

CASE SERIES

Chronic vulvitis in pre-pubertal girls

Gayle Fischer

Department of Dermatology, Royal North Shore Hospital and University of Sydney, Sydney, New South Wales, Australia

ABSTRACT

Pre-pubertal girls with inflammatory chronic vulval disease excluding lichen sclerosus are often described as having ‘non-specific vulvovaginitis’. The aim of this retrospective case series was to determine the aetiology of chronic vulvovaginitis in pre-pubertal (Tanner Stage 1) girls, with particular reference to candidiasis. A chart review recorded and compared the characteristics of 38 girls and 68 post-menarchal adolescents and pre-menopausal women with chronic vulvitis. Nineteen (50%) of the pre-pubertal children had been previously diagnosed with candidiasis and 21 (55%) had been treated unsuccessfully with topical antifungal agents. *Candida albicans* was isolated in two (5%) of the children and 37 (54%) of the adults ($P < 0.001$). A positive *Candida* culture was causally associated with chronic vulvovaginitis in 50% of the adults but in none of the children ($P < 0.001$). In 28 (74%) of the children and 28 (41%) of the adults, no pathogens were isolated on microbiological testing. General skin examination of the girls revealed signs of psoriasis in 27 (71%) and atopic dermatitis in nine (24%). Symptoms were controlled with topical anti-inflammatory treatment and environmental modification, including cessation of topical antifungals. Pre-pubertal girls with chronic vulvitis are likely to have either psoriasis or atopic dermatitis. Chronic vulvovaginal candidiasis is not seen in Tanner Stage 1 girls.

Key words: *Candida*, child, paediatric, vulva, vulvovaginitis.

setting in which a patient presents with a history of pruritus vulvae, a clinical finding of vulval inflammation evidenced by erythema and a non-diagnostic biopsy. The commonest causes of this scenario in adults are eczema, psoriasis and chronic vulvovaginal candidiasis (CVVC).¹

Little has been written on the subject of chronic vulvovaginitis in children. Despite this, a recent review article described vulvovaginitis as ‘one of the most common gynaecological complaints in pre-pubertal girls’.² This article and others use the term ‘non-specific vulvitis’.^{2–4} Explanations offered include anatomical proximity to the rectum, lack of labial fat pads and pubic hair, small labia minora, thin, ‘delicate’ vulval skin, lack of oestrogen, poor hygiene, ‘vulnerability to irritants’ in the pre-pubertal age group and even self-exploration as possible aetiological factors.

Vulvovaginal infections seen in pre-pubertal girls are frequently caused by group A β -haemolytic streptococcus⁵ and occasionally by other bacteria such as *Haemophilus influenzae* and *Shigella*.^{6–8} Controversy exists regarding *Candida albicans* as a cause of pre-pubertal vulvovaginitis.

In the author’s experience, pre-pubertal girls referred with vulvitis or vulvovaginitis have often been previously treated with topical antifungal agents and a provisional diagnosis of ‘thrush’ has been made before specialist consultation.

This retrospective chart review documents the clinical features, previous diagnosis and treatment, and results of microbiological cultures in pre-pubertal Tanner Stage 1 girls presenting with ‘non-specific’ inflammatory chronic vulvitis or vulvovaginitis, and compares them with a group of post-menarchal adolescents and pre-menopausal women with the same clinical presentation.

The primary aims of the study were to determine whether pre-pubertal girls with chronic ‘non-specific vulvovaginitis’ might have a specific endogenous dermatological aetiology and whether *C. albicans* was a cause of chronic vulvovaginitis before puberty.

INTRODUCTION

The descriptive term ‘non-specific’ is commonly encountered in articles on vulval disease. It refers to a clinical

Abbreviations:

CVVC	chronic vulvovaginal candidiasis
LPC	liquor picis carbonis
LS	lichen sclerosus
TCS	topical corticosteroid

Correspondence: Dr Gayle Fischer, PO Box 440 Killara, New South Wales, 2071, Australia. Email: gayle.fischer@sydney.edu.au
Gayle Fischer, FACD.
Submitted 5 January 2010; accepted 4 February 2010.

MATERIALS AND METHODS

The computerized database (Genie Solutions) of the private practice of the author was searched for patients seen between January 2008 and December 2009. The charts of girls with a current problem of 'Pre-pubertal vulvitis' were reviewed. A control group of post-menarchal, pre-menopausal women who presented with an erythematous vulvitis or vulvovaginitis was drawn from consecutive cases seen between January and June 2009, allowing time for adequate follow up to determine response to therapy, which in many cases was prolonged.

