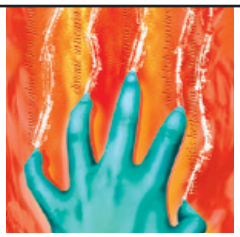
Vulval itch



BACKGROUND Vulval itch is common. Patients presenting with this symptom can have a long history involving visits to several general practitioners. Self diagnosis as thrush is common, and inappropriate use of over-the-counter antifungal preparations can lead to further irritation and distress. Excoriation, rubbing, maceration, secondary infection and the effects of topical applications frequently complicate matters.

OBJECTIVE This article identifies the common causes of vulval itch in adults and children, and highlights key features of the diagnosis and management of these conditions. Vulval pain syndromes are beyond the scope of this article and are therefore not discussed.

DISCUSSION The cause of vulval itch can often be multifactorial, but with careful assessment, a primary diagnosis can be reached in most cases. A good history requires patience, and gentle direct questioning, as patients often feel uncomfortable discussing their problems and may not disclose self applied remedies. Care should be taken during examination, as vulval rashes may be subtle. All postpubertal patients should have a low vaginal swab to diagnose candidiasis rather than treating empirically.

Vulval itch is common, and generally the differential diagnosis is fairly straightforward. In both children and adults, the most frequently seen vulval dermatoses are dermatitis, psoriasis and lichen sclerosus.[1] In both age groups, the majority of patients with dermatitis are atopic.[1,2] Streptococcal vulvovaginitis is seen only in the paediatric group, whereas chronic vulvovaginal candidiasis is an oestrogen dependent condition and is only seen postpuberty.[2]

Eczema and dermatitis

Eczema and dermatitis are synonymous terms. Whitening and thickening of the skin develops with chronic rubbing and scratching and is termed lichen simplex chronicus. Dermatitis is the most common cause of chronic vulval itch.[1] Major categories of dermatitis include atopic dermatitis, irritant contact dermatitis, allergic contact dermatitis, and seborrhoeic dermatitis. Multiple causative factors are often present in the one patient.

Table 1. Potential irritants to vulval skin

Chemical	Physical
Retained sweat	Sanitary pads,
Vaginal secretions	Tampon strings
Urine	Tight clothing,
Soaps, gels, bath oils, bubblebath	synthetic underwear
	Toilet paper
Disinfectants, tea tree oil	Overzealous
Douches, perfumes	cleansing, scrubbing
Lubricants, spermicides	Shaving, plucking hair
Medicaments, especially antifungal creams	Prolonged sitting
Depilatory creams	
Semen	

Belinda Welsh,
MBBS, MMed, FACD, is consultant dermatologist, St Vincent's Hospital, Melbourne, Skin and Cancer Foundation, Carlton, Dermatology/Vulval Conditions Clinic, Mercy Hospital for Women, East Melbourne, and is in private practice, Sunbury, Victoria.

Anne Howard,
MBBS, FRACP, FACD, is consultant dermatologist, Western Hospital, Footscray, Skin and Cancer Foundation, Carlton,Dermatology/Vulval Conditions Clinic, Mercy Hospital for Women, East Melbourne, and is in private practice, Footscray, Victoria.

Kathy Cook,
MBBS, FRACOG, is obstetrician and gynaecologist, Dermatology/Vulval Conditions Clinic, Mercy Hospital for Women, East Melbourne, Victoria.

Table 2. Potential allergens

Anaesthetics	Benzocaine (common), lignocaine (rare)
Antibacterial agents	Neomycin, chlorhexidine, tea tree oil
Preservatives	Ethylenediamine, parabens, propylene, glycol, chlorocresol, lanolin
Contraceptives	Condoms, spermicides, diaphragm
Clothing	Dyes
Cosmetics	Perfumes, deodorants
Sanitary products	Sanitary pads
Other medicaments	Antifungals
Semen	

Figure 1. Vulval dermatitis

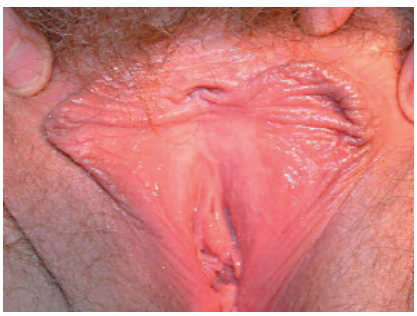


Figure 2. Vulval dermatitis with lichenification of the labia minora due to chronic rubbing

