Dermatologic Therapy, Vol. 26, 2013, 157–167
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DERMATOLOGIC THERAPY
ISSN 1396-0296

INVITED ARTICLE

Female-specific pruritus from childhood to postmenopause: clinical features, hormonal factors, and treatment considerations

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ABSTRACT: There have been considerable advances in our understanding of the pathophysiology of pruritus in recent years. The purpose of this review was to highlight itch entities in women, and in particular pruritic vulvar dermatoses that women experience among different age groups. Unique temporal shifts may contribute to the etiology of many of these conditions. These changes lead to cyclical changes in the skin's basic composition. Specifically, estrogen receptors have been detected on keratinocytes that respond to rising and falling levels of estrogen. These receptors lead to changes in skin hydration, collagen content, and in the concentration of glycosaminoglycans that form the skin barrier. In addition, hormonal pH changes associated with the menstrual cycle may be an important factor in the aggravation of itch as increasing pH is known to activate the proteinase-activated receptor-2, a well-known itch mediator. Common pruritic conditions in women that will be discussed include atopic and irritant dermatitis, psoriasis, lichen sclerosus, infectious vulvovaginitis, vulvovaginal candidiasis, atrophic vulvovaginitis, squamous cell carcinoma, lichen simplex chronicus, and neuropathic itch. We also examine pruritic conditions associated with pregnancy including pemphigoid gestationis, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy and atopic eruption of pregnancy. Finally, acceptable and contraindicated antipruritic agents in pregnancy are examined.

KEYWORDS: itch, pruritus, vulvovaginal itch, female itch

Introduction

Recent studies have shed new light onto the pathophysiology of pruritus and the various etiologies underlying itch. Pruritic conditions are being more widely studied, and there are an increasing number of reports describing the pruritic dermatoses specific to women. It is important to distinguish between genders when considering the basic mechanisms and manifestations of these dermatoses. On an anatomical level, the vulva has distinct epithelial characteristics in its different regions (FIG. 1) that are important to a number of disease processes (see Table 1). In addition, unique to women are temporal hormonal shifts that lead to

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Funding/Conflicts of Interest: None.

cyclical changes across the age spectrum in the skin's basic composition.

Estrogen receptors have been detected on keratinocytes; as such, rising and falling levels of estrogen throughout the life cycle lead to changes in hydration, collagen content, and concentration of glycosaminoglycans (1). Downstream effects include changes in vulvovaginal pH and varying microflora compositions (1). As a result, there exists a diversity of skin pathologies and phenotypes that tend to fall into hormonal-dependent groupings (prepubertal, reproductive age, postmenopausal). These conditions impact the quality of women's lives and are known to cause significant discomfort. Yet to date, little effort has been placed on studying female-specific itch. This is particularly true in the case of vulvar dermatoses, which are often under-recognized and undertreated. Herein, we will examine the causes, manifestations, and management options of vulvar itch in women from childhood to postmenopause.

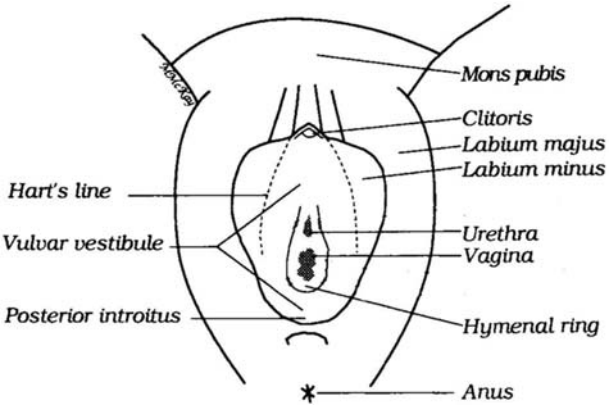

FIG. 1. Vulvar anatomy showing Hart's line, the demarcation between keratinized (lateral to Hart's line) and nonkeratinized epithelium. ***Figure courtesy of Marilynne McKay, MD.***

Prepubertal vulvar pruritus

In childhood, the vulvar and vaginal epithelium is characterized by low estrogen levels, a high vulvovaginal pH, and a lack of genital lactobacillus colonization. The most common causes of vulvar itch before menarche are atopic and irritant dermatitis, psoriasis, and lichen sclerosus (LS). Streptococcal infection of the vulva exclusively occurs in prepubertal girls, and poor hygiene, foreign bodies, and sexual abuse should also be considered as causes of vulvar pruritus in this age group.

