by physical or chemical (podophyllin, 5-fluorouracil, trichloroacetic acid) agents

mount to rule out fungal infections, or even acetowhitening, tests for other sexually transmitted infections and biopsy wherever indicated. Dermoscopy of vulvar area is now widely practiced and can help in the diagnosis of conditions such as psoriasis, lichen planus, and various malignancies.

Occasionally, more than one pathology may co-exist (e.g., LS with LSC or psoriasis with allergic contact dermatitis); hence, it is prudent to keep a discerning eye.

TCS are the treatment of choice in most inflammatory conditions. Topical steroid sparing agents, systemic steroids, and other immunosuppressives may be added in severe, non-responsive cases.

Fungal superinfection should be prevented by prophylactic antifungal therapy whenever prolonged treatment is anticipated.

Associated factors such as malnutrition, anemia, and psychosexual distress should be dealt with. Proper genital hygiene measures should be discussed with the patient. The need for prompt and timely follow-up should be overemphasized.

CONCLUSION

Vulvar inflammatory disorders are more common than encountered, due to reticence of females to seek professional help. A thorough knowledge of the nuances of presentation of various inflammatory conditions, along with judicious investigation and apt management will go a long way in providing physical and psychological comfort to the patient.

Declaration of patient consent

Not required as there are no patients in this article.

Financial support and sponsorship

Nil.

Conflicts of interest

Dr. Smitha Prabhu is on the editorial board of the Journal.

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How to cite this article: Prabhu S, Krishna S. Vulvar inflammatory disorders: A review. J Skin Sex Transm Dis 2022;4:188-95.