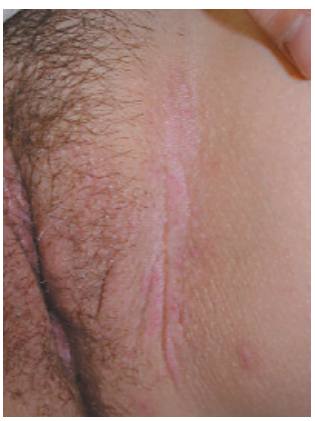


Figure 3. Dermatitis with prominent lichenification involving the groin crease

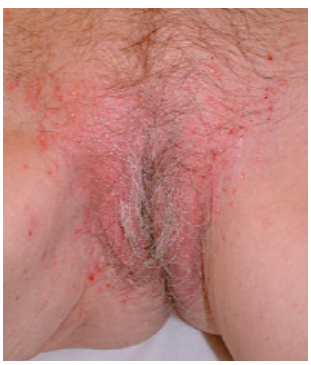


Figure 4. Psoriasis in an adult

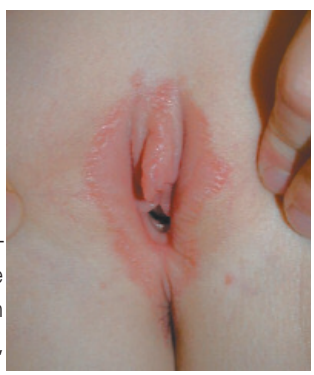


Figure 5. Psoriasis in a child

Aetiology

Irritant reactions are common on the vulva, and there may be a background of atopy (eczema, asthma, hayfever) (*Table 1*). In the early stages, candida is often diagnosed (by the patient, friends or a GP) and topical creams applied that are irritant in themselves, thereby compounding the problem. Urinary and faecal incontinence can worsen eczema, as can heat, friction and stress. Allergic reactions are less common, but need to be considered if there is poor response to treatment[3] (*Table 2*).

Clinical features

These conditions present similarly with itch being the predominate symptom. Burning will occur if the mucosal surface is involved and fissures can lead to pain and dyspareunia. Clinical signs need to be sought carefully as they may be subtle, such as interlabial erythema or small skin splits (*Figure 1*). Poorly defined erythema, with or without scale, may extend onto the pubis and thighs. Thickening and whitening of the skin (lichenification) (*Figure 2, 3*) with multiple small excoriations can be seen in more severe longstanding cases. Unlike lichen sclerosus, normal anatomy is preserved and the vagina is normal. Tinea should be considered in the differential diagnosis of dermatitis. Toenail and foot involvement is a helpful clue.

Management

Principles of management is that of most vulval diseases and includes:

- patient education and environmental modification (see *Patient education* page 517 this issue) – this is of vital importance as control rather than cure is usually the realistic aim. Washing without soap should be emphasised
- treatment of any secondary infection
- management of incontinence
- the use of bland, nonirritating moisturiser (eg. aqueous cream, petroleum jelly)
- if moderate to severe, a potent topical steroid such as methylprednisolone aceponate is recommended initially once per day (maximum of 1 month) until symptoms settle. Application frequency can be reduced (ie. every second day, 3 times per week) as symptoms improve. Follow with 1% hydrocortisone 1–2 times per day for maintenance if required, and
- cycling back to the more potent steroid may be necessary for flare-ups.

Psoriasis

Psoriasis is also common in the vulva (*Figure 4*) and

can be easily mistaken for dermatitis. It is generally not nearly as itchy as dermatitis, except in children (*Figure 5*). Diagnostic clues include a family history of psoriasis and evidence of psoriatic lesions elsewhere on the skin (postauricular erythema and scale, natal cleft, nail pitting). Clinically, psoriasis tends to be symmetrical, well defined and a beefy, red colour compared to dermatitis. The classic scale of psoriasis is absent. Psoriasis often requires more aggressive and prolonged treatment. Weaker potency steroids are usually insufficient to induce a response. Moderate to potent topical steroids such as methyl prednisolone aceponate 0.1% can be used once per day for 3–4 weeks followed by weak tar preparations such as 3% liquor picis carbonis (LPC) in aqueous cream, and/or 1% hydrocortisone for maintenance. Concurrent infection will worsen the psoriasis, therefore it is important this is identified and treated. Any condition not responsive to corticosteroids requires biopsy to exclude diseases such as extramammary Paget disease and Langerhan cell histiocytosis.

