

Vulvovaginitis: clinical features, aetiology, and microbiology of the genital tract

A Jaquier, A Stylianopoulos, G Hogg, S Grover

Abstract

Aim—To clarify the contribution of clinical and environmental factors and infection to the aetiology of vulvovaginitis in premenarchal girls, and to determine clinical indicators of an infectious cause.

Design—It was necessary first to define normal vaginal flora. Cases were 50 premenarchal girls > 2 years old with symptoms of vulvovaginitis; 50 controls were recruited from girls in the same age group undergoing minor or elective surgery.

Results—Interview questionnaire showed no difference between cases and controls in regards to hygiene practices, exposure to specific irritants, or history of possible sexual abuse. Normal vaginal flora was similar to that described in previous studies, with the exception of organisms likely to be associated with sexual activity. 80% of cases had no evidence of an infectious cause. In the 10 cases in whom an infectious cause was found, there was significantly more visible discharge and distinct redness of the genital area on examination compared with other cases.

Conclusions—The findings suggest that vulvovaginitis in this age group is not usually infectious or necessarily related to poor hygiene, specific irritants or sexual abuse, although any of these can present with genital irritation. The possibility of sexual abuse should always be considered when a child presents with genital symptoms, but our data indicate it is not a common contributing factor. Infection is generally associated with vaginal discharge and moderate or severe inflammation.

(Arch Dis Child 1999;81:64-67)

Keywords: vulvovaginitis; premenarchal; vaginal flora

Vulvovaginitis is generally considered to be the most common gynaecological problem in premenarchal girls, although the actual incidence is unknown. Several factors are said to contribute to inflammation of the genital area. These include relatively less protective covering of the introitus by the labia majora, low oestrogen concentrations leaving the vaginal mucosa susceptible to irritation and infection, exposure to irritants (such as bubble bath), poor hygiene, and infection by specific pathogens.¹ Previous investigators have in particular emphasised the last two factors.^{2,3} However, it was the authors' impression that most girls presenting with vulvovaginitis had good

personal hygiene, and usually responded to simple symptomatic treatment rather than requiring antimicrobial agents. Our hypothesis was that most vulvovaginitis in this age group is not infectious in origin and that clinical indicators exist when symptoms are primarily caused by infection.

The aims of this study were therefore to clarify the contribution of clinical factors and infection in the aetiology of vulvovaginitis, and to determine indicators of an infectious cause. It was necessary first to define normal vaginal flora in premenarchal girls in our population.

Patients and methods

Cases were 50 premenarchal girls > 2 years old who presented to the emergency department, to general paediatric outpatients at the Royal Children's Hospital, Melbourne, or to a paediatric gynaecology clinic, with symptoms of redness, soreness or itching of the genital area, with or without genital bleeding. A group of 50 premenarchal controls > 2 years old was recruited from girls undergoing minor or elective surgery at the Royal Children's Hospital. Exclusion criteria were non-English speaking, absence of the child's mother (it was felt that the female parent should be involved in consenting to participate in this study), previously known history of sexual abuse, current antibiotics or treatment of vulvovaginitis, or where the child's condition or surgery were likely to affect vaginal flora. Children who were particularly distressed before surgery, or whose parents were distressed, were also excluded. Consent was obtained from parents and the child if she was old enough, according to the parents' wishes. A total of 69 families were approached; 17 declined involvement and two were excluded because the children were undergoing treatment for vulvovaginitis at the time.

From both groups, clinical data were obtained using a brief interview questionnaire (table 1). Examination was performed to assess hygiene, redness, and excoriation of the genital and anal areas, the presence of vaginal discharge, and any other relevant physical findings. Redness and excoriation were graded 0, 1 or 2+ for absent, mild or distinct findings. For microbiology, two or three low vaginal swabs were taken, according to the child's tolerance of the procedure, a urine sample was collected, and parents were requested to take a "Sellotape test" (for pinworm) in the early morning. Girls in the control group had swabs taken under anaesthetic before surgery, with nursing staff present.

A primary infective cause was considered likely in a symptomatic child if a pure or

Alice Springs Hospital,
Alice Springs, NT
0870, Australia
A Jaquier
A Stylianopoulos
G Hogg
S Grover

Correspondence to:
Dr Jaquier.