The study was submitted to the Human Research Ethics Committee of Northern Sydney Central Coast Health and exempted from full review as data collected was de-identified.

Girls were included in the study if they were Tanner Stage 1 (pre-pubertal), presenting with an erythematous vulvitis or vulvovaginitis and otherwise healthy. Girls were excluded from the study if they had a specific vulval condition such as lichen sclerosus (LS), were immunosuppressed, diabetic or insulin resistant.

Data about the pre-pubertal patients were entered in an Excel database documenting: age; duration of disease; symptoms including itch, pain, discharge, night waking, dysuria; previous diagnosis; previous treatment including use of antifungal agents; exposure to irritants including night nappies; personal and/or family history of atopy and psoriasis; genital and general skin examination; result of microbiological testing; diagnosis; treatment, and response to treatment either at follow-up appointment or by subsequent communication by phone or email.

Post-menarchal, pre-menopausal patients who presented with an erythematous vulvitis which had been investigated with microbiological testing were entered in the control group. Patients were excluded from the study if they had a specific vulvovaginal condition such as LS, an erosive vulvovaginal condition, a vaginitis without evidence of vulval involvement, were diabetic, insulin resistant, immunosuppressed or post-menopausal. Patients on the oral contraceptive pill were included in the study. Data about these patients were entered in an Excel database recording age, diagnosis, result of microbiological testing, treatment, and response to treatment.

Microbiological testing in the pre-pubertal group was carried out using a moistened cotton swab to sample secretions from the vaginal introitus. In the post-menarchal group, a low vaginal swab was carried out.

Statistical analysis was carried out using Fisher's exact test (GraphPad Software 2009).

RESULTS

Thirty-eight girls and 68 women met the inclusion criteria.

The mean age of the girls was 5.8 years (range 2–12 years). All were pre-pubertal (Tanner Stage 1).

The mean duration of disease in the girls was 2 years (range 3 months to 6 years).

The mean age of the women was 32.7 years (range 15–56 years).

Patients were classified as having psoriasis as a probable aetiology of their vulvitis if they had obvious vulval psoriatic plaques, a chronic or recurrent episodic vulvitis with other cutaneous signs of psoriasis (such as knee and elbow plaques, scalp scale or typical nail changes) or a family history of psoriasis in a first-degree relative. A total of 27 (71%) of the girls met these criteria. Of this group, 18 (66%) had family history of psoriasis in a first-degree relative. All of these patients were treated with and responded to a combination of topical corticosteroid (TCS) and 2% liquor picis carbonis (LPC) in aqueous cream. The latter was tolerated by the majority of children, particularly if the inflammatory phase was suppressed initially with TCS alone. In most cases, 1% hydrocortisone ointment was adequate to suppress inflammation, but four girls required initial treatment with a stronger TCS, methylprednisolone aceponate 0.1% fatty ointment.

Patients were classified as having atopic dermatitis as a probable cause of their vulvitis if they had a chronic or episodic vulvitis and had other evidence of atopy: asthma, hay fever or other cutaneous signs of atopic dermatitis or a family history of atopy in a first-degree relative. A total of nine (24%) of the girls met these criteria. Six of these girls (66%) had a family history of atopy in a first-degree relative. All patients in this group were treated with and responded to topical 1% hydrocortisone ointment, emollient and environmental modification (Table 1).

Of the 38 girls, 28 (74%) had a negative swab. Group A *β*-haemolytic streptococcus was isolated from four patients, *Staphylococcus aureus* from one patient and *H. influenzae* from three patients. In only one case whose duration of disease, 4 months, was significantly less than the mean, was bacterial infection the primary disease. In the others, it occurred on a background of a dermatosis or in one case a foreign body. *C. albicans* was isolated from two patients, both of whom had psoriasis complicated by chronic maceration: one from nappies and the other a voiding problem.

Bacterial superinfection was treated with appropriate oral antibiotics. The two children with *Candida* were treated with topical antifungal medication. However, in all children with a dermatosis, anti-inflammatory treatment was also required to control symptoms.