Atopic and irritant dermatitis

The most common causes of prepubertal vulvar itch are atopic and irritant dermatitis, often occurring together (2). These dermatoses manifest as fluctuating, poorly defined erythematous patches and plaques involving the vulvar area that are exacerbated by excessive washing and overuse of antifungal creams (3). Scale and slight rugosity may be seen on the labia majora, while desquamation and redness often occur in the labia minora (3). The skin is so pruritic that scratching is difficult to control; this may interfere with sleep and cause parental embarrassment (FIG. 2).

Superinfection by *Staphylococcus aureus* may be present, but rarely yields positive cultures (3). Management includes reduction in irritant exposures (commonly urine, feces, soaps, bubble baths), as well as application of low-dose topical steroids (2). A mainstay of therapy for secondary skin infections are bleach baths consisting of a half cup of clorox in a tub of water.

Psoriasis

Psoriasis more commonly affects the vulva in children than it does adults (2). These lesions often

Table 1. Vulvar structures, epithelial patterns, and associated diseases

Vulvar skin type	Epithelial characteristics	Associated diseases
Labia majora and outer minora	Keratinized stratified squamous epithelium Eccrine, apocrine, and sebaceous glands	Psoriasis Lichen sclerosus Allergic and irritant dermatitis Atopic eczema
Inner labia minora	Nonkeratinized stratified squamous epithelium No adnexal structures	Lichen planus
Vaginal vestibule	Mucosa No adnexal structures	Lichen planus Vulvovaginal candidiasis Atrophic vulvovaginitis

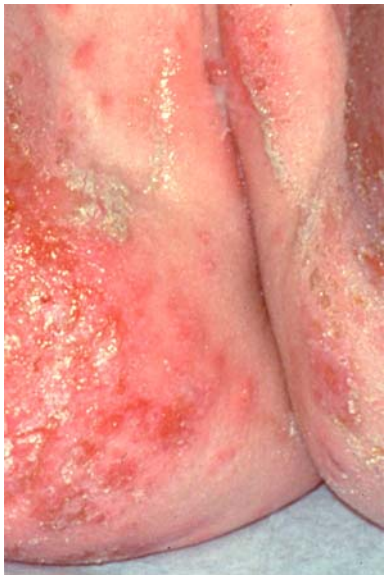

FIG. 2. Secondarily infected dermatitis in an atopic child with intense vulvar itching. Staphylococcus and streptococcus were both present, but the skin cleared promptly with bleach baths (bleach baths, 15 min daily for 1 week) and a midpotency topical steroid ointment. *Photograph courtesy of Marilynne McKay, MD.*

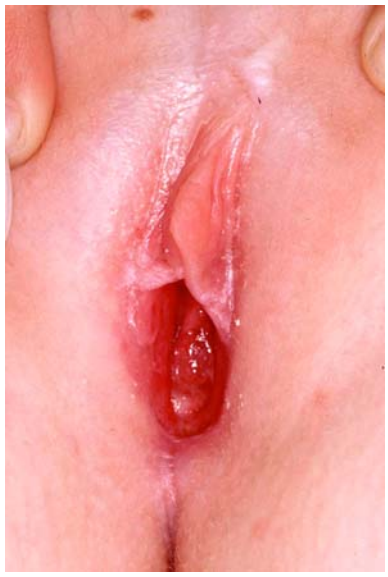

FIG. 3. Lichen sclerosus in a 7-year-old. Note pale appearance of the inner labia majora with early resorption of the labia minora. Early involvement of the clitoral hood is typical and the perineal body shows early atrophic changes. The vagina is easily visualized because of loss of vulvar architecture. *Figure courtesy of Marilynne McKay, MD.*

initially present in babies as a persistent diaper rash; in older children, they become pruritic, well-demarcated, symmetric red plaques without scale in the vulvar and perianal regions (3). When psoriasis is limited to the vulva diagnosis may be difficult, but other findings such as nail pitting, history of cradle cap, and presence of scalp/post-auricular rashes can help confirm the etiology (3). Vulvar psoriasis is managed with high-potency topical steroids.

Lichen sclerosus

Perhaps the most well recognized vulvar dermatosis of childhood, lichen sclerosus (LS) affects approximately 1 in 900 girls in the United States, with 7–15% of all cases found in the prepubertal age group (4). This chronic, autoimmune, mucocutaneous inflammatory dermatosis of unknown etiology classically presents as a "figure-of-eight" shaped white plaque with secondary atrophy and subcutaneous hemorrhage of the vulvar and perianal skin (2). Pale "confetti" spots can sometimes be seen on the inner wrists or elsewhere on the body; extragenital lesions are often asymptomatic. The vulvar rash is extremely pruritic and can cause soreness, dysuria, and chronic constipation (3). The presence of petechiae and purpura may lead to inappropriate investigation into sexual abuse in this age group (2). Onset typically occurs between the ages of 4 and 5 years old, but patients experience symptoms for an average of 1 year prior to diagnosis (4). Complications include loss of genital architecture secondary to scarring and subsequent effacement of the labia minora and clitoris (5) (FIG. 3). While some pediatric LS resolves with puberty, other cases may silently progress into adulthood. Longstanding LS is associated with a less than 5% chance of squamous cell carcinoma (SCC) later in life (6–8). LS is treated with high-potency topical steroids and frequent follow-up (5).