Accepted 15 February 1999

Table 1 Interview questionnaire

Date of birth			
Current history	Symptoms	Redness	Y/N
		Soreness	Y/N
		Itching	Y/N
		Discharge	Y/N
		Bleeding	Y/N
		Duration of symptoms, previous episodes	
	Past history	UTI	Y/N
Eneuresis		Y/N	
Constipation		Y/N	
Encopresis		Y/N	
Skin disorder		Y/N	
Other illness in last 4 weeks		Y/N	
Antibiotic use in last 4 weeks		Y/N	
Toileting	Toilet trained	Y/N	
	Wipe front to back	Y/N	
	Need assistance with wiping	Y/N	
Hygiene	Frequency of bathing (daily, 2–4 times/week, once weekly or less)		
	Underwear (cotton, synthetic, both)		
	Bubble bath in last month	Y/N	
Possible sexual abuse			
UTI, urinary tract infection.			

predominant growth of a probable pathogen was isolated on culture of a low vaginal swab or if pinworm ova were found following the “Sellotape test”.

MICROBIOLOGICAL METHODS

In both cases and controls, a film for Gram stain was prepared at the bedside and examined for bacteria, pus, epithelial cells, and clue cells. The first swab was placed in charcoal transport media (Interpath Services, Australia) and was immediately cultured onto a number of agar plates. Horse blood, MacConkey and Sabouroud’s agar plates were incubated aerobically at 37°C for 48 hours. Human blood agar with *Gardnerella vaginalis* supplement and gonococcal selective media plates were incubated at 36°C with 3.5% carbon dioxide. Ureaplasma modified A8 agar and a second horse blood agar plate were incubated anaerobically at 35°C. The second swab was inoculated into a *Trichomonas vaginalis* medium, which was then incubated at 35°C for 48 hours. A wet preparation was performed looking for *T vaginalis*. A cotton tipped wire swab was directly placed into chlamydia transport medium, dispensed in 0.5 ml vials, and frozen immediately at –70°C until polymerase chain reaction (PCR) was performed in one batch using standard methods.^{4,5}

Urine samples were examined by microscopy for white and red blood cells and bacteria according to standard laboratory methods. Protein and glucose were determined by Nephur Dipstick Test (Boehringer Mannheim, UK). Horse blood and cysteine lactose electrolyte deficient agar plates were incubated aerobically for 18–24 hours and isolates identified by standard procedures.

A “Sellotape test” was performed to detect the presence of *Enterobius vermicularis* (pinworm). A piece of Sellotape approximately 5 cm long was pressed onto the skin adjacent to the anus, then removed and stuck onto a slide,

which was examined by microscopy for ova. Parents were requested to collect the specimen as soon as the child woke in the morning.

STATISTICS

Clinical parameters and microbiological data from the two groups were compared using the χ^2 test.

Results

The mean age in the case group was 6.22 years, and 6.13 years in controls. There was no significant difference between the groups in hygiene or toileting habits. Cases were significantly more likely to wear only cotton underwear (93% v 66%, $p = 0.007$). They were less likely to have used bubble bath in the last month (25% v 42%), although this failed to reach significance. There was no difference between the groups with regard to a past history of urinary tract infection, eneuresis, constipation, encopresis, or skin disorder, or for having illness in the preceding four weeks. One case and two controls were subsequently found to have a possible history of sexual abuse. However, there was no history of genital contact or vaginal penetration in these subjects, at least one incident involving indecent exposure only. Of the controls, 46% reported at least one previous episode of redness, soreness or itching of the genital area, or vaginal discharge. However, most of these symptoms were very minor and shortlived, without medical attention being sought.

For the cases, redness and soreness were the predominant symptoms with an incidence of 82% and 74%, respectively, with itching and discharge reported in 58% and 62%, respectively. Only 10% had bleeding. One patient had lichen sclerosis, one had moderately severe eczema, and one was taking oral steroids. Twenty one per cent had received antibiotics in the preceding four weeks. Twenty girls (40%) had relatively acute onset of symptoms (≤ 14 days). The remainder reported recurrent episodes over a few weeks to several years. Examination confirmed redness of the introitus in 87% of cases, but also in 30% of the control group. All but one of the latter were graded as mild, whereas more than half the cases had distinct redness (56%).

Microbiological flora was similar overall in both groups (table 2). Mixed anaerobes and *Streptococcus viridans* were significantly more common in the control group. *Staphylococcus aureus* and group A streptococcus were more commonly isolated from cases, but numbers were small and this failed to reach significance. Four cases but no controls had no growth from swabs, despite material being obtained and seen on Gram stain; however, all had taken antibiotics in the preceding 1–2 weeks. No child in either group had *G vaginalis*, *Trichomonas hominis*, urogenital mycoplasma or *Neisseria gonorrhoeae* isolated on culture, and PCR amplification performed for *Chlamydia trachomatis* was negative for all specimens. Yeast was found only in the presence of lactobacilli, and the one case from whom can-