Table 1 Environmental modification suggested in pre-pubertal girls with chronic vulvitis

Washing: soap substitute, bath rather than shower or supervise showering to make sure vulva is washed. Do not shampoo hair in the bath. No bubble bath
Wiping: wipe front to back, avoid perfumed toilet paper
Exogenous irritants: avoid antifungal creams, perfumed products including wet wipes or any other cream which causes stinging.
Attempt to stop night nappies
Clothes: avoid lycra ballet clothes, leotards, nylon stockings, underpants overnight. Wear only cotton underwear
Sport: avoid prolonged contact with chlorinated swimming pool water, wash after and do not go home in wet swimming costume. White soft paraffin may be applied before swimming
Constipation and voiding problems: make sure this is addressed by your local doctor

Table 2 Diagnosis and microbiology in pre-pubertal versus post-menarchal groups

	Pre-pubertal group (<i>n</i> = 38) Average age 5.8 years (range 2–12 years)	Post-pubertal group (<i>n</i> = 68) Average age 32.7 years (range 13–56 years)
Diagnosis (<i>n</i> , %)		
Chronic vulvovaginal candidiasis	0	42 (62)
Psoriasis	27 (71)	8 (12)
Dermatitis	9 (24)	14 (21)
Streptococcal vulvovaginitis as a primary diagnosis	1 (2.5)	0
Bacterial infection in addition to dermatosis	5 (13)	3 (4)
Foreign body	1 (2.5)	0
Oestrogen hypersensitivity vulvovaginitis	0	3 (4)
Desquamative inflammatory vulvovaginitis	0	3 (4)
Organisms isolated (<i>n</i> , %)		
<i>C. albicans</i> (causal)	0	34 (50)
No pathogen isolated	28 (74)	28 (41)
<i>C. albicans</i> (colonization)	2 (5)	3 (4)
<i>S. aureus</i>	1 (2.5)	2 (3)
Group A β -haemolytic streptococcus	4 (10)	0
<i>H. influenzae</i>	3 (7.5)	0
<i>Gardnerella</i>	0	1 (1.5)

All of the children presented with symptoms of itch and/or soreness. In addition to this, five had a discharge and three complained of dysuria. Fifteen children reported waking at night in distress.

All of the children had a vulvitis involving the labia majora and in 12 children this extended to the perianal skin. Of those with perianal involvement, 10 had psoriasis and two had dermatitis. Six girls, all with psoriasis, had extension of the rash into the natal cleft. Eight girls (21%) had an associated vaginitis: in six of these a bacterial pathogen (group A β -haemolytic streptococcus, four cases; *H. influenzae*, two cases) was isolated and in two (both of whom had a urinary voiding dysfunction resulting in maceration) no pathogen was isolated.

Data on the presence of irritants were incomplete but had been recorded in 17 of the children. They had historical evidence of irritant triggers: night nappies, swimming lessons in chlorinated pools, bubble baths, ballet lessons wearing occlusive Lycra leotards and dysfunctional voiding resulting in maceration, as well as prolonged use of anti-fungal creams. Faecal incontinence and poor hygiene were not encountered in any patient.

Previous treatment included topical antifungal cream in 21 (55%) patients. An empirical trial of mebendazole before consultation, without benefit, was recorded in seven children. It is probable that more of the children had previously been 'wormed' before presentation, but data were incomplete. Other treatments included oral antibiotics, TCS, xylocaine, triclosan, paw-paw ointment, emollient, topical oestrogen and topical tacrolimus.

A previous diagnosis of 'thrush' had been made in 19 (50%) patients. Seven (18%) had been previously diagnosed as having a urinary tract infection. In all these cases urine culture had been negative. A diagnosis of LS had been made in three patients. One patient had been correctly diagnosed

with psoriasis and one with dermatitis. No diagnosis had been made in five children.

Of the 68 women in the control group, 28 (41%) had a negative low vaginal swab. *C. albicans* was isolated in 37 patients (54%). In 34 (50%) of these patients this was clinically relevant and there was a response to anti-fungal treatment alone without the use of topical anti-inflammatory agents. *C. albicans* was the predominant isolate: in two patients, both of whom had dermatitis, *S. aureus* was cultured and was considered a secondary infection; in one patient with psoriasis, *Gardnerella* was cultured. This represented an intercurrent episode of bacterial vaginosis. In each case there was clinical improvement from appropriate antibiotic treatment; however, treatment of the underlying dermatosis was also required for complete relief of symptoms.

Of the control group, 42 (62%) patients were diagnosed as having CVVC and responded to antifungal treatment. Fourteen (21%) patients had a vulval dermatitis and responded to TCS and environmental modification. Eight (12%) patients had psoriasis and responded to TCS, 2% LPC and in some cases topical calcipotriol treatment. One patient had desquamative inflammatory vulvovaginitis⁹ and responded to intravaginal clindamycin, and three patients had an oestrogen hypersensitivity vulvitis¹⁰ and responded to cycle suppression with cyproterone acetate.