Infectious vulvovaginitis

Group A beta-hemolytic streptococcal infection can cause vulvar symptoms that typically occur in the female prepubertal age group (9). These infections may present in acute or subacute forms. In the more severe, acute form, patients present with erythematous, painful, edematous plaques with discharge. Alternatively, they may present with subacute inflammation, manifested as pruritic erythematous patches and plaques in the vulvar and perianal regions. Streptococcal infections are diagnosed by vaginal swabs and treated with penicillin, amoxicillin, or cephalexin (if penicillin allergic). More rare infections in

prepubertal girls include staphylococcus, hemophilus, and shigella (2). Pinworm is a common etiology of vulvar and perianal pruritus and may be associated with an eczematous rash. Pinworm is treated with mebendazole (3). Although tinea infections can occasionally be seen in girls, vulvovaginal candidiasis (VVC) does not occur before menarche in immunocompetent patients.

Reproductive age vulvar pruritus

With menarche, an increase in baseline estrogen and the initiation of cyclic hormonal changes create a new cutaneous environment. At puberty, estrogen begins to act on maturing keratinocytes, causing vulvovaginal pH to decrease from an average of 7 in prepubertal girls to an average of 4 in adult women (10). During this hormonal transition, the vulvar epithelium becomes rich in glycogen, and lactobacilli begin to colonize the vulvovaginal area. With the onset of the menstrual cycle, monthly variations in vaginal physiology and pH come into play. In the first half of the cycle, estrogen levels rise, and vulvovaginal epithelial cells proliferate, whereas in the second, progesterone-mediated half of the cycle, these keratinocytes desquamate (11). The bacterial flora of the vulvovaginal region also changes in a cyclic manner. Finally, fluctuations in hormonally controlled pH levels may cause pruritus, as increasing pH is known to activate the proteinase-activated receptor-2 (PAR-2) (12), a well known itch mediator.

Common causes of vulvar pruritus in reproductive-age, nonpregnant women include VVC, allergic and irritant dermatitis, lichen simplex chronicus (LSC), psoriasis, and to a lesser extent lichen sclerosus. Other, less common causes are lichen planus, vaginal infections, herpes vulvovaginitis, and seborrheic dermatitis.

Vulvovaginal candidiasis

The majority of women of reproductive age experience at least one episode of vulvovaginal candidiasis (VVC) in their lifetime, and approximately 50% experience multiple episodes (13). Vulvovaginal colonization with yeast is an estrogen-dependent process, and as a result, occurs almost exclusively in the reproductive years, usually in the premenstrual period when hormone levels are high (13). Vulvovaginal candidal colonization has been estimated to have an 11–22% prevalence rate among women of different age groups (14–16). Certain conditions and medications may increase estrogen levels and lead to more frequent colonization as well as infections. These include pregnancy, antibiotic use, the use of hormonal birth control methods, hormone replacement therapy, and tamoxifen (2,13–16). Changes in immune regulation can also cause yeast infections, including diabetes, HIV, thyroid disease, lupus, corticosteroid use, and inheritance of a polymorphism associated with low production of mannose binding lectin (13). Recurrent VVC is defined as the occurrence of least four episodes within 1 year, or at least three episodes in one year not associated with antibiotic use (2). These women are usually otherwise healthy and develop a hypersensitivity-like reaction to candida.

VVC presents as itching and burning of the vulva, often with a white discharge and vulvovaginal redness. Dysuria and dysparenunia may be present as well. VVC is largely overdiagnosed in women with vulvar itch; self-diagnosis is poor, particularly in cases of recurrent vulvovaginal symptoms (13). Diagnosis can be made by wet prep to visualize fungal elements, culture or polymerase chain reaction when wet prep is indeterminate, and a normal vaginal pH to rule out bacterial vaginosis, atrophic vaginitis, and trichomoniasis (13). Treatment centers around topical or systemic antifungal azoles, with resolution of symptoms in 2–3 days (13).

