ORIGINAL ARTICLE

Vulvovaginitis in prepubertal girls

T Stricker, F Navratil, F H Sennhauser

Arch Dis Child 2003:88:324-326

See end of article for authors' affiliations

Correspondence to: Dr T Stricker, University Chidren's Hospital, Steinwiesstrasse 75, CH-8032 Zurich, Switzerland; Tamar.Stricker@kispi.unizh.ch

Accepted 19 September 2002

This retrospective study evaluated the clinical features and findings in bacterial cultures and in microscopic examination of vaginal secretions in 80 prepubertal girls, aged 2–12 years, with vulvovaginitis. Vaginal secretions were obtained directly from the vagina with a sterile catheter carefully inserted into the vagina. Pathogenic bacteria were isolated in 36% of cases. In 59% of these cases the isolated pathogen was group A β -haemolytic streptococcus. Candida was not found in any of the patients. The finding of leucocytes in vaginal secretions as an indicator for growth of pathogenic bacteria had a sensitivity of 83% and a specificity of 59%. Antimicrobial treatment should therefore be based on bacteriological findings of vaginal secretions and not on the presence of leucocytes alone.

ulvovaginitis is the most common gynaecological problem in prepubertal girls.¹⁻³ Factors that explain the increased susceptibility of children to vulvovaginitis include: the close anatomical proximity of the rectum; lack of labial fat pads and pubic hair; small labia minora; thin and delicate vulvar skin; thin, atrophic, anoestrogenic vaginal mucosa; and children's tendency to have poor local hygiene and to explore their bodies.¹⁻⁴ Most cases of vulvovaginitis are of non-specific aetiology. However, in some patients the symptoms are caused by infections with specific bacterial pathogens.

The present retrospective report summarises the clinical features, results of bacterial cultures, and findings in microscopic examination of vaginal secretions from 80 prepubertal girls suffering from vulvovaginitis. Subjects with suspected sexual abuse were excluded.

PATIENTS AND METHODS

Between January 1995 and December 1997, 80 prepubertal girls (Tanner stage I) who were not suspected of being sexually abused were seen in the outpatient clinic for paediatric and adolescent gynaecology of the university children's hospital of Zurich because of vulvovaginitis. Pertinent clinical data were recorded and physical examination performed. Samples of vaginal secretions were obtained for evaluation under the light microscope during the patient's visit and for bacterial cultures and Gram film. Secretions were collected using a sterile newborn suction catheter, 2–3 cm of which were carefully inserted into the vagina. Secretions were cultured onto the following agar plates: sheep's blood agar, heated blood agar, blood agar containing nalidixic acid and colistin sulphomethate, and Sabouraud's agar.

Table 1 Clinical features of 80 girls with vulvovaginitis

Features	No. (%)
Vaginal discharge	74 (92%)
Itching	36 (45%)
Redness	24 (30%)
Dysuria	15 (19%)
Pain	6 (8%)
Bleeding	4 (5%)

RESULTS

......

The age of the girls ranged from 2 to 12 years (fig 1). Table 1 summarises the clinical features of the patients. Vaginal discharge was the most common symptom (92%). Table 2 summarises microbiological findings. Bacterial pathogens were isolated in vaginal secretions of 29 (36%) girls, four cases having a mixed infection with two bacteria. Isolated pathogens included group A β-haemolytic streptococcus found in 17 girls; Haemophilus influenzae type b in two, and other types or non-typeable in five; Klebsiella pneumoniae in one; and Shigella flexneri in one. Staphylococcus aureus alone was isolated in three patients. It also appeared in mixed infections: in the patient with Shigella; in two patients with group A β-haemolytic streptococcus; and in one patient with Haemophilus influenzae. Other bacteria (such as Streptococcus viridans, Proteus species, Pseudomonas species) known to occur in healthy prepubertal girls6 were not considered the cause of illness. Candida was not isolated in any of the patients.