Lichen sclerosus

Lichen sclerosus (previously known as lichen sclerosus et atrophicus) is an uncommon inflammatory skin disease of unknown cause. It has a predilection for genital skin, and although seen in both sexes, is more commonly reported in women.[4] It can affect all age groups with reported onset from 6 months of age to late adulthood. Lichen sclerosus has been reported in association with a number of autoimmune conditions.[5] Patients diagnosed with lichen sclerosus, however, do not seem to be at continued excessive risk of developing autoimmune disease, so an exhaustive search for these diseases is not mandated.

Symptoms include pruritus, soreness, burning, dyspareunia, and – especially in children – dysuria, bleeding and chronic constipation.[6] It may occur anywhere over the vulval, perineal or perianal skin, and rarely at extragenital sites.[7] Typical features include well defined white plaques with an atrophic wrinkled surface. Telangiectasia, purpura, hyperpigmentation, erosions, fissures and oedema of the clitoral foreskin may also be seen (*Figure 6*). In longstanding disease, there may be an alteration of the normal anatomy of the vulva. The labia may shrink and the opening to the vagina may become narrowed, resulting in difficult and painful intercourse. The clitoris can become buried due to scarring of the clitoral hood and the labia minora may be lost. This diagnosis should be confirmed by skin biopsy. Although not itself considered a premalignant condition, longitudinal studies suggest a roughly 4% (reports range from 1–11%) lifetime risk of the development of squamous cell carcinoma within affected skin in patients with vulval lichen sclerosis.[8]

The aims of treatment are:

- control of symptoms such as itch, soreness, and dyspareunia
- minimisation of scarring and alteration of normal vulval architecture, and
- lifelong 12 monthly surveillance for squamous cell carcinoma of the vulva.

Potent topical steroids are the mainstay of treatment in adults and children and response is usually rapid (*Figure 7a, b*). Betamethasone diproprionate 0.05% ointment in optimised vehicle is used initially twice per day for 1 month, then daily for 2 months, gradually tapered to an 'as needs' basis, ideally only 1–2 times per week. If symptoms flare-up, candida and bacterial swabs should be performed to exclude co-existent pathology.

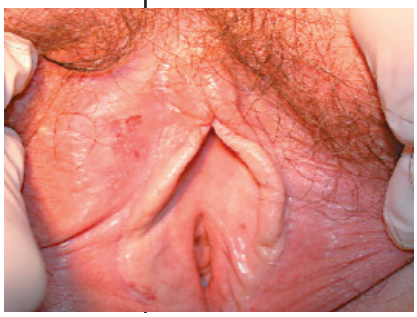


Figure 6. Lichen sclerosus with prominent purpura, whitening and resorption of the labia minora and burying of the clitoris

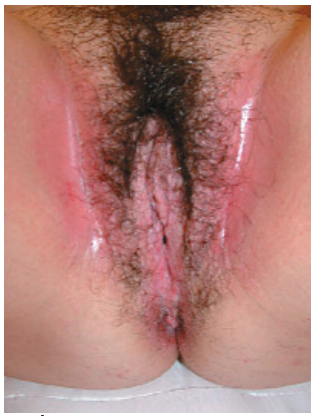


Figure 7a. Florid lichen sclerosus pretreatment

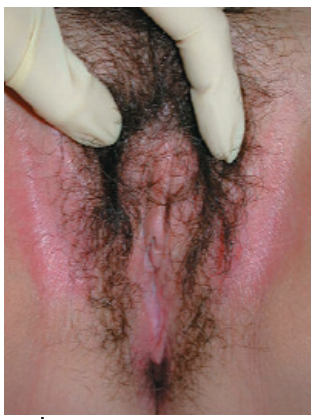


Figure 7b. Six weeks post-treatment

Vulvovaginal candidiasis

More than 50% of women over 25 years of age have had one episode of acute vulvovaginal candidiasis. It is generally easily recognised and treated. However, there is a small subset of women (less than 5%) who suffer repeated or intractable clinical candidiasis. Vulvovaginal candidiasis is considered recurrent when at least four discrete episodes occur in 1 year or at least three in 1 year that are not related to antibiotic therapy.[9]

The vast majority of these women are otherwise healthy. The pathophysiology of recurrent infection is unclear but appears to involve an abnormality in the host micro-organism relationship. These women often develop a florid dermatitic reaction to low levels of candida organisms and a hypersensitivity reaction to candida antigen has been postulated.[10,11]

The vast majority of cases are caused by *Candida albicans*. Other candida species may be responsible such as *C. glabrata*, *C. tropicalis*, and *C. parapsilosis*, and these may be relatively treatment resistant. For this reason, any fungi grown on culture should be charac