While the majority of cases are associated with *Candida albicans*, other species such as *C. glabrata*, *C. tropicalis*, and *C. parapsilosis* may occur and be relatively treatment resistant. In such cases, fungal culture, rather than wet mount alone, is necessary for diagnosis (17). Recurrent VVC (RVVC) may require a long-term treatment regimen of a weekly or biweekly suppressive azole antifungal (18).

Allergic and irritant contact dermatitis

Allergic and irritant contact dermatitis account for approximately 50% of cases of chronic vulvovaginal pruritic symptoms (13). These dermatoses present in a nonspecific fashion, with sudden or gradual onset of itching, burning, and erythema. Table 2 displays common offending agents in allergic and irritant dermatitis. With acute irritants, redness can be localized to the area of contact (if in solid or cream form), or may be diffuse (if in water soluble or liquid form). In subacute and chronic cases, erythema, swelling, and lichenification may occur (FIG. 4); vesiculation is uncommon on the vulva (13). Because of recent trends of cosmetic hair removal, the skin may be more sensitive to a host of irritants that were previously unknown.

Table 2. Common offending agents in allergic and irritant contact dermatitis of the female genital area (2,13)

Allergic contact dermatitis	Neomycin
	Clobetasol
	Benzocaine
	Lanolin
	Dyes (clothing and black hair dye)
	Thiuram (in rubber condoms)
	Sanitary pads
	Perfumes
	Sodium metabisulfite (in topical antifungal creams)
Irritant contact dermatitis	Antifungal or menstrual-related topical creams and liquids
	Harsh soaps and antiseptics
	Urine
	Douches
	Lubricants and spermicides
	Tampons and sanitary pads
	Synthetic underwear

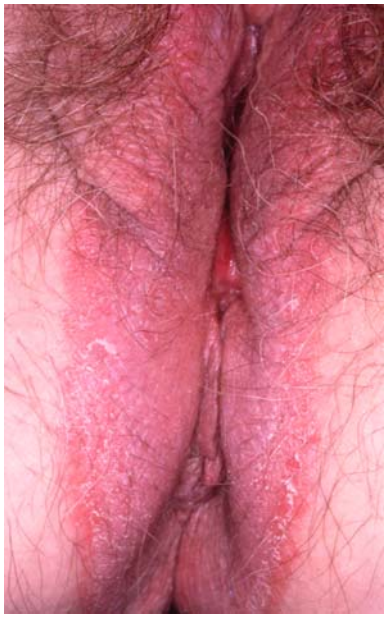

FIG. 4. Chronic contact dermatitis due to neomycin ointment. Note that the rash extends into the gluteal cleft and perineal area where the patient applied the medication. *Figure courtesy of Marilynne McKay, MD.*

Allergic contact dermatitis of the vulva has a similar presentation, but may be delayed or intermittent in nature. Continuous exposure to an allergen triggers an itch-scratch cycle that leads to the development of the typical thickened plaques of LSC. Diagnosis is made by medical history, and while patch testing is not routinely performed, it can be helpful in certain cases. These dermatoses are treated by removing the offending agent, avoiding overwashing, and applying topical steroids until the skin returns to normal.

Lichen simplex chronicus and neuropathic itch

Chronic rubbing and scratching of the skin results in the typical appearance of lichen simplex chronicus (LSC). On the vulva, the skin becomes thickened, lichenified, and often hyperpigmented (FIG. 5). Various etiologies of LSC exist; the condition can occur secondary to pruritic conditions such as LS or allergic contact dermatitis, may be part of a systemic neuropathy, or in some cases, is a primary psychogenic process (13). When LSC is suspected in the vulvar area, it is important to rule out neuropathic itch associated with sacral spinal compression: a lumbar X-ray may be helpful in identifying possible involvement of the dorsal root ganglia (19). Other types of neuropathic itch include postherpetic neuralgia and diabetic neuropathy. Treatment hinges on breaking the cycle of itching and scratching with behavior modification, anti-itch medications, anticonvulsants such as gabapentin and pregabalin (20) and topical steroids.

Psoriasis

Genital itch in psoriatic women is very common, and in rare cases, the vulva may exclusively be

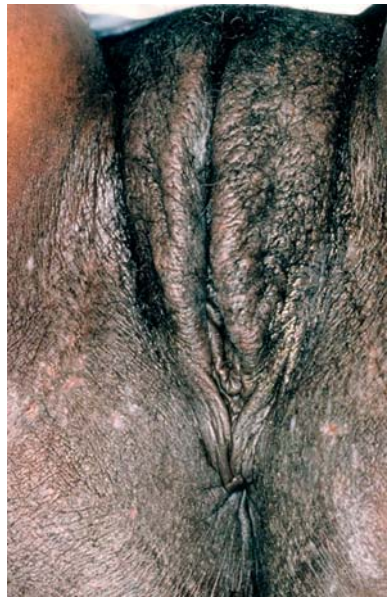

FIG. 5. Lichen simplex chronicus due to chronic rubbing and scratching of pruritic skin. The surface shows thickening with increased skin markings and hairs have been broken because of trauma. Hyperpigmentation is a common finding in black patients with lichen simplex chronicus. *Figure courtesy of Marilynne McKay, MD.*

