Approach to Vulvovaginal Dermatologic Conditions in Primary Care for Pediatric and Adolescents: Diagnostic and Therapeutic Challenges: A Narrative Review

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ABSTRACT

This narrative review synthesizes literature from key databases including PubMed/MEDLINE and Embase, employing a non-systematic methodology to provide a clinical overview for primary care providers. Pediatric and adolescent patients with vulvovaginal dermatologic conditions pose unique diagnostic and management challenges. Young patients may struggle to articulate symptoms, necessitating a sensitive and caring approach to both examination and conversation. Accurate diagnosis relies on a careful history, a thorough physical examination, and, in certain cases, laboratory testing. Cultural awareness and an understanding of developmental stages are essential for establishing a conducive atmosphere for these sensitive discussions. Management is complex, often requiring a multidimensional approach to address individual patient needs. Treatment options range from topical corticosteroids and antifungals to educational interventions regarding hygiene and irritant avoidance. Adherence can be a significant concern, particularly among adolescents. Primary care providers must also address psychological factors, as these conditions can severely impact self-esteem and body image, especially during adolescence. Collaboration with dermatologists or gynecologists is invaluable for complicated cases, highlighting a team-based approach to provide comprehensive care and navigate ongoing management challenges.

Keywords: Vulvovaginal dermatoses; Pediatric and adolescent gynecology; Primary care; Lichen sclerosus; Vulvovaginitis; Diagnosis; Management.

INTRODUCTION

Vulvovaginal dermatologic conditions represent a significant yet underrecognized component of pediatric and adolescent primary care, encompassing a broad spectrum from inflammatory dermatoses to infections[1]. These disorders are a leading cause of referrals to pediatric gynecology, with many cases first presenting to primary care providers (PCPs)[2].

The vulvar region's unique anatomy and physiology in young patients, combined with profound psychosocial factors, create distinct diagnostic and therapeutic hurdles[3]. For the PCP, navigating these challenges is critical, as timely management can alleviate discomfort, prevent long-term sequelae like scarring, and mitigate psychological distress[4].

The prevalence of vulvovaginal symptoms in young females is substantial, though underreporting is common.

Vulvovaginitis is the most frequent gynecologic complaint in prepubertal children[6]. Among specific dermatologic conditions, lichen sclerosus (LS) affects an estimated 1 in 900 to 1 in 1100 prepubertal girls[7], while eczematous dermatitis accounts for a significant portion of chronic vulvar pruritus[8]. This high prevalence necessitates proficiency from PCPs in initial evaluation.

Diagnosis is fraught with complexity. Presentations are often nonspecific, with pruritus, pain, or discharge common to many etiologies[9]. The prepubertal, estrogen-deficient vulva is both susceptible to irritation and challenging to examine[10]. Significant psychosocial

Received: 01/12/2025
Accepted: 31/12/2025

barriers further complicate care; children may be unable to articulate symptoms, while adolescents frequently experience embarrassment, leading to delayed presentation[3]. Many PCPs also report limited confidence in diagnosing vulvar dermatoses[9].

Treatment presents further challenges. The use of topical therapies like corticosteroids requires careful consideration of potency and side effects[7]. Adherence is often poor, particularly for chronic conditions like LS, which demands long-term maintenance to prevent scarring and has high clinic dropout rates[11]. The associated psychological impact—including anxiety and impaired body image—is profound and can hinder engagement[5].

The PCP's role as the first point of contact is therefore critical. They are positioned to build trust, initiate management, and determine referral needs[2], yet they often operate with time constraints and variable comfort in genital examination[6]. A multidisciplinary approach is ideal but not always accessible[8].

This review synthesizes current evidence to provide PCPs with a structured, practical approach to diagnosis and initial management of these common conditions, addressing a gap in comprehensive primary care resources[7],[9].

Anatomical and Physiological Considerations:

The vulva is not a static structure; it undergoes a series of transformative stages influenced by hormonal milieu, which in turn dictate tissue integrity, local immune environment, and microbial ecology[4],[10]. These developmental stages—prepubertal, peripubertal, and postpubertal—each present a unique clinical landscape that directly influences disease presentation, susceptibility, and appropriate therapeutic intervention[11]. For the primary care provider, recognizing these norms is the first critical step in distinguishing pathological findings from normal physiological variants.

The Prepubertal Stage (Infancy to ~8 Years)

The prepubertal vulva is characterized by a state of relative hormonal quiescence following the withdrawal of placentally derived maternal estrogens. Anatomically, the labia majora are flat and underdeveloped, offering minimal protection to the sensitive structures within. The labia minora are thin, delicate, and may appear erythematous, which can be misinterpreted as inflammation by the untrained eye[12]. The hymen is estrogen-deficient, making it exquisitely sensitive; even minor irritation or inflammation can cause significant discomfort. The vaginal vestibule is particularly vulnerable due to this lack of adipose padding and protective hair[13].