A comparison of diagnosis and microbiology results in the pre-pubertal and post-menarchal groups is summarized in Table 2. The relative frequency of positive *C. albicans* swabs and clinical candidiasis responsive to antifungal treatment in the two groups reached statistical significance ($P < 0.001$).

It should be noted that it took 24 months to collect 38 girls who met inclusion criteria for the study cohort and 6 months to collect 68 women who met criteria for the control group.

DISCUSSION

Vulvovaginal candidiasis is a common problem in adults. Point prevalence studies show that approximately 20% of asymptomatic healthy women carry *Candida* in the vagina and it is considered a commensal of the genital and gastrointestinal tracts.¹¹ In adults and children, *Candida* may behave as a pathogen or commensal in the genital tract and positive swabs must be interpreted in light of the clinical symptoms and signs.

In adults, symptomatic vulvovaginal candidiasis occurs in three clinical settings: episodic acute attacks, recurrent episodic attacks and chronic disease. It has been postulated that the latter situation is not an infection but an abnormal inflammatory response to a commensal organism;¹² however, no formal definition of this condition has been proposed. It is important to appreciate that it is chronic, not acute or recurrent, vulvovaginal candidiasis (CVVC) which comes into the differential diagnosis of chronic vulvitis.

Physical examination of adults with CVVC typically reveals erythema of the labia minora, vagina and perineum. The main differential diagnoses include psoriasis, dermatitis (atopic, allergic, seborrhoeic or irritant) and less often extramammary Paget's disease, oestrogen hypersensitivity vulvitis¹⁰ and desquamative inflammatory vulvovaginitis.⁹ All of these conditions can appear similar, presenting with scaling and erythema of the external vulva with or without vaginitis, hence the term 'non-specific vulvovaginitis'. The usefulness of this term has been queried,¹⁵ as it suggests a lack of aetiology and might suggest a 'one-size fits all' approach to management.

Despite the so-called 'non-specific' appearance, all of these diagnoses require specific treatment based on diagnosis (Table 3). Vulvovaginal candidiasis is an oestrogen-dependent condition¹¹ and therefore is not expected before puberty or after menopause in women who are not taking

oestrogen replacement therapy. A study which found that children suffer from *Candida* vulvovaginitis included post-menarchal adolescents in the cohort.¹⁴ However, more recent studies that have specifically studied pre-pubertal children (as opposed to cohorts that include adolescents) have confirmed that *C. albicans* is rarely found in vaginal isolates or in this age group unless they have taken oral antibiotics, are diabetic or immunosuppressed.^{3,7,15}

Despite this, 50% of the pre-pubertal girls presenting with chronic vulvitis in this study had been diagnosed as having 'thrush' and 55% had been treated, unsuccessfully, with topical antifungal agents, for periods of up to 5 years. The prolonged use of antifungal agents had in some cases acted as an irritant that had exacerbated the underlying dermatosis.

The commonest causes of chronic symptomatic vulvitis in children are dermatitis, psoriasis and LS.¹⁶ Of these three conditions, only LS is immediately diagnosable because of its characteristic morphological features and diagnostic histopathology. As in adults, the other two conditions, dermatitis and psoriasis, may appear 'non-specific'. Differentiating these conditions from the other may be difficult and involves a value judgment by the clinician which relies more on symptoms, clinical signs elsewhere on the skin, personal and family history and microbiological testing than on the appearance of the vulva. This is not unreasonable. Neither psoriasis nor atopic dermatitis has accepted diagnostic criteria and dermatologists usually make a clinical diagnosis when these conditions occur on skin other than the vulva.