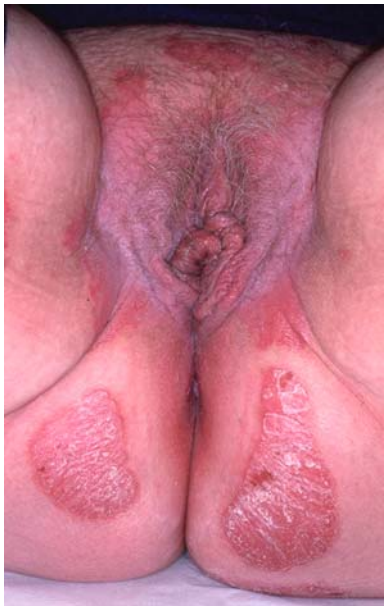

FIG. 6. Psoriasis on the vulva can be intensely red. This patient has typical dry scaly psoriatic plaques on the buttocks, but the vulva appears white becauase of maceration and retention of moisture in the thickened stratum corneum. ***Figure courtesy of Marilynne McKay, MD.***

affected (21). Because the vulva has more hydration relative to exposed skin, psoriatic plaques lack scale, although maceration is common (13). Psoriasis only affects keratinized skin, such as the labia majora and mons pubis; as such, the vestibule and vagina are spared (FIG. 6). On the labia majora, lesions tend to be well-defined, symmetric, salmon pink to beefy red plaques, whereas intertriginous areas may appear glossy, smooth, and red. Vulvar itching may occur in the absence of visible vulvar lesions in psoriatic patients. Personal or family history of psoriasis and visualization of psoriatic lesions elsewhere on the body are integral to the diagnosis of vulvar psoriasis (2). Treatment includes moderate to high-potency topical steroids; oral antifungals are helpful for concomitant candidiasis.

Pruritus in pregnancy

Of all the distinct hormonal stages of a woman's life, pregnancy is the most well studied with regard to pruritic skin conditions. In addition to the specific dermatoses of pregnancy, pregnancy is also associated with physiologic changes in normal skin, as well as alterations of preexisting or acquired skin disease including other dermatoses.

In this section, we will focus on the four specific dermatoses of pregnancy: pemphigoid gestationis, polymorphic eruption of pregnancy, intrahepatic cholestasis of pregnancy (ICP), and atopic eruption of pregnancy (specific features of these conditions can also be found in Table 3). Another cause of itch that is common in pregnancy (but not exclusive to it) is meralgia paresthetica (22), which often features itch over the lateral and anterolateral thigh. Meralgia paresthetica is secondary to neuropathy of the lateral femoral cutaneous nerve. Finally, Table 4 provides a list of acceptable and contraindicated antihistamines in the treatment of itchy dermatoses of pregnancy.

Polymorphic eruption of pregnancy

Polymorphic eruption of pregnancy, also referred to as pruritic urticarial papules and plaques of pregnancy, is a self-limited pruritic inflammatory condition that usually occurs in late pregnancy or postpartum. While the pathophysiology of the condition is unknown, it is associated with maternal weight gain and multiple pregnancies. As such, some theories suggest that the lesions are spurred by distention and overstretching of skin. Patients present with severely pruritic papules that coalesce into plaques; eventually, they may become vesicular, targetoid, and eczematous. They begin within the striae distensae on the abdomen and can spread to the buttocks and proximal thighs. Characteristically, this rash spares the umbilical region, unlike other dermatoses of pregnancy. The condition is diagnosed by clinical picture and history, and the rash generally resolves within 6 weeks of delivery. The pruritic lesions can be managed symptomatically with topical corticosteroids with or without antihistamines.