Physiologically, this stage is defined by a neutral to alkaline vaginal pH, typically ranging from 6.5 to 7.5[10]. The vaginal mucosa is thin, with a relative paucity of glycogen-rich epithelial cells. Consequently, the primary microbial flora is not the lactobacillus-dominant ecosystem of reproductive-aged women but is instead composed of a mixed aerobic population, including *Staphylococcus epidermidis*, *Streptococcus* species, *Escherichia coli*, and anaerobes[14].

The absence of a protective acidic environment and mature, glycogen-rich epithelium creates a milieu with lower innate defenses against pathogens and irritants. Furthermore, the thin, atrophic mucosa is more susceptible to chemical, mechanical, and microbial injury, explaining the high prevalence of nonspecific vulvovaginitis and irritant contact dermatitis in this age group[6],[13]. Any dermatosis affecting this fragile tissue, such as lichen sclerosus, often appears more erosive or inflamed than it might in a mature, estrogenized vulva[7].

The Peripubertal Transition (~8-14 Years)

This transitional phase, encompassing thelarche and adrenarche, marks the beginning of significant hormonal and anatomical change. The initial changes are often subtle and can precede menarche by several years. Adrenarche leads to the development of pubic hair (pubarche), initially sparse and fine on the labia majora, progressing to coarser, curlier hair[15]. The labia majora begin to accumulate subcutaneous adipose tissue, becoming fuller and providing increased protection. The labia minora may show mild thickening and pigmentation[11].

The most critical physiological shift begins with the onset of ovarian estrogen production. Estrogen stimulation induces proliferation and maturation of the vaginal squamous epithelium, which becomes multilayered and begins to accumulate glycogen[10],[14]. This process alters the local environment in two key ways: first, the thickened epithelium provides a more robust physical barrier against irritants and trauma; second, the cellular glycogen serves as a substrate for bacterial metabolism, initiating a shift in microbial flora. The vaginal pH begins its gradual decline towards acidity. This transition period can be one of clinical ambiguity, as the vulvar tissues may appear mottled with areas of mature and immature epithelium, and symptoms may fluctuate with hormonal cycles. Dermatoses may begin to manifest more classically, yet the incomplete maturation can modify their appearance[11].

The Postpubertal/Adolescent Stage (Menarche and Beyond)

Following menarche, the vulvovaginal anatomy reaches its mature, estrogenized state. The labia majora are full and well-developed, with a full complement of pubic hair. The labia minora are pigmented, cushioned, and well-

defined. The vaginal mucosa is thick, rugated, and moist[16]. Physiologically, the system is now characterized by a consistently acidic vaginal pH, maintained between 3.8 and 4.5. This acidity is the direct result of a lactobacillus-dominant microbiome, where these bacteria convert epithelial glycogen to lactic acid[14]. This ecosystem provides a formidable defense against colonization by pathogenic bacteria and yeast, though it does not confer complete immunity.

The mature environment has direct implications for dermatologic disease. Estrogenization provides relative protection against irritants and some infections; however, it also creates conditions favorable for other pathologies. For example, *Candida* species, which are uncommon pathogens in prepubertal girls, become a frequent cause of vulvovaginitis in adolescents, as they thrive in a glycogen-rich environment[14]. Furthermore, the clinical morphology of chronic dermatoses changes. Lichen sclerosus in an estrogenized vulva may present with more subtle hypopigmentation and wrinkling rather than the intense erythema, purpura, and fissuring commonly seen in prepubertal patients[7]. The presence of mature hair follicles also introduces pathologies like folliculitis and hidradenitis suppurativa, which are not seen in childhood[16]. A summary of these key developmental changes is provided in Table 1.

Table 1: Key Developmental Changes in Vulvovaginal Anatomy and Physiology

Developmental Stage	Hormonal Milieu	Anatomic Features	Vaginal pH	Dominant Microflora	Clinical Implications
Prepubertal	Low estrogen	Thin, delicate mucosa; flat labia majora; minimal fat padding & hair[12,13].	Neutral-Alkaline (6.5-7.5)[10].	Mixed aerobes (*Staph*, *Strep*, *E. coli*)[14].	High susceptibility to irritants & nonspecific vulvovaginitis; dermatoses appear inflamed [6,13].
Peripubertal	Rising estrogen (adrenarche/thelarche)	Labia thicken; pubic hair develops; adipose tissue increases[11,15].	Transitional (declining).	Shift from mixed aerobes to lactobacilli begins[10,14].	Clinical presentation can be variable; onset of cyclical hormonal influences on symptoms[11].
Postpubertal	Cyclic estrogen & progesterone	Mature, rugated mucosa; full labia majora & minora; protective pubic hair[16].	Acidic (3.8-4.5)[14].	Lactobacillus-dominant[14].	Increased risk of candidiasis; chronic dermatoses may manifest with altered morphology[7,14,16].