A significant confounding factor in making a specific diagnosis in a child relates to the difficulty not only of recognizing dermatological signs on the rest of the skin, but of visualizing the vagina and performing diagnostic tests. CVVC in adults is a vulvovaginitis, and inflammation of the vagina can be confirmed on speculum examination. This

Table 3 Causes of chronic erythematous vulvitis in adults and children

	Vagina involved	Organism causally associated	Seen in children	Treatment
Dermatitis (atopic, irritant, allergic)	No	Nil	Yes	TCS Environmental modification
Dermatitis: seborrhoeic	No	Nil	Yes (infants)	TCS Environmental modification
Psoriasis	No	Nil	Yes	TCS 2% liquor picis carbonis Calcipotriol
Chronic vulvovaginal candidiasis	Yes	<i>C. albicans</i>	No	Oral and topical antifungals
Intravaginal foreign body	Yes	Nil	Yes	Lavage Oral antibiotic for superinfection
Oestrogen hypersensitivity vulvitis	Yes	Nil ¹⁰	No	Cycle suppression
Extramammary Paget's disease	No	Nil	No	Surgical Topical imiquimod
Desquamative inflammatory vulvovaginitis	Yes	Nil or group B streptococcus ⁹	No	Intravaginal clindamycin
Bacterial vulvovaginitis [†]	Yes	Group A β -haemolytic streptococcus <i>H. influenzae</i>	Yes	Oral antibiotic

[†]Unusual as a chronic condition. May represent superinfection of a dermatosis. TCS, topical corticosteroid.

is not possible in young children in whom vaginitis must be inferred clinically by the presence of discharge and erythema of the introitus. When it comes to diagnosing paediatric psoriasis, the signs are subtle and often different from those found in adults.¹⁷ Furthermore, when other cutaneous signs as well as family history suggest that a chronic vulval skin condition in a child could be psoriasis or dermatitis, the possibility of confirming this by biopsy is difficult to justify, particularly as it might be non-diagnostic. Vulval conditions in adults which one would not hesitate to biopsy, such as LS, are usually diagnosed clinically in children.

In the majority of the children, 74% of cases, swabs did not show any pathogens. In those with positive cultures and a concurrent dermatosis, treatment with oral antibiotics or antifungals alone did not resolve symptoms and anti-inflammatory agents were also required.

It is interesting that many girls with vulvitis wake at night in distress. General practitioners and pharmacists are aware of the connection between night waking and enterobius infestation, and as a result most children presenting to a dermatologist with a long history of vulvitis have been treated for this already. Previous treatment with mebendazole was recorded in seven children but had probably been given to many more. In this series, none of the children who had been given mebendazole had responded to this treatment.

Although many children were engaging in activities that aggravated their vulvitis, it is unlikely that the frequency of these activities would be different from any healthy cohort of Australian primary school children without vulvitis, and more likely that the susceptibility to irritancy from these activities related to the presence of an underlying dermatosis. Poor hygiene and incontinence of faeces, both commonly implicated as causes of paediatric vulval disease, were not found to be aetiological factors in any of the children in this study. Most parents had been correctly advised with regard to good hygiene practices and irritant minimization before referral.

In children, 27 (71%) had cutaneous signs of psoriasis and nine (24%) had current or present atopic dermatitis. In adults, psoriasis, found in eight (12%) and dermatitis, found in 14 (21%) were important but less common causes of vulvitis. It is interesting that dermatitis was commoner in adults relative to psoriasis, the reverse of the situation in children. This possibly relates to the greater range of vulval irritants to which adult women are exposed.

By contrast to children, 42 (62%) of adult patients presenting with an erythematous vulvitis had an associated vaginitis, 34 (81%) of whom had a positive vaginal swab for *C. albicans*. These patients were diagnosed with CVVC and responded to antifungal treatment without the use of anti-inflammatory treatment. Eight patients with a history consistent with candidiasis had a negative swab but responded to antifungal treatment.

The most significant difference between the pre-pubertal and post-menarchal group was the complete lack of CVVC, an oestrogen-dependent condition, in the pre-pubertal group.

Two previous publications which have postulated an 'increased susceptibility to vulvovaginitis' in children have collected a relatively small cohort (44 and 80 cases) over long study periods (57 and 36 months respectively).^{3,4} It should be noted that in this study only 38 children were collected over a period of 24 months. This suggests that vulval disease in children is relatively uncommon.

The findings in this study are that chronic vulval disease is most often found in pre-pubertal children with common specific dermatoses rather than infective conditions, and that this is poorly recognized by referring general practitioners, who appear to be applying an adult frame of reference to diagnosis. There is a significant difference in the occurrence of *Candida* in the pre- and post-menarchal groups and true CVVC is not seen in the former. The assumption that pre-pubertal children have 'an increased susceptibility' to vulvovaginitis relative to adults is not evidenced by the present study, and in fact the susceptibility to *Candida* conferred by the adult oestrogenized vagina would appear to result in the reverse being true.