A total of 26 patients were treated with systemic antibiotics: 25 of the 29 with growth of pathogenic bacteria and an additional patient without growth. The four patients with bacterial

 Table 2
 Bacteria isolated from vaginal secretions of girls with vulvovaginitis

Organisms cultured	No. of positive cultures
Pathogens	
Group A β-haemolytic streptococcus	17
H influenzae type b	2
H influenzae non-b	5
Klebsiella pneumoniae	1
Shigella flexnori	1
Staphylococcus aureus*	7 (4 in mixed cultures)
Non-pathogens	· · · · · · · · · · · · · · · · · · ·
Coagulase negative staphylococci	7
Enterococcus spp.	4
Escherichia coli	4
Viridans streptococci	4
Corynebacterium	3
Pseudomonas aeruginosa	3
Proteus mirabilis	1

Eighty girls with vulvovaginitis were studied. A total of 29 patients had growth of one or more pathogens. Since four were asymptomatic by the time culture results became known, only 25 of these 29 patients received systemic antimicrobial treatment.

*The role of Staphylococcus aureus as a pathogen is questionable

rine role or *Stapnylococcus aureus* as a patnogen is que see text).

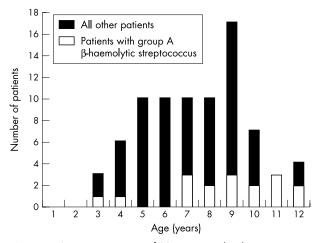


Figure 1 Age at presentation of 80 patients with vulvovaginitis.

pathogens who did not receive antimicrobial treatment were asymptomatic when culture results became known; these included three girls with isolated growth of *Staphylococcus aureus* and one with non-typeable *Haemophilus influenzae*. The additional patient who received systemic antibiotics although she did not have growth of a pathogen in vaginal secretions, was a 5 year old girl with persistent discharge and repetitive isolations of *Escherichia coli*. Selection of antibiotics was based on the susceptibility of the isolated microorganisms. Patients were followed up 2–3 weeks after completing antimicrobial treatment. Symptoms resolved in all girls, the majority reporting significant improvement 2–3 days after treatment was started.

Examination of vaginal secretions under the light microscope revealed leucocytes in 24 of the 29 patients whose culture results revealed a bacterial pathogen. The five cases in which no leucocytes were seen and yet pathogenic bacteria grew in cultures included: three cases of group A β -haemolytic streptococcus, one of *Haemophilus influenzae*, and one of *Staphylococcus aureus*. The latter was one of the patients who were asymptomatic when culture results were known and therefore did not receive antimicrobial treatment. Leucocytes were present in 21 of 51 patients who had no growth of pathogens. Thus the finding of leucocytes in vaginal secretions as an indicator for growth of bacterial pathogens had a sensitivity of 83% and a specificity of only 59%.

Group A β-haemolytic streptococcus

Group A β -haemolytic streptococcus was isolated in 21% of the girls. Their ages are shown in fig 1 and the yearly distribution in fig 2. In these patients symptoms tended to be more acute. Seven of the 17 patients (41%) had a recent history of sore throat and an additional patient had a sister with recurrent tonsillitis. In two cases a coinfection with *Staphylococcus aureus* was found.

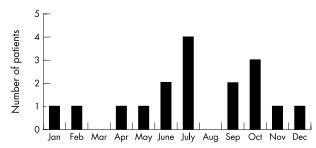


Figure 2 Yearly distribution of 17 cases of vulvovaginitis caused by group A β -haemolytic streptococcus.

DISCUSSION

When evaluating vulvovaginitis it is very important to realise that pubertal development and its hormonal influence is more important than the chronological age. This study was limited to prepubertal girls (Tanner stage I) as opposed to other studies in which patients were categorised according to age or occurrence of menarche.³ ⁷⁻¹² Patients were not routinely screened for gonorrhoea, *Chlamydia trachomatis*, or *Trichomonas*. These sexually transmitted pathogens were sought only in cases of suspected sexual abuse, all of which were excluded from the study. Since sexual abuse is not always disclosed in this population, this is a flaw with the study.