Table 3. Safety of treatments for candidiasis in pregnancy

Drug	Pregnancy category	Compatibility with breastfeeding
Oral		
Fluconazole	D	Not recommended
Ketoconazole	B3	Not recommended
Intravaginal		
Nystatin	A	Compatible
Miconazole	A	Compatible
Clotrimazole	A	Compatible
Econazole	A	Compatible
Boric acid	Not recommended	Not recommended

Category A
Drugs taken by a large number of pregnant women and women of childbearing age without any proven increase in the frequency of malformations or other direct or indirect harmful effects on the fetus having been observed

Category B3
Drugs taken by only a limited number of pregnant women and women of childbearing age without an increase in the frequency of malformation or other direct or indirect harmful effects on the human fetus having been observed. Studies in animals have shown evidence of an increased occurrence of fetal damage, the significance of which is considered uncertain in humans

Category D
Drugs that have caused, are suspected to have caused or may be expected to cause an increased incidence of human fetal malformations or irreversible damage. These drugs may also have adverse pharmacological effects. Accompanying texts should be consulted for further details

Table 4. Treatment options for recurrent vulvovaginal candidiasis

Agent	Dosing regimen options	Comment
Topical		
Nystatin pessaries	100 000 units alternate nights for 6 months	Messy, requires patient motivation
Clotrimazole	100 mg pessary intravaginally twice weekly or 500 mg pessary once weekly	Potential for irritation, but less costly
Oral		
Fluconazole	150 mg immediately then 50 mg weekly or 150 mg monthly in premenstrual week	This drug is used at our institution with success. It has a good safety profile and is well tolerated
Itraconazole	100 mg daily during the premenstrual week or 200 mg once per month or 400 mg once per month	Good safety profile, interacts with astemizole and terfenidine
Ketoconazole	200 mg daily for 3 months then 100 mg daily for 3 months or 100 mg daily for 6 months	Hepatotoxicity a risk, requires monthly monitoring of LFTs, interacts with astemizole and terfenidine

terised even if reported as 'nonpathogenic' by laboratory.

An environment rich in glycogen favours growth of candida. As oestrogen levels increase, so to do glycogen containing superficial cells in the vagina. Thus, the typical patient is premenopausal or taking hormone replacement therapy. It is characteristic for these symptoms to flare-up in the premenstrual week and improve with the onset of menstruation.[12] It is common to elicit a history of repeated episodes of vulvovaginal candidiasis (often this diagnosis is presumptive) and exacerbations with oral antibiotics. Temporary relief is gained from topical or short term oral antifungals. A secondary irritant contact dermatitis from topical imidazoles is very common. Vulval itch is the most frequent symptom, but soreness, splitting (particularly at the posterior forchette) and burning after intercourse is also common. Clinical appearance is often not helpful in making the diagnosis. Varying degrees of vulvar erythema and swelling is often seen, but vaginal discharge is rare.[13]

Diagnosis rests on a history of itch and burning with the key feature being premenstrual exacerbation and repeated positive low vaginal swabs (performed a week after ceasing topical antifungals) and/or fungal hyphae present on biopsy.

Optimal treatment for recurrent vulvovaginal candidiasis has not yet been defined. The realistic aim is control rather than cure with an understanding that long term, low dose maintenance is usually required. There are numerous regimens cited in the literature, but overall treatment needs to be individualised based on convenience, cost and potential side effects. Topical agents need to be used during pregnancy and lactation (*Table 3*). *Table 4* gives an example of treatment options. Compliance tends to be better with oral therapy and avoids the development of irritation with the topical treatments. We get good responses to 50 mg per week of fluconazole. Treatment is continued for 6 months then slowly tapered but this may vary depending on patient response. If there is a significant dermatitic reaction, 1% hydrocortisone ointment is useful at least in the early stages. Boric acid 600 mg (extemporaneously prepared in a gelatin capsule) intravaginally daily for 10–14 days can be used to treat infection with non-*C. albicans* species. Treatment strategies for which there is little or no evidence include a role for dietary modification, elimination of candida from the gastrointestinal tract with oral nystatin, and treatment of male sexual partners.[14] Although progesterone only oral contraception is preferable, the combined oral contraceptive pill can continue to be used as long as the oestrogen dose is low (ie. 20–30 µg).