This developmental framework is essential for contextualizing symptoms and signs. For instance, vulvar complaints in a 5-year-old are more likely rooted in the fragility of atrophic tissue and an alkaline pH, whereas similar complaints in a 16-year-old must be evaluated through the lens of a mature, cyclical, and glycogen-rich ecosystem. Failure to appreciate these distinctions can lead to misdiagnosis, such as mistaking normal prepubertal redness for pathology or underestimating the significance of recurrent candidiasis in an adolescent. Therefore, a developmentally informed approach forms the cornerstone of effective primary care for pediatric and adolescent vulvovaginal health.

Common Vulvovaginal Dermatologic Conditions:

A systematic approach to vulvovaginal complaints in pediatric and adolescent patients requires familiarity with a core group of prevalent dermatologic diagnoses. These conditions often present with overlapping symptoms—primarily pruritus, discomfort, and erythema—making a careful history and meticulous physical examination paramount[17]. The following section details the clinical presentation, diagnostic pearls, and management considerations for the most frequently encountered entities: nonspecific and specific vulvovaginitis, lichen sclerosus, and the spectrum of dermatitis (irritant, allergic, and atopic).

1. Vulvovaginitis: Vulvovaginitis is the most common gynecologic complaint in prepubertal children, accounting for the vast majority of presentations with vulvar redness, discharge, or discomfort[6],[18]. It is broadly categorized as nonspecific (irritant) or specific (infectious).

- **Nonspecific (Irritant) Vulvovaginitis:** This accounts for approximately 60-75% of cases in prepubertal girls[13],[18].
- It is not a primary infection but an inflammatory response triggered by the fragile, hypoestrogenic vulvovaginal anatomy coupled with local irritants. Common triggers include harsh soaps, bubble baths, tight-fitting synthetic clothing, poor perineal hygiene, and fecal contamination. Symptoms include vulvar erythema, mild discomfort, and sometimes a minor mucoid discharge. Diagnosis is clinical, based on the history of irritant exposure and the absence of specific pathogens on culture. Management centers on eliminating irritants: recommending cotton underwear, front-to-back wiping, bland emollients as barriers (e.g., plain petroleum jelly), and sitz baths with warm water only[13],[19].
- **Specific Infectious Vulvovaginitis:** Less common than the nonspecific type, infectious causes require identification for targeted therapy. In prepubertal girls, these are often respiratory or enteric pathogens spread via the fecal-oral route or poor hand hygiene. Common organisms include *Streptococcus pyogenes* (Group A Strep), which presents with bright-red erythema, often with perianal involvement ("perianal dermatitis"), and *Haemophilus influenzae*[18]. *Candida*vulvovaginitis is uncommon but possible in specific contexts uncommon but possible in specific contexts uncommon but possible in specific contexts uncommon but possible in specific contexts uncommon in healthy prepubertal children~~ but becomes~~ yet exceedingly common post-menarche; driven by the glycogen-rich, estrogenized environment[14]. In adolescents, sexually transmitted infections (STIs) like *Chlamydia trachomatis* and *Neisseria gonorrhoeae* must be considered, necessitating a confidential sexual history and appropriate testing[20]. Diagnosis relies on vaginal culture (obtained via swab of the vaginal introitus, not a speculum exam in children) and wet mount microscopy in adolescents. Treatment is pathogen-directed with appropriate antibiotics or antifungals[19].

Table 2: Common Etiologies and Features of Vulvovaginitis in Pediatric & Adolescent Patients [13],[14],[18],[20]

Type	Common Age Group	Typical Etiology/Pathogen	Key Clinical Features	Diagnostic Clue/Test
Nonspecific	Prepubertal	Local irritants (soaps, moisture, friction) [13],[18].	Diffuse erythema, mild discomfort, no foul discharge.	Clear history of irritant exposure; negative cultures.
Bacterial	Prepubertal	Respiratory/enteric flora (e.g., Group A *Streptococcus*, *H. influenzae*)[18].	Bright-red "beefy" erythema, may have perianal involvement, can have purulent discharge.	Positive vaginal culture for specific pathogen.
Candidal	Post-menarche	*Candida albicans* (primarily)[14].	Intense pruritus, curd-like discharge, erythema, satellite lesions.	KOH microscopy showing hyphae/pseudohyphae; positive culture.
Sexually Transmitted	Sexually active adolescents	*C. trachomatis, N. gonorrhoeae*, *T. vaginalis*[20].	Variable: can be asymptomatic, or present with discharge, spotting, dysuria.	NAAT testing on vaginal swab (first-catch urine also acceptable).