Table 2 Microbiology of the genital tract in cases and controls

Organism	Case (%)	Control (%)	Difference
Anaerobes	29 (58)	46 (92)	p = 0.0005
Diphtheroids	24 (48)	31 (62)	
Coagulase negative staphylococci	30 (60)	28 (56)	
<i>Escherichia coli</i>	15 (30)	17 (34)	
<i>Streptococcus viridans</i>	3 (6)	14 (28)	p = 0.003
Enterococcus	6 (12)	5 (10)	
<i>Haemophilus influenzae</i>	2 (4)	2 (4)	
<i>Streptococcus pneumoniae</i>	0	1 (2)	
Group G streptococcus	1 (2)	1 (2)	
Group B streptococcus	1 (2)	1 (2)	
Lactobacilli	(1 seen)	1 + 1 seen	
<i>Proteus</i> species	4 (8)	1 (2)	
<i>Staphylococcus aureus</i>	5 (10)	1 (2)	p = 0.08
Group A streptococcus	3 (6)	0	p = 0.07
<i>Klebsiella</i> species	1 (2)	0	
<i>Candida albicans</i>	1 (2)	0	
<i>Streptococcus milleri</i>	2 (4)	0	
<i>Pseudomonas aeruginosa</i>	1 (2)	0	
Enterobius	1 (2)	1 (2)	

Table 3 Cases with infectious aetiology

Case	Age (years)	Organism	Other relevant data
5	4.5	<i>S aureus</i>	Distinct introital inflammation, greenish discharge seen
7	15.1	<i>E coli</i>	Delayed puberty, associated UTI, white discharge seen
8	8.2	Anaerobes (profuse)	Profuse offensive discharge, distinct introital inflammation, foreign material (toilet paper) in genital area
13	7.3	Enterobius	Prednisolone treatment, lymphoedema, thin clear discharge
21	5.1	Group A streptococci	Distinct inflammation, throat infection 5 weeks before, profuse thin clear discharge
26	3.6	<i>Proteus</i> species	Lichen sclerosis, distinct excoriation, slight discharge
30	10.2	<i>C albicans</i>	Early pubertal changes, clear watery discharge, papules on surrounding skin
38	3.4	<i>H influenzae</i>	Distinct inflammation, yellow discharge
42	6.8	Group A streptococci	Impetigo of genital area, including clitoris
50	3.2	<i>E coli</i>	Associated UTI

UTI, urinary tract infection.

did not have clear evidence of pubertal change with Tanner 3 breast development, though still premenarchal.

Ten cases were diagnosed as having a predominant infective contribution to genital inflammation (table 3). Although symptoms reported by these girls were similar to other cases, examination findings in the nine cases for whom full examination data were available showed significantly more observed distinct redness of the genital area extending beyond the introitus (33% v 2.5%, p = 0.003) and visible discharge (88% v 26%, p = 0.009) in cases with probable pathogens.

Gram stains showed considerable overlap between cases and controls regarding the presence of pus, epithelial cells, and bacteria. No clue cells were seen in either group.

Discussion

The findings of this study suggest that vulvovaginitis in this age group is not usually infectious or necessarily related to poor hygiene, specific irritants or sexual abuse, although any of these can present with genital irritation. The possibility of sexual abuse should always be considered when a child presents with genital symptoms, but our data indicate it is not a common contributing factor. The fact that almost half the control group had at some time had symptoms of genital irritation

Key messages

- In a large majority of cases of premenarchal vulvovaginitis, no infectious cause can be identified
- Cases with a demonstrable infectious cause tend to have more visible discharge and distinct redness of the genital area
- Although poor hygiene, specific irritants (for example, bubble bath), and sexual abuse can all present with genital irritation, this series suggests that these factors do not contribute in most cases
- Antibiotics and antifungal creams should be used only if the relevant pathogen is identified. Initial treatment should be simple and symptomatic—for example, salt or vinegar baths

and that 30% of them had observed mild introital redness at the time of examination, and that pus cells were a frequent finding on Gram stain, suggests that genital inflammation is indeed very common in prepubertal girls. However, there is obviously a spectrum of severity with minor self-limiting episodes at one end and prolonged or recurrent episodes at the other with notable associated discomfort. Presentation to medical services will also be influenced by the level of parental concern.

There was no significant difference between cases and controls in hygiene habits, confirming our hypothesis that poor hygiene is not a major contributing factor to vulvovaginitis in our population. The anecdotal comment from several mothers of girls in the symptomatic group was that symptoms had persisted despite increased attempts at ensuring frequent bathing and good overall hygiene. If anything, there was less exposure to potential irritants such as bubble bath and synthetic underwear in symptomatic girls.