A major question in the management of vulvovaginitis is whether bacteria isolated from the patients' vaginal secretions are the actual cause of the symptoms or are part of the normal flora. A number of studies attempted to define the normal flora of the vagina by performing cultures in asymptomatic subjects, with variable results.3 7 10 13 Bacteria which are not sexually transmitted and are generally considered pathogens include: group A β-haemolytic streptococcus (Streptococcus pyogenes), Haemophilus influenzae, Staphylococcus aureus, Moraxella catarrhalis, Streptococcus pneumoniae, Neisseria meningitidis, Shigella, and Yersinia entercolitica.6 A significant weakness of this study is that there is no control group, either simultaneously or using follow up cultures in the same patients well past treatment without symptoms. In the current study a specific bacterial pathogen was found in 29 of 80 girls (36%). Other studies have reported lower rates.7 A possible explanation is that the patients were seen in a subspecialty clinic and not in a primary care setting. It is interesting to note that in a previous study from 1992,8 in which 160 girls presenting with vulvovaginitis between July 1987 and March 1990 had a bacterial swab, the most frequently isolated pathogen was Haemophilus *influenzae* (types not specified) which occurred in 22 patients. In the current study the isolation of group A β -haemolytic streptococcus by far exceeded that of Haemophilus influenzae, the former being isolated in 17 cases and the latter only in seven, two of which were Haemophilus influenzae type b. A plausible explanation is the introduction of vaccination against Haemophilus influenzae type b: purified polyribosylribitol phosphate polysaccharide (PRP) was introduced in 1985, with limited immunogenicity in infants and young children, followed by polyribosylribitol phosphate-diphtheria toxoid conjugate vaccine (PRP-D) in 1987, and Haemophilus b oligosaccharide conjugate vaccine (HbOC) in 1988.14

Staphylococcus aureus was isolated in seven patients, in four as coinfection with another pathogen and in three as a single pathogen. It is interesting to note that all three patients infected with Staphylococcus aureus alone were asymptomatic when culture results were known and did not require systemic antimicrobial treatment. It follows that when girls suffering from vulvovaginitis have isolated growth of Staphylococcus aureus in vaginal secretions it is important to inquire about persistence of symptoms before prescribing antimicrobial treatment.

Group A β -haemolytic streptococcus was isolated in 21% of the girls. The Nottingham Public Health Laboratory isolated this organism in only 11% of vaginal swabs received from children aged less than 12 years.15 The pubertal stage of the girls and the indication for examination were not specified. In a study of girls aged 1-15 years with vulvovaginitis, swabs were performed and cultured.9 Group A β-haemolytic streptococcus was found in 18% of patients. In the current study patients with group A β-haemolytic streptococcus vulvovaginitis were among the older prepubertal girls, in accordance with the fact that streptococcal pharyngitis is most common among school age children.16 In temperate climates streptococcal pharyngitis occurs more frequently in the late autumn, winter, and spring, and impetigo in warm seasons. 16 Therefore, the fact that vulvovaginitis caused by group A β-haemolytic streptococcus occurred all year round suggests that both serotypes 326 Stricker, Navratil, Sennhauser

causing pharyngitis and those causing impetigo were involved. Two other studies also suggest that infections arise from a previous respiratory or skin source, $^{15 \ 17}$ though both show a winter predominance. Approximately half of the patients had a personal or family history of pharyngitis. Vulvovaginitis caused by group A β -haemolytic streptococcus occurs typically in prepubertal girls and rarely in postpubertal patients. During the period studied, 47 pubescent patients with vulvovaginitis were seen in our outpatient clinic; in all cases bacterial cultures were performed. Group A β -haemolytic streptococcus was only found in two girls, aged 10 and 13 years, with pubertal Tanner stage II.

We routinely examine vaginal secretions of patients with vulvovaginitis under light microscope during the patient's visit. The results of this study suggest that finding leucocytes does not necessarily imply the presence of bacterial pathogens, but when leucocytes are absent such an infection is unlikely. We are not aware of other reports concerning this observation.

The role of *Candida albicans* as a cause of vulvovaginitis in prepubertal girls is controversial. In some studies it was found in asymptomatic girls and in others as a cause of symptoms.^{10 11} In this study *Candida albicans* was not isolated in any of the patients.

Another known specific cause of vulvovaginitis is pinworms (*Enterobius vermicularis*). These should be considered in girls whose major symptom is perineal pruritus, especially at night. Our past experience has shown that attempting to collect eggs using a Sellotape slide test is difficult for the parents and has a relatively low yield. Moreover, we have had patients with several negative tests who in a follow up clinical examination showed the typical thread-like worm in the vagina or anus. We therefore do not search for pinworms and in cases of clinical features suggesting infestation with them, empirically treat the patients with a single dose of 100 mg mebendazole, repeated two weeks later.

Vaginal foreign bodies cause an intense inflammation, resulting in vaginal bleeding and foul smelling, blood stained vaginal discharge. These cases were not included in the study.