Pemphigoid gestationis

Pemphigoid gestationis, otherwise known as herpes gestationis, is a rare bullous autoimmune disease. This condition tends to occur in the third trimester or immediately postpartum, and presents as intensely pruritic erythematous urticarial papules and plaques on the abdomen that eventually progress to tense bullae. Pruritus may precede lesions in many cases. The rash typically involves the umbilical region, but can involve all skin surfaces (although bullae rarely appear in the mucous membranes). In the prebullous stage, this condition presents as erythematous papules and plaques involving the periumbilical regions and

Table 3. Pregnancy-related skin diseases associated with itch (23,30)

Condition	Presentation	Pathophysiology	Diagnosis	Treatment
Polymorphic eruption of pregnancy	Polymorphous eruption Urticarial papules within striae distensae Papules and plaques spread to buttocks and thighs Spares periumbilical region	Unknown Thought to be due to abdominal distension, hormonal, and immunological factors	Negative immune studies Typical histology spongiosis and superficial perivascular infiltrate	Topical or oral corticosteroids Antihistamines
Pemphigoid gestationis	Pruritic urticarial and bullous eruption Prebullous stage with urticarial papules Bullous stage with tense bullae	Autoimmune Immunoglobulin G antibodies bind BP-180 in hemidesmosomes of DEJ	Direct IF of perilesional skin Linear C3 deposition along DEJ on IF	Topical or oral corticosteroids
Intrahepatic cholestasis of pregnancy	Exclusively secondary changes caused by scratching Pruritus begins on palms/soles and then generalizes Excoriations and prurigo nodules on extensor surfaces	Defect in excretion of bile salts Elevated bile acids in serum cause pruritus	Rise in serum bile acids level >11 micromol/L LFTs may be normal	Ursodeoxy-cholic acid (15 mg/kg/day or 1g/day qday or BID-TID)
Atopic eruption of pregnancy	Widespread eczematous eruption Excoriated papules and nodules	Immunologic changes of pregnancy trigger underlying atopic reaction	Elevated Immunoglobulin E	Topical or oral corticosteroids Antihistamines

BID-TID, twice to three times a day; DEJ, dermo-epidermal junction; IF, immunofluorescence; LFT, liver function tests.

Table 4. Acceptable and contraindicated anti-pruritic medications in pregnancy (31)

Acceptable anti-pruritic medications during pregnancy (Classes A and B)	Contraindicated anti-pruritic medications during pregnancy (Classes C, D and X)
Loratidine Second-generation antihistamine, FDA class B Drug of choice for embryonic, fetal, perinatal periods Safest antihistamine in lactation period	Hydroxyzine First-generation antihistamine, FDA class C May be associated with increased risk of congenital malformations May induce infant withdrawal in lactation period
Cetirizine Second-generation antihistamine, FDA class B Drug of second choice in embryonic, fetal, perinatal periods Moderately safe in lactation period	Fexofenadine Second-generation antihistamine, FDA class C Adverse fetal effects in animals Moderately safe in lactation period
Diphenhydramine First-generation antihistamine, FDA class B Drug of third choice in embryonic and fetal periods May cause uterine contractions with third trimester exposure Moderately safe in lactation period	Doxepin TCA, FDA Class B topically, FDA Class C orally May cause hypotonia, emesis, weak suck with third trimester exposure May cause dangerous respiratory depression in lacation period

FDA, Food and Drug Administration; TCA, tricyclic antidepressant.

cannot be differentiated from polymorphic eruption of pregnancy clinically or histopathologically (23). The later bullous phase is characterized by tense bullae similar to bullous pemphigoid, but without mucosal involvement (10). The lesions tend to wax and wane during pregnancy, with a flare at delivery in 75% of patients (23).

The rash of pemphigoid gestationis stems from circulating immunoglobulin G antibodies that bind to bullous pemphigoid antigen 2 (BP-180) in the hemidesmosomes of the dermo-epidermal junction, with subsequent damage to the membrane and tense bullae production (23). Diagnosis is made with histopathology and direct immunofluorescence of perilesional skin. The condition is self-limited and tends to resolve within weeks to months of delivery. Although there is no impact on fetal or maternal mortality, pemphigoid gestationis has been associated with a higher risk of premature and small-for-gestational-age babies.

Intrahepatic cholestasis of pregnancy

Intrahepatic cholestasis of pregnancy (ICP) is a condition of late pregnancy characterized by intense pruritus and hormonally triggered cholestasis. This is a unique pruritic condition of pregnancy in the sense that it is not associated with any primary skin lesions; instead, patients present with excoriations and prurigo nodules secondary to scratching and rubbing. These lesions tend to appear on the extensor surfaces of the arms and legs. Patients will describe a sudden-onset pruritus that starts in the palmoplantar regions and quickly become generalized to the entire body. Jaundice only occurs in severe forms (approximately 10% of cases).

This condition is characterized by an inability to effectively excrete bile salts, which leads to elevated serum bile acid levels that cause pruritus in the mother and deleterious effects in the fetus. The condition is relatively harmless to mothers, but can result in an increased risk of prematurity, intrapartal fetal distress, and stillbirths; as such, prompt diagnosis and treatment is necessary. ICP was recently found to be associated with highly elevated levels of serum autotaxin (24), an enzyme that converts lysophosphatidylcholine into lysophosphatidic acid as the active compound. Lysophosphatidic acid is a very potent signaling lipid that can activate nerve cells to transmit itch (25).