2. Lichen Sclerosus (LS): Lichen sclerosus is a chronic, inflammatory dermatosis of significant importance due to its potential for causing scarring and its strong association with vulvar symptoms in childhood. Up to 15% of all LS cases present in the prepubertal years, and it is the most common cause of persistent vulvar pruritus and soreness in this age group[7,21]. The classic presentation involves intense pruritus, often leading to nocturnal awakening and excoriations. The pathognomonic physical findings include porcelain-white, atrophic papules or plaques that may coalesce into a figure-eight configuration around the vulva and anus. Other hallmark signs include purpura (ecchymoses, fissures), and architectural changes over time such as labial resorption, burying of the clitoris, and narrowing of the introitus[7,22]. In children, the fragility of the skin often makes erosions and fissures more prominent than the hypopigmentation, which can initially lead to misdiagnosis as abuse or eczema. Diagnosis is clinical but should be confirmed by a dermatologist or gynecologist, with biopsy reserved for atypical or treatment-resistant cases. First-line treatment is ultrapotent topical corticosteroids (e.g., clobetasol propionate 0.05%) used in an acute, daily treatment phase followed by a long-term, twice-weekly maintenance regimen to prevent disease progression and scarring[7,21]. Patient education on the chronic nature of the disease and the necessity of maintenance therapy is critical for preventing long-term sequelae.

3. Dermatitis (Eczematous Disorders)

Eczematous inflammation is a leading cause of chronic vulvar pruritus across all age groups. It is crucial to distinguish between subtypes, as management differs[8,23].

- **Irritant Contact Dermatitis:** This is the most common form of vulvar dermatitis, directly resulting from contact with chemical or physical irritants that damage the skin barrier. The pathophysiology and triggers are identical to those described for nonspecific vulvovaginitis, and the two often coexist. Presentation features erythema, burning, and soreness, with signs ranging from mild redness to glazed, weepy, or even fissured skin in severe cases. The pattern often corresponds to the area of contact (e.g., napkin area in infants, entire vulva with bubble bath use). Management is strictly avoidance of irritants and implementation of barrier protection strategies[23].
- **Allergic Contact Dermatitis:** Less common but important to recognize, this is a type IV delayed hypersensitivity reaction. Potential allergens include fragrances and preservatives in soaps, wipes, or topical medications (e.g., neomycin, bacitracin in over-the-counter antibiotic ointments), and even components of sanitary pads or latex[23]. It presents with intense pruritus and marked erythema, which can become edematous and vesicular. The distribution may extend beyond the primary contact site. Diagnosis relies on a meticulous exposure history and can be confirmed by patch testing, though this is rarely performed in primary care. Treatment involves identification and strict avoidance of the allergen, combined with a short course of topical corticosteroids to quell the inflammation[23].
- **Atopic Dermatitis (Eczema):** Children with a personal or family history of atopy (eczema, asthma, allergic rhinitis) are prone to vulvar involvement. It presents with the classic signs of eczema: poorly demarcated erythema, scaling, lichenification (thickened skin) from chronic scratching, and intense pruritus. It is often part of a more generalized skin condition. Management follows general eczema principles: consistent, daily use of bland emollients (e.g., petroleum jelly) to restore the skin barrier, avoidance of soaps and hot water, and judicious use of low- to mid-potency topical corticosteroids (e.g., hydrocortisone 1-2.5%, triamcinolone 0.1%) during flares to control itching and inflammation[8,23].

A structured approach to these common conditions, as summarized in Table 3, enables the primary care provider to initiate appropriate management, improve patient quality of life, and make timely referrals for complex or refractory cases.

Table 3: Diagnostic and Therapeutic Overview of Common Vulvar Dermatoses[7,8,13,21,22,23].

Condition	Primary Symptom	Key Physical Findings	First-Line Management in Primary Care
Lichen Sclerosus	Severe, intractable pruritus; soreness[7,21].	Porcelain-white patches, purpura, fissures, architectural changes (e.g., labial resorption)[7,22].	Ultrapotent topical corticosteroid (e.g., clobetasol) daily for initial control, then maintenance[21].
Irritant Contact Dermatitis	Burning, soreness[23].	Erythema, glazed or weepy skin, may have fissures; corresponds to irritant contact.	Strict irritant avoidance. Use of bland barrier ointment (petroleum jelly)[13,23].
Allergic Contact Dermatitis	Intense pruritus[23].	Marked erythema, edema, may be vesicular; may extend beyond contact area.	Identify and avoid allergen. Short course of topical corticosteroid (e.g., triamcinolone 0.1%)[23].
Atopic Dermatitis	Intense pruritus[8].	Poorly demarcated erythema, scaling, lichenification; history of atopy.	Daily bland emollient. Low-mid potency topical corticosteroid for flares (e.g., hydrocortisone 2.5%) [8,23].

Clinical Presentation and Diagnostic Approaches:

The accurate diagnosis of vulvovaginal dermatologic conditions in pediatric and adolescent patients hinges on a methodical clinical approach that expertly navigates often vague symptoms, employs a sensitive and thorough physical examination, and systematically considers a broad differential diagnosis. The primary challenge lies in the nonspecific nature of presenting complaints; pruritus, pain, burning, and discharge are common to nearly all vulvovaginal disorders, from benign irritant dermatitis to chronic autoimmune conditions[24].