Girls suffering from vulvovaginitis not caused by a specific pathogen were treated with hygienic measures: avoiding tightly fitting clothing or other irritants like harsh soaps to the vulva, front to back wiping after using the toilet, sitz baths, and protective ointments.⁴

In future studies a control group should be included and all subjects screened for *Chlamydia*, gonorrhoea, and *Trichomonas*.

Conclusion

In prepubertal girls presenting with vulvovaginitis, vaginal secretions should be obtained for examination under the light microscope during the patient's visit and for microbiological investigation. The presence of leucocytes increases the likelihood of finding bacterial pathogens which require specific treatment. In this study a bacterial pathogen was found in 36% of the cases, the most frequent being group A β -haemolytic streptococcus, probably from both respiratory and skin sources.

ACKNOWLEDGEMENT

We wish to thank the staff of the Infectious Diseases Laboratory of the University Children's Hospital Zurich for their help in processing the specimens.

Authors' affiliations

T Stricker, F Navratil, F H Sennhauser, University Chidren's Hospital, Zurich, Switzerland

REFERENCES

- 1 Altchek A. Pediatric vulvovaginitis. J Reprod Med 1984;29:359-75.
- 2 Arsenault PS, Gerbie AB. Vulvovaginitis in the preadolescent girl. Pediatr Ann 1986;15:577–9,583–5.
- 3 Jaquiery A, Stylianopoulos A, Hogg G, et al. Vulvovaginitis: clinical features, aetiology, and microbiology of the genital tract. Arch Dis Child 1999:81:64–7
- 4 Vandeven AM, Emans SJ. Vulvovaginitis in the child and adolescent. Pediatr Rev 1993;14:141–7.
- Navratil F. A simple device for removal of vaginal secretions in the prepubertal child. In: Book of abstracts. XI world congress paediatric and adolescent gynecology, Singapore, 23–26 February 1995:240.
 Emans SJ, Laufer MR, Goldstein DP. Vulvovaginal problems in the
- 6 Emans SJ, Laufer MR, Goldstein DP. Vulvovaginal problems in the prepubertal child. In: *Pediatric and adolescent gynecology*, 4th edn. Philadelphia: Lippincott-Raven, 1998:75–107.
- 7 Paradise JE, Campos JM, Friedman HM, et al. Vulvovaginitis in premenarchal girls: clinical features and diagnostic evaluation. *Pediatrics* 1982;70:193–8.
- 8 Pierce AM, Hart CA. Vulvovaginitis: causes and management. Arch Dis Child 1992;67:509–12.
- Dhar V, Roker K, Adhami Z, et al. Streptococcal vulvovaginitis in girls. Pediatr Dermatol 1993;10:366–7.
- 10 Hammerschlag MR, Alpert S, Rosner I, et al. Microbiology of the vagina in children: normal and potentially pathogenic organisms. Pediatrics 1978;62:57–62.
- Koumantakis EE, Hassan EA, Deligeoroglou EK, et al. Vulvovaginitis during childhood and adolescence. J Pediatr Adolesc Gynecol 1997:10:39–43.
- 12 Gerstner GJ, Grünberger W, Boschitsch E, et al. Vaginal organisms in prepubertal children with and without vulvovaginitis. Arch Gynecol 1982;231:247–52.
- 13 Hill GB, St Claire KK, Gutman LT. Anaerobes predominate among the vaginal microflora of prepubertal girls. CID 1995;20(suppl 2):s269–70.
- 14 Ward JI, Zangwill KM. Haemophilus influenzae vaccines. In: Plotkin SA, Orenstein WA, eds. Vaccines. Philadelphia: WB Saunders Company, 1999;183–221
- 15 Donald FE, Slack RCB, Colman G. Streptococcus pyogenes vulvovaginitis in children in Nottingham. *Epidemiol Infect* 1991;106:459–65.
- 16 American Academy of Pediatrics. Group A streptococcal infections. In: Pickering LK, ed. Red Book: Report of the Committee of Infectious Diseases, 25th edn. Elk Grove Village, IL: American Academy of Pediatrics, 2000:526–36.
- 17 Morris CA. Seasonal variation of streptococcal vulvo-vaginitis in an urban community. J Clin Pathol 1971;24:805–7.