ICP is diagnosed by clinical history and a rise of serum bile acid levels over 11.0 μmol/L. Liver function tests may be normal or elevated, and hyperbilirubinemia only occurs in approximately 10–20% of patients. This condition is treated with ursodeoxycholic acid, which reduces serum bile acid levels to reduce maternal pruritus and fetal prognosis. Close follow-up with early delivery after complete lung maturity is recommended.

Atopic eruption of pregnancy

Atopic eruption of pregnancy is a new classification that combines pruritic conditions previously known as atopic dermatitis, prurigo of pregnancy, and pruritic folliculitis of pregnancy. This new umbrella condition is a benign pruritic disorder that serves as a diagnosis of exclusion in patients with atopy. It accounts for half of patients with a dermatosis of pregnancy and tends to recur in subsequent pregnancies. Only 20% of cases have a preexisting atopic dermatitis; 80% are experiencing eczematous changes for the first time (or have a distant childhood eczema history). Unlike other dermatoses of pregnancy, atopic eruption tends to occur early, and most present with widespread pruritic, eczematous lesions generalized to the face, neck, and flexural surfaces of the extremities. A third of patients will alternatively present with papular lesions disseminated on the trunk and limbs, usually with prurigo nodules on the extremities.

Diagnosis is clinical, and elevated immunoglobulin E levels can be seen in approximately half of patients. Fetal prognosis is unaffected, and mothers respond well to topical corticosteroids. In severe cases, systemic corticosteroids, antihistamines, and ultraviolet B phototherapy may be used.

Postmenopausal vulvar pruritus

In menopause, a drop in systemic estrogen and a rising vaginal pH essentially returns the vulva to a premenarcheal stage. However, changes in epidermal thickness, skin moisture, collagen content, wound healing, capillary strength, and vulvovaginal microflora distinguish the two phases from each other pathophysiologically. Common causes of vulvar itch in the postmenopausal age group are atrophic vulvovaginitis, lichen sclerosus, SCC of the vulva, and irritant dermatitis.

Atrophic vulvovaginitis

Atrophic vulvovaginitis affects a majority of postmenopausal women and tends to worsen with time (26). Although most women experience mild genital changes, up to half of patients complain of

at least one debilitating symptom such as vulvovaginal itching, dryness, dyspareunia abnormal discharge, and recurrent urinary tract infections (26). The thin, atrophic tissue of the postmenopausal vulva is easily irritated and susceptible to secondary infection (27). This condition presents with pale, thin vulvovaginal epithelium lacking rugae; petechiae may be present (27,28). While diagnosis is often clear based on clinical appearance, a swab or biopsy may be indicated to rule out other diagnoses. The treatment of atrophic vulvovaginitis hinges on topical estrogen therapy (rings, creams, or pessaries), which reverses atrophy to premenopausal levels within 1–2 weeks (27). Systemic low-dose estradiol therapy may also be initiated, with the understanding that long-term hormone replacement therapy may increase breast cancer risk (28). Superinfections should be treated and bland soaps and lubricants should be adopted to prevent further irritation.

Lichen sclerosus

Similar to LS of childhood, adult LS is a remitting and relapsing mucocutaneous disease of primarily women that presents with intense vulvar itch and scarring. This condition affects all ages and races, but occurs with a bimodal peak in prepubertal and peri/postmenopausal Caucasian women (13). Recent investigations have identified a more frequent incidence of autoimmune disorders in patients with LS compared with controls (29). Patients present with white, polygonal, atrophic papules and plaques on the vulvar and perineal regions. Advanced disease often manifests as a "figure-of-eight" sclerotic lesion around the introitus and anus, sparing the vaginal mucosa (FIG. 7A). Vulvar architecture is often altered or lost, with flattening of the folds of the labia majora and minora and fibrotic binding of the clitoral hood. Petechiae, purpura, and fissures can occur with scratching (FIG. 7B). Extragenital lesions of the neck, shoulders, inner thigh, and beneath the breast occur in approximately 11% of patients (13). Patients with this condition are at increased risk for vulvar carcinoma. LS is treated with high-potency topical steroids to manage symptoms and prevent scarring, which should theoretically reduce the risk of cancer. Biopsies should be performed of thickened areas, as older patients may not be aware of asymptomatic changes. Close follow-up and counseling are recommended, as the disease can be quite disfiguring and emotionally challenging.