Vulval itch – prepubertal girls

Atopic and irritant dermatitis are the commonest causes of vulval itch in prepubertal girls, and often the two go together.[15] Irritant factors in this age group may include urine, soap and bubblebath, and creams. Psoriasis is a more common vulval presentation in children than in adults.[15] It presents as a well demarcated red patch usually involving the perianal area also. Lichen sclerosus is not uncommon in young girls. A white plaque in a 'figure of eight' is the classic presentation. Haemorrhage into the skin is quite frequent and may lead to the issue of sexual abuse, sometimes inappropriately. It is treated the same way as in adults, although it is not usually necessary to use such potent preparations as betamethasone dipropionate 0.05% in optimised vehicle. Methyl prednisolone aceponate fatty ointment is potent, has low irritancy and is a good alternative. As for adults, prolonged follow up is recommended.

In prepubertal girls, vulvovaginitis caused by streptococcus species is common, staphlococcus, haemophilus and shigella are rare. Streptococcal vulvovaginitis due to group A ß-haemolytic streptococcus is seen only in this age group and may present acutely with erythema, pain, swelling and discharge; but may also present with a subacute vulvitis and itch. Vaginal and perianal swabs demonstrate the organism. Treatment of streptococcal infection is with phenoxymethyl penicillin 10 mg/kg up to 500 mg orally every 6 hours for 7 days.[16] Vulvovaginal candidiasis is not found in prepubertal girls.

Conclusion

Vulval itch is a common and distressing problem. Presentation to a GP is often delayed due to embarrassment and the condition is often complicated by self application of home remedies which may have worsened the situation. There are many similarities between the adult and paediatric age group when considering the dermatoses (dermatitis, lichen sclerosus and psoriasis), however, it is important to remember streptococcal vulvovaginitis is seen only prepuberty, and candidiasis seen postpuberty. Swabs should always be performed as often conditions may co-exist and all patients should be instructed about the importance of avoidance of irritants as part of an overall management plan.

Summary of important points

- The most frequently seen vulval dermatoses are dermatitis, psoriasis and lichen sclerosus.
- Streptococcal vulvovaginitis is seen only prepuberty and candidiasis postpuberty.
- Clinical appearance is often not helpful in making the diagnosis of candidiasis.
- Candidiasis causes itch and burning, typically worse premenstrually.
- All postpubertal patients should have a low vaginal swab to diagnose candidiasis rather than treating empirically.
- A secondary irritant contact dermatitis from topical imidazoles is very common.

Conflict of interest: none declared.

References

1. Fischer,GO. The commonest causes of symptomatic vulvar disease: a dermatologists perspective. Australas J Dermatol 1996;37:12–18.
2. Fischer GO, Rogers M. Vulvar disease in children. A clinical audit of 130 cases. Pediatr Dermatol 2000;17:1–6.
3. Sonnex C. Genital allergy. Sex Transm Infect 2004;80:4–8.
4. Wallace HJ. Lichen sclerosus et atrophicus. Trans St Johns Hosp Derm Soc 1971;57:9–30.
5. Thomas RHM, Ridley CM, McGibbon DH, et al. Lichen sclerosus et atrophicus and autoimmunity: a study of 350 women. Br J Dermatol 1988;118:41–46.
6. Fischer GO. Vulval disease in prepubertal girls. Australas J Dermatol 2001;42:225–236.
7. Ball SB, Wojnarowska F. Vulvar dermatoses: lichen sclerosus, lichen planus and vulval dermatitis/lichen simplex chronicus. Semin Cut Med Surg 1998;17:183–188.
8. Hart WR. Relation of lichen sclerosus of the vulva to the development of carcinoma. Obstet Gynecol 1975;45:369–377.
9. Ringdhal EN. Treatment of recurrent vulvovaginal candidiasis. Am Fam Physician 2000;61:3306–3312.
10. Fidel PL Jr, Sobel JD. Immunopathogenesis of recurrent vulvovaginal candidiasis. Clin Microbiol Rev 1996;9:335–348.
11. Ferrer J. Vaginal candidosis: epidemiological and etiological factors. Int J Gynaecol Obstet 2000;71(Suppl 1):S21–S27.
12. Mardh P-A, Rodrigues AG, Genc M, et al. Facts and myths on recurrent vulvovaginal candidosis: a review on epidemiology, clinical manifestations, diagnosis, pathogenesis and therapy. Int J STD AIDS 2002;13:522–539.
13. Sobel JD. Vulvovaginitis. Dermatol Clin 1992;10:339–359.
14. Reed B. Risk factors for candida vulvovaginitis. Obstet Gynecol Surv 1992;47:551–560.
15. Fisher GO. Vulval disease in prepubertal girls. Austral J Dermatol 2001;42:225–236.
16. Therapeutic Guidelines: Antibiotic, Version 12. Melbourne: Therapeutic Guidelines, 2003.

Email: bwrk@optusnet.com.au