One of the aims of this study was to define clinical indicators of an infectious cause. When trying to determine the presence of infection, one must first define normal flora for the study population. The landmark studies by Hamerschlag *et al* in the late 1970s on vaginal flora in children drew subjects from a disadvantaged inner city population in the US, included girls who were sexually active, and gave no information about sexual abuse.^{6,7} Comparing our control data with the 49 girls of equivalent age in the original work, the only significant difference between the groups was in organisms thought to be associated with sexual activity, namely the urogenital mycoplasmas, *G vaginalis*, and *N gonorrhoeae*. There was also a higher incidence of yeasts found, but no information is given about pubertal status, making comparison with our group difficult.

When comparing data from different studies, methods of specimen collection should also be comparable. Some investigators have attempted to obtain vaginal specimens by lavage via a vaginal catheter or from high vaginal swabs to avoid contamination by perineal organisms.^{8,9} These studies show *Staphylococ-*

cus epidermidis, diphtheroids, and anaerobes to be the predominant vaginal organisms, as did our study on low vaginal and introital swabs. This suggests simple non-invasive methods of specimen collection yield useful results.

Diagnosing infection is confounded by the overlap between normal flora and potential pathogens. A similar situation exists on other mucosal surfaces—for example, in the throat. The presence of an organism does not of itself denote causation, and the clinical picture as well as microbiology should be considered before infection is assumed. Gram stain has become the main diagnostic tool for bacterial vaginosis in adult women,¹⁰ but there is very little literature regarding either normal or pathological appearance of vaginal smears in children, which should therefore be regarded as an adjunctive rather than a primary diagnostic tool. For this study, a pure or predominant growth of a probable pathogen in conjunction with symptoms of inflammation was considered diagnostic of a primary infective cause. However, in some girls, both cases and controls, the same organism may have been present in small numbers as part of a mixed growth but not felt to be the cause of symptoms.

Our data suggest that microbiological investigation is indicated if there is visible vaginal discharge on examination, reflecting profuse discharge, and inflammation is moderate or severe. This should include a swab and smear for Gram stain, and a pinworm test particularly if itch is a prominent feature. Simple symptomatic treatment with salt or vinegar baths is usually appropriate in the first instance. Antibiotics should only be used if a pathogen is identified. Obviously some infections may also respond to simple measures or spontaneously resolve. A vaginal foreign body or other cause should be excluded if the discharge is blood-stained, especially if there is no identified

infectious cause, the child fails to respond to treatment, or both. Although our data suggest that most episodes of vulvovaginitis are not related to sexual abuse, isolation of an organism that has a strong association with sexual transmission (for example, *N gonorrhoeae*) must prompt further investigation. That yeast was found only in the presence of lactobacilli, indicative of pubertal change in vaginal flora, confirms that candida is an unlikely pathogen in toilet trained prepubertal girls, and antifungal creams have no place in the initial treatment of vulvovaginitis in this group.

An understanding of aetiology focuses clinical management. Unfortunately, for many girls with recurrent vulvovaginitis, no clear cause is found, and treatment remains empiric. The relative contribution of either hormonal factors or host response to irritation of the genital area has yet to be defined.

Thanks to Janet Strachan, Dr Anne Smith, Marion Easton and the microbiology staff, day surgical unit and emergency department at Royal Children's Hospital, Melbourne.

- 1 Vandevan AM. Vulvovaginitis in the child and adolescent. *Paediatrics in Review* 1993;14:141–6.
- 2 Pierce AM. Vulvovaginitis: causes and management. *Arch Dis Child* 1992;67:509–12.
- 3 Straumanis JP. Group A beta-hemolytic streptococcal vulvovaginitis in prepubertal girls: a case report and review of the past twenty years. *Paediatr Infect Dis J* 1990;9:845–8.
- 4 Holland SM. Detection and differentiation of Chlamydia trachomatis, Chlamydia psittaci, and Chlamydia pneumoniae by DNA amplification. *J Infect Dis* 1990;162:984–7.
- 5 Ausubel FM. *Current protocols in molecular biology*. New York: John Wiley, 1988.
- 6 Hammerschlag MR. Microbiology of the vagina in children: normal and potentially pathogenic organisms. *Pediatrics* 1978;62:57–62.
- 7 Hammerschlag MR. Anaerobic microflora of the vagina in children. *Am J Obstet Gynaecol* 1978;131:853–6.
- 8 Gerstner GJ. Vaginal organisms in prepubertal children with and without vulvovaginitis. *Arch Gynaecol* 1982;231:247–52.
- 9 Hill GB. Anaerobes predominate among the vaginal microflora of prepubertal girls. *Clinical Infectious Diseases* 1995;20(suppl 2):S269–70.
- 10 Spiegel CA. Bacterial vaginosis. *Clinical Microbiology Reviews* 1991;October:485–502.