Irritant contact dermatitis

Although allergic contact dermatitis is much less common in the postmenopausal group, irritant

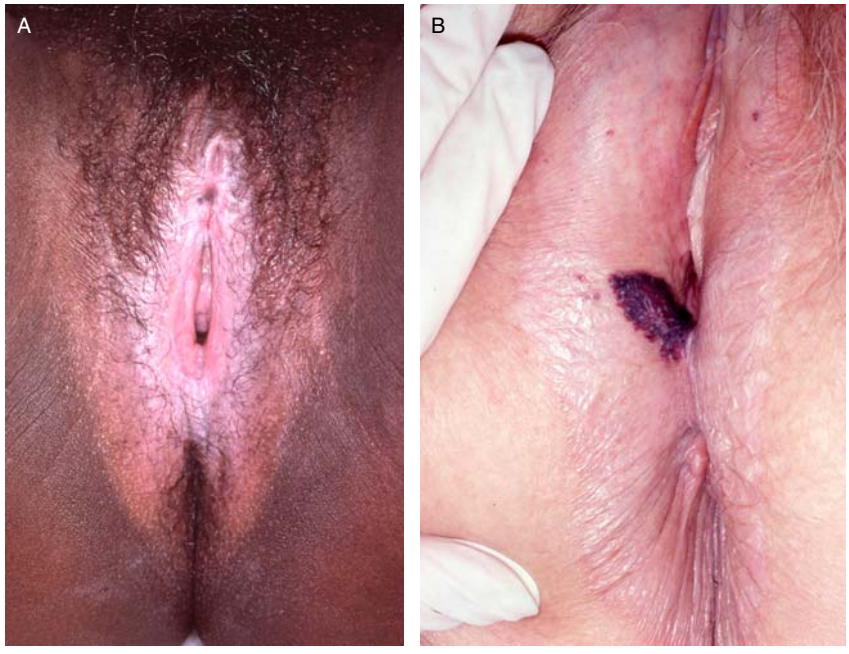

FIG. 7. Lichen sclerosus often forms a "figure 8" pattern of vulvar and perianal involvement (A and B). The black patient was refereed for a "pigmented lesion," but this is merely bleeding under atrophic skin. Figure 7B clearly shows marked sclerotic loss of vulvar architecture with narrowing of the introitus consistent with lichen sclerosus as well as purpura (Fig. 7B). ***Figure courtesy of Marilynne McKay, MD.***

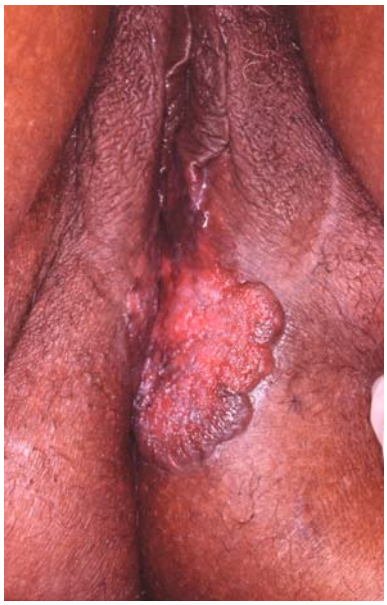

FIG. 8. A plaque of squamous cell carcinoma (vulvar intraepithelial neoplasia) in an elderly patient. ***Figure courtesy of Marilynne McKay, MD.***

dermatitis may be seen secondary to increased sensitivity of atrophic skin, especially with fecal and/or urinary incontinence. While postmenopausal pH is already high, urinary incontinence causes an even more potent alkaline environment, which leads to activation of the PAR2 receptor with subsequent increased itching (12). Examination often reveals redness in the area of contact, but may also include edema, scaling, and erosions (28). With chronic scratching, secondary LSC may occur. To treat irritant contact dermatitis, offending agents must be identified and avoided. Low- to medium-potency topical steroids may be used short-term, and superinfections should be treated appropriately (28). Protective barrier ointments help to protect skin from irritants.

SCC

SCC should always be considered as a potential diagnosis in postmenopausal women presenting with vulvar symptoms. Common presentations include bleeding, nonhealing ulcers, persistent plaques, lumps, or pain and pruritus that does not respond to treatment (28) (FIG. 8). Any suspicion of SCC should prompt immediate lesional biopsy.

Conclusion

Pruritic conditions in women highly impact quality of life and early diagnosis and treatment can greatly improve morbidity. However, vulvar dermatoses are still underdiagnosed and undertreated.

Acknowledgements

The authors would like to thank Dr. Marilynne McKay for providing important editorial suggestions and high quality clinical photographs.

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