CONCLUSION

Vulvovaginitis is uncommon in pre-pubertal in comparison with post-menarchal females. Pre-pubertal girls presenting with a chronic inflammatory vulvitis, particularly when there is no associated vaginitis, are likely to have psoriasis or atopic dermatitis on other parts of their skin, suggesting that their vulvitis is part of a specific dermatosis. Once bacterial infection is ruled out, most will respond to topical anti-inflammatory agents. In children with a dermatosis, remission is maintained with TCS and environmental modification, including cessation of topical antifungal agents. Most children with psoriasis in this study benefited from the use of 2% LPC cream in addition to these measures. In children with infection, who usually have a vulvitis associated with vaginitis, group A β -haemolytic streptococcus and *H. influenzae* are the most likely aetiological agents. These children respond to appropriate antibiotics; however, if there is also a dermatosis present, this must be treated as well or symptoms will persist. Although CVVC was common in the adult control group and accounted for 62% of cases, it was not encountered in any of the children. Pre-pubertal children presenting with chronic vulval disease appear most often to have an endogenous dermatosis which involves other parts of their skin as well as their vulva, and which in many cases has been aggravated by exogenous irritants, including prolonged use of antifungal agents, but is not caused either by irritants or poor hygiene. CVVC does not occur in non-oestrogenized healthy children in this age group and antifungal agents are not indicated.

REFERENCES

1. Fischer GO, Rogers M. Vulval disease in children: a clinical audit of 130 cases. *Pediatr. Dermatol.* 2000; 17: 1-6.
2. Dei M, Di Maggio F, Di Paolo G *et al.* Vulvovaginitis in childhood. *Best Pract. Res. Clin. Obstet. Gynaecol.* 2009; Oct 31. [Epub ahead of print] doi: 10.1016/j.bpobgyn.2009.09.010.

3. Stricker T, Navratil F, Sennhauser FH. Vulvovaginitis in pre-pubertal girls. *Arch. Child Dis.* 2003; **88**: 324–6.
4. Paek SC, Merritt DF, Mallory SB. Pruritus vulvae in prepubertal girls. *J. Am. Acad. Dermatol.* 2001; **44**: 795–802.
5. Heymann W. Streptococcal vulvovaginitis. *J. Am. Acad. Dermatol.* 2009; **61**: 94–5.
6. Jasper JM, Ward MA. Shigella vulvovaginitis in a prepubertal child. *Pediatr. Emerg. Care* 2006; **22**: 585–6.
7. Cuadros J, Mazon A, Martinez R *et al.* The aetiology of paediatric inflammatory vulvovaginitis. *Eur. J. Pediatr.* 2004; **163**: 105–7.
8. Cox RA, Slack MP. Clinical and microbiological features of Haemophilus influenzae vulvovaginitis in young girls. *J. Clin. Pathol.* 2002; **55**: 961–4.
9. Newbern EC, Foxman B, Leaman D *et al.* Desquamative inflammatory vaginitis: an exploratory case-control study. *Ann. Epidemiol.* 2002; **5**: 546–52.
10. Fischer GO, Ayer B, Frankum B *et al.* Vulvitis attributed to estrogen hypersensitivity: report of 11 cases. *J. Reprod. Med.* 2000; **45**: 493–7.
11. Fidel PL, Cutright J, Steele C. Effects of reproductive hormones on experimental vaginal candidiasis. *Infect. Immun.* 2000; **68**: 651–7.
12. Fidel PL Jr, Barousse M, Espinosa T *et al.* A live intravaginal Candida challenge in humans leads to new hypotheses for the immunopathogenesis of vulvovaginal candidiasis. *Infect. Immun.* 2004; **72**: 2939–46.
13. Fischer GO, Margesson L. Vulvar itching in prepubertal girls: let's be specific (Letter). *J. Am. Acad. Dermatol.* 2003; **48**: 986–7.
14. Koumantakis EE, Hassan EA, Deigeoroglou EK *et al.* Vulvovaginitis during childhood and adolescence. *J. Pediatr. Adolesc. Gynecol.* 1997; **10**: 39–43.
15. Jones R. Childhood vulvovaginitis and vaginal discharge in general practice. *Fam. Pract.* 1996; **13**: 39–43.
16. Fischer GO. Vulvar disease in prepubertal girls. *Australas. J. Dermatol.* 2001; **42**: 225–34.
17. Morris A, Rogers M, Fischer G *et al.* Childhood psoriasis: a clinical review of 1262 cases. *Pediatr. Dermatol.* 2001; **18**: 188–98.