The clinical history must be adapted to the patient's age and cognitive level, often requiring information from both the child and the caregiver. In prepubertal children, symptoms are rarely self-reported as localized genital complaints. Instead, caregivers may observe behavioral cues such as increased irritability, scratching or rubbing the genital area (pruritus), crying during urination (dysuria), or avoidance of activities like bicycle riding or gymnastics. Vulvovaginal pain may manifest as generalized abdominal pain or behavioral changes[17]. Discharge, if present, may be noted on underwear. It is critical to inquire about potential irritants: hygiene practices (bubble baths, scented wipes, harsh soaps), clothing (tight leggings, non-cotton underwear), and recent illnesses (streptococcal pharyngitis preceding vulvovaginitis)[13].

In adolescents, who can provide their own history, a confidential interview is paramount. Open-ended questions should be used to explore the character, timing, and location of symptoms. Is the pruritus constant or intermittent? Is it associated with the menstrual cycle, suggesting a hormonal influence or recurrent candidiasis? Is there a history of atopy (eczema, asthma) pointing toward vulvar dermatitis? A carefully conducted sexual history is a required component of the adolescent evaluation, assessing for the risk of sexually transmitted infections (STIs) and the possibility of sexual abuse or assault, which can both present with vulvovaginal symptoms[20]. The psychosocial impact—embarrassment, anxiety, school avoidance—should also be assessed, as it is often severe and may guide the urgency and manner of intervention[3].

Table 4: Referral Criteria

Clinical Scenario	Specific Criteria	Recommended Action / Specialty
Diagnostic Uncertainty	Atypical presentation, failure to respond to initial appropriate therapy, or suspected but unconfirmed diagnosis (e.g., erosive lichen planus, complex dermatoses)[24].	Refer to **Pediatric Dermatology**or **Pediatric/Adolescent Gynecology** for expert evaluation and possible biopsy.
Suspected Lichen Sclerosus	Clinical findings of porcelain-white plaques, purpura, fissuring, or architectural changes[7],[22].	**Urgent referral** to Pediatric Dermatology or Gynecology for confirmation, education, and initiation of potent topical steroid to prevent scarring.
Chronic/Recurrent Conditions	Conditions requiring long-term, specialized management (e.g., confirmed lichen sclerosus, severe/recalcitrant vulvar eczema, suspected vulvodynia)[24],[28].	Ongoing co-management or consultation with a **sub-specialist**(Dermatology/Gynecology) for treatment optimization and monitoring.
Persistent Symptoms Despite Treatment	No improvement after 4-6 weeks of appropriate first-line therapy (e.g., candidiasis unresponsive to antifungals, dermatitis unresponsive to low-potency steroids)[13],[14].	Refer to **Pediatric Dermatology**or **Gynecology** for re-evaluation, culture, or alternative diagnosis.
Suspected STI or Need for Confidential Services	Adolescent patient with risk factors, request for confidential testing, or positive STI screen requiring complex management[20].	Refer to **Adolescent Medicine**, **Pediatric Gynecology**, or **Infectious Disease** for comprehensive sexual health care and partner management.
Suspicion of Sexual Abuse	Any disclosure, concerning physical findings (e.g., unexplained injuries, recurrent STIs), or high-risk behavioral indicators[20].	**Immediate, mandatory referral**to local **Child Protection Services** and a **specialized pediatric child abuse assessment team** (e.g., Child Advocacy Center).

Note: Timely referral to an appropriate specialist is crucial for accurate diagnosis, preventing disease sequelae (e.g., scarring from lichen sclerosus), and providing comprehensive, age-appropriate care.

The vulvar examination must be performed with utmost sensitivity to minimize fear and trauma, thereby ensuring cooperation and yielding more accurate findings. For all ages, explanation and consent are mandatory. A parent or chaperone should be present. The examination is visual and does not require speculums or internal maneuvers in prepubertal patients. No speculum exams are performed in children. The approach should also respect cultural sensitivities and family dynamics, engaging guardians appropriately to provide reassurance and support.

1. **General Inspection:** Begin with the patient in the frog-leg or lithotomy position. Use a gentle, distraction-based conversation. Observe the general appearance of the vulva, noting the architecture of the labia majora and minora, the presence and distribution of pubic hair (Tanner staging), and any signs of systemic skin disease (e.g., psoriatic plaques elsewhere).

2. **Focused Vulvar Examination:** Good lighting is essential. Gently separate the labia to fully expose the vestibule, clitoris, hymen, and vaginal introitus. Systematically assess for:
    - **Color:** Diffuse erythema (suggestive of dermatitis or vulvovaginitis) versus focal or patterned changes (e.g., the porcelain-white plaques of lichen sclerosus)[7].
    - **Surface Texture:** Moisture, dryness, lichenification (thickened skin from chronic scratching, typical of atopic dermatitis), atrophy (thin, shiny skin in lichen sclerosus), or erosions[25].
    - **Lesion Morphology:** Note any papules, vesicles, pustules, ulcers, or warty growths. The presence of purpuric areas (bruising, blood blisters) is a highly suggestive sign of lichen sclerosus and should be carefully distinguished from traumatic injury[7],[22].
    - **Discharge:** Note the color, consistency, and quantity of any discharge from the introitus.

3. **Additional Maneuvers:** A digital examination is not part of the routine prepubertal vulvar dermatologic exam. In adolescents, if indicated by history (e.g., concern for pelvic inflammatory disease), a single-digit bimanual exam may be performed. A rectal exam is rarely needed. Sampling for culture or wet mount in a prepubertal child can be achieved by gently touching a moistened swab to the vaginal introitus or by using a catheter-within-a-catheter technique without instrumentation[26].

Formulating a Differential Diagnosis

The differential diagnosis is constructed by synthesizing historical clues with physical findings. Key distinguishing features can guide the clinician. For example, the symptom of **pruritus** leads to a primary differential of inflammatory dermatoses: lichen sclerosus (with its classic whitish plaques and purpura), atopic or contact dermatitis (poorly demarcated or patterned erythema), and lichen planus (erosive or lacy white lesions)[27].

Pain or burning is more characteristic of erosive conditions (lichen planus, herpes simplex), vulvodynia (pain without visible findings), or severe irritant dermatitis

. **Discharge** shifts focus toward infectious vulvovaginitis, with the character (purulent, curd-like, frothy) and associated findings guiding testing.

A critical step is distinguishing between conditions that can be managed in primary care and those requiring prompt specialty referral. Red flags for referral include: 1) Findings suspicious for lichen sclerosus (to confirm diagnosis and initiate potent topical therapy), 2) Treatment-resistant or severe dermatitis, 3) Erosive, ulcerative, or blistering lesions, 4) Persistent or recurrent discharge unresponsive to first-line therapies, and 5) Any concerning lesion for malignancy (though exceedingly rare in this age group)[28]. Table 5 provides a symptom-based framework to guide the initial differential diagnosis.

Table 5: Symptom-Based Differential Diagnosis of Common Pediatric Vulvovaginal Conditions [7,8,13,14,18,20,22,23,27–29]

Most Common Diagnoses	Key Distinguishing Physical Findings	Supportive Diagnostic Actions
Primary Symptom: Pruritus (Dominant)		
Lichen Sclerosus	Porcelain-white plaques (hypopigmentation), purpura, fissures, loss of architecture (fusion, resorption)[7],[22].	Clinical diagnosis. Dermatology referral for confirmation & management. Biopsy if atypical.
Atopic / Irritant Dermatitis	Poorly demarcated (atopic) or geometric (irritant) erythema, scaling, lichenification; history of atopy or irritant exposure[8],[23].	Trial of irritant avoidance & low-potency topical steroid. Patch testing for suspected allergen.
Candidiasis (Post-pubertal)	Erythema, satellite pustules, curdy white discharge, vulvar edema[14].	KOH microscopy (hyphae), vaginal pH (<4.5). Culture if recurrent/complicated.
Lichen Simplex Chronicus	Focal, well-demarcated lichenified plaque (thickened skin with accentuated markings) from chronic scratching[24].	Clinical diagnosis, often secondary to another pruritic condition. Biopsy to rule out neoplasia if persistent.
Pinworm Infection	Often nocturnal pruritus; may see excoriations; worms sometimes visible on perianal skin[25].	"Scotch tape test" on perianal skin in AM to identify ova.
Primary Symptom: Pain/Burning (Dominant)		
Acute Irritant Dermatitis	Glazed, weepy, or fissured erythema in a pattern matching exposure (e.g., diaper area, wipes)[13],[23].	History of exposure (soaps, creams, moisture). Resolves with strict barrier protection/avoidance.
Herpes Simplex Virus (HSV)	Painful, grouped vesicles or shallow ulcers on an erythematous base[29].	Viral PCR swab of lesion base. Type-specific serology if needed for recurrence/atypical presentation.
Lichen Planus (Erosive)	Bright-red, well-demarcated erosions; lacy white striae (Wickham's striae) may be seen at periphery[27].	Requires gynecology/dermatology referral. Biopsy from edge of lesion (peri-lesional skin) for diagnosis.
Vulvodynia (Provoked)	Severe pain to light touch (allodynia) or pressure (Q-tip test) in absence of visible pathology[28].	Diagnosis of exclusion after ruling out infectious/dermatologic causes. Map areas of tenderness.
Candidiasis (Acute)	Intense burning with erythema, edema, and satellite lesions. Discharge may be minimal[14].	KOH microscopy and culture. Rule out secondary irritant dermatitis from creams/treatments.
Primary Symptom: Discharge (Dominant)*		
Physiologic Leukorrhea(Prepubertal & Adolescent)	Clear to white, mucoid, non-irritating discharge. Normal vulvar and vaginal mucosa[13].	Diagnosis of exclusion. Reassurance after ruling out infection.
Nonspecific Vulvovaginitis(Prepubertal)	Mild mucoid or mucopurulent discharge, diffuse vulvar erythema[13],[18].	Diagnosis of exclusion. Bacterial culture often shows mixed flora. Rule out foreign body, pinworms.
Bacterial Infection (e.g., **Group A Strep**, *S. aureus*)	Purulent discharge, bright-red "beefy" or intensely erythematous vulvovaginitis, may be painful[18].	Bacterial culture of introital discharge (not cervical). Rapid strep test possible.
Candidiasis (post-pubertal)	Thick, white, curdy discharge with associated pruritus/burning and erythema[14].	KOH microscopy, vaginal pH (<4.5).
Sexually Transmitted Infection (Adolescents: **C. trachomatis, N. gonorrhoeae, T. vaginalis**)	Variable discharge (purulent, frothy), but may be asymptomatic. Cervical friability possible[20].	NAAT testing for *C. trachomatis/N. gonorrhoeae*. Wet mount for *Trichomonas*.

Therapeutic Strategies and Management Guidelines:

The management of vulvovaginal dermatologic conditions in pediatric and adolescent patients extends beyond accurate diagnosis to encompass a nuanced, evidence-based therapeutic strategy. Effective treatment must be tailored to the specific diagnosis, the patient's developmental stage, and her unique psychosocial context. The therapeutic armamentarium includes pharmacologic agents, essential non-pharmacologic interventions, and foundational preventive measures. Success hinges on a patient-centered approach that prioritizes clear education, addresses treatment barriers (such as embarrassment with topical application), and establishes realistic expectations for chronic conditions[30]. Before initiating any pharmacologic treatment, implementing consistent, gentle vulvar care is a critical first-line and adjunctive strategy for nearly all conditions. This "vulvar hygiene" protocol aims to eliminate irritants, restore the skin barrier, and minimize friction[31].

Key recommendations include: using lukewarm water only for cleansing (no soap on vulvar tissue); patting the area dry gently with a soft towel; wearing loose-fitting, breathable cotton underwear; avoiding occlusive clothing like tights or leggings for extended periods; and using a protective barrier ointment (such as plain petrolatum or zinc oxide) to shield skin from moisture and irritants, particularly in prepubertal children or those with dermatitis[13],[31].

For pruritus, strategies such as cool compresses, colloidal oatmeal baths, and keeping fingernails short can help prevent excoriation and secondary infection. These measures are not merely supportive; for conditions like irritant contact dermatitis, they are often curative.

Condition-Specific Pharmacologic Management

1. Lichen Sclerosus (LS): The treatment of LS is one of the most critical interventions in pediatric vulvar dermatology, as appropriate therapy can prevent disease progression and scarring. The cornerstone of treatment is superpotent topical corticosteroids. The standard regimen involves an initial, intensive phase using clobetasol propionate 0.05% ointment applied once daily for 4-12 weeks to achieve symptom remission and resolve active inflammation[21],[32].

This is followed by a long-term maintenance phase to prevent flares, typically applying the same steroid once or twice weekly indefinitely. Studies confirm that this maintenance regimen is safe and effective in preventing architectural changes[7],[32].

Patient and caregiver education is paramount to ensure adherence to maintenance therapy. Regular follow-up every 6-12 months is recommended to assess response, adjust therapy, and monitor for side effects (although local atrophy is rare with this intermittent regimen). Tacrolimus 0.1% ointment, a topical calcineurin inhibitor, is a second-line option for cases unresponsive or intolerant to corticosteroids, though its use may be off-label in children and it carries a black box warning[33].

2. Eczematous Dermatitis (Atopic and Allergic Contact)

Management follows a stepwise approach based on severity. For mild to moderate flares, low to mid-potency topical corticosteroids are first-line. Hydrocortisone 1-2.5% or triamcinolone 0.1% ointment applied thinly once or twice daily for a short course (e.g., 2 weeks) is usually sufficient[8],[23]. For allergic contact dermatitis, identifying and eliminating the allergen is curative, with corticosteroids serving to calm the acute hypersensitivity reaction. For chronic management and prevention of atopic dermatitis flares, daily use of a bland emollient (e.g., petrolatum) as a moisturizer and barrier is essential. In severe or refractory cases, referral to a dermatologist is warranted for consideration of advanced therapies.

3. Infectious Vulvovaginitis

Treatment is pathogen-directed:

- **Nonspecific Vulvovaginitis:** Managed solely with non-pharmacologic measures and irritant avoidance[19].
- **Bacterial Infections (e.g., Group A Streptococcus):** Treated with a course of appropriate oral antibiotics, such as amoxicillin or a first-generation cephalosporin, based on culture and sensitivity results[18].
- **Candidiasis:** Treated with topical azole antifungals. For adolescents, a single-dose fluconazole 150 mg oral tablet is often highly effective and preferred for adherence[14].
- **Sexually Transmitted Infections:** Managed strictly according to current CDC guidelines, which emphasize single-dose, directly observed therapy for conditions like gonorrhea and chlamydia to ensure cure and prevent spread[20],[29]. Partner notification and treatment are essential components.

Table 6: Summary of First-Line Therapeutic Strategies for Common Vulvovaginal Conditions

Condition	First-Line Pharmacologic Therapy	Key Non-Pharmacologic & Preventive Measures	Monitoring & Referral Considerations
Lichen Sclerosus	**Acute:** Clobetasol propionate 0.05% ointment daily for 4-12 wks. **Maintenance:** Same, 1-2x/week indefinitely [7],[32].	General vulvar care. Education on chronic nature & critical importance of maintenance therapy.	Follow-up q 6-12 mos. Refer to specialist for diagnosis confirmation & initiation of therapy.
Atopic/Contact Dermatitis	**Flares:** Low-mid potency topical corticosteroid (e.g., triamcinolone 0.1%) for 2-wk course[8],[23].	Strict irritant avoidance. Daily use of bland emollient/barrier (petrolatum).	Refer if refractory to 2-4 weeks of treatment or if severe.
Candidiasis (Adolescent)	Fluconazole 150 mg single oral dose **OR**topical azole cream (e.g., miconazole) nightly for 3-7 days[14].	Avoid unnecessary antibiotics; wear cotton underwear; manage blood glucose if diabetic.	Recurrence >4x/year warrants workup for predisposing factors.
Bacterial Vulvovaginitis	Culture-directed oral antibiotics (e.g., amoxicillin for GAS)[18].	Improved perineal hygiene; handwashing; treat concurrent perianal symptoms.	If recurrent, consider evaluation for foreign body.
STI (e.g., Chlamydia)	Azithromycin 1g single dose **OR** Doxycycline 100 mg BID x 7 days[20],[29].	Condom use; partner notification & treatment; sexual health counseling.	Mandatory reporting per local regulations. Test-of-cure recommended in 3 months.

Preventive Measures and Long-Term Health
Prevention is a cornerstone of management, particularly for chronic or recurrent conditions. For all patients, education on general vulvar care is a preventive foundation[31]. Specific strategies include:

- **For LS:** Emphasizing that maintenance topical steroid therapy is preventive, not merely for symptom relief, and is necessary to prevent scarring and sexual dysfunction later in life[32].
- **For Dermatitis:** Identifying and avoiding personal irritant and allergen triggers through a careful exposure history.
- **For Recurrent Candidiasis:** Counseling on avoiding excessive moisture, wearing breathable fabrics, and discussing potential triggers like prolonged antibiotic use.
- **For STIs:** Providing comprehensive sexual health education, promoting condom use, and encouraging regular screening for sexually active adolescents.

Primary care management has its limits. Indications for referral to a pediatric/adolescent gynecologist or dermatologist include: 1) Diagnostic uncertainty, especially for suspected LS, lichen planus, or vulvar intraepithelial neoplasia; 2) Failure to respond to 4-6 weeks of appropriate first-line therapy; 3) Severe, erosive, or scarring disease; 4) Need for procedural intervention (e.g., biopsy, adhesiolysis); and 5) Complex chronic pain conditions like refractory vulvodynia[28],[34]. A collaborative, multidisciplinary model often yields the best outcomes for complex patients[35].

CONCLUSION

Vulvovaginal conditions in children and adolescents present a complex clinical challenge, complicated by nonspecific symptoms, developmental physiology, and significant psychosocial barriers. A methodical approach, grounded in recognizing age-specific norms and mastering common presentations like lichen sclerosus, dermatitis, and infection, is essential for accurate diagnosis.

Management requires targeted treatment paired with diligent hygiene and irritant avoidance. For chronic conditions, successful long-term outcomes depend on thorough patient and caregiver education. The primary care provider is pivotal as the first point of contact, a key educator, and the coordinator of specialty care when necessary.

LIMITATIONS

It is important to note the limitations inherent in a narrative review such as this. The conclusions are based on a selective synthesis of available literature rather than a systematic, protocol-driven analysis of all evidence, which may introduce author bias in study selection and interpretation. Furthermore, the dynamic nature of

clinical guidelines and the relative scarcity of high-level pediatric-specific research mean that some recommendations are necessarily grounded in expert opinion and adult data extrapolation. Therefore, while this review provides a structured clinical framework, its guidance should be integrated with ongoing clinical judgment and the latest evidence.

DECLARATIONS

Ethics Approval and Consent to Participate
Not Applicable.

Funding
None.

Competing Interests
None.

Authors' Contributions
M.M.A., Y.A.G., and M.A.A. were responsible for the conceptualization, methodology, and initial draft preparation of the narrative review. W.S.A., B.S.A., R.M.J., and A.K.I.E.E. conducted the formal literature analysis, data curation, and creation of the clinical tables. A.M.B., A.M.S., and H.A.A.A. contributed to the critical review, editing, and visualization of the manuscript. A.A.A. provided expert supervision, project administration, and final validation. All authors reviewed, edited, and approved the final manuscript for submission.

Acknowledgements
None.

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