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# *Streptococcus pyogenes* vulvovaginitis in children in Nottingham

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## SUMMARY

Isolates of *Streptococcus pyogenes* from vaginal swabs of children with vulvovaginitis received at Nottingham Public Health Laboratory during 1986–9 were studied. A total of 159 isolates was made during the 4 years, increasing from 17 in 1986 to 64 in 1989 and accounting for 11% of all vaginal swabs received from children. The numbers of throat swabs yielding *S. pyogenes* also showed an increase from 974 in 1986 to 1519 in 1989. A winter peak of isolates was noted for both vaginal swabs and throat swabs. A total of 98 strains from vaginal swabs were serotyped: 22 different types were identified, 61% of which were the common types M4, M6, R28 and M12. Erythromycin sensitivity was done on 89 strains; 84% were highly sensitive (MIC < 0·03 mg/l). There are no other reports of such large numbers in the literature; the reason for seeing this increase in Nottingham is unclear.

## INTRODUCTION

Vulvovaginitis in prepubertal girls is a common problem presenting mainly to general practitioners but occasionally of such severity to be seen by paediatricians or gynaecologists. It may be infective or non-infective in origin; *Streptococcus pyogenes* is one of the recognized infective agents [1]. Streptococcal vulvovaginitis is characterized by a serosanguinous discharge with a fiery red appearance to the vaginal mucosa and vulva, and usually responds well to penicillin treatment [2]. The first reports [3, 4] of streptococcal vulvovaginitis made an association with other streptococcal infections such as scarlet fever or pharyngitis, and this was supported by serotyping of strains from the two sites. More recent studies [2, 5, 6] support the view that vulvovaginitis is a secondary complication of streptococcal infection in another site, for example, the throat or the skin. Seasonal variation has also been noted in accord with upper respiratory tract infections [6].

The accepted treatment for streptococcal infections has been penicillin with erythromycin as an alternative for allergic patients. Erythromycin resistance in *S. pyogenes* has been relatively rare in the UK; however there are reports of sporadic outbreaks caused by resistant strains [7], and the problem seems to be becoming more widespread particularly among types M4 and M12. This may present a problem with choice of antibiotic for treating a child.

In Nottingham, since 1986, we have noticed an increase in numbers of isolates

of *S. pyogenes* from childrens' vaginal swabs. A search of the recent literature on the subject of streptococcal vulvovaginitis in children revealed only a few case reports [2, 5, 8], so it was decided to look more closely at our experience in Nottingham with regard to numbers received, seasonal variations in incidence, the serotypes involved and the prevalence of erythromycin resistance.

## MATERIALS AND METHODS

Nottingham Public Health Laboratory serves a population of more than 600000 which includes two large hospitals and approximately 350 general practitioners.

Vaginal/vulval swabs from children were transported to the laboratory in Amies transport medium. An initial wet preparation was made to look for yeasts and trichomonas, then the swab was plated onto the following media: blood agar (7% horse blood); blood agar containing 2 mg/l crystal violet, 10 mg/l nalidixic acid and 50000 units/l colistin sulphomethate (CV agar); chocolate (heated blood) agar; Modified New York City agar (Oxoid); Thayer-Martin agar with VCNT supplement (Oxoid) and CLED medium (Oxoid). Haemolytic streptococci growing in pure culture or as the predominant organism were grouped by rapid latex test (Streptex: Wellcome Reagents Ltd). Mixed cultures with fewer than 10 colonies of haemolytic streptococci were thought not to be significant and not identified further. Group A strains were sent from January 1988 onwards to the Streptococcus Reference Laboratory, Central Public Health Laboratory, Colindale for M and T typing. Heavy growths of other organisms were identified by standard methods [9].

Throat swabs from patients of all ages were plated onto a CV agar with a 0·1 unit bacitracin disk (Mast Laboratories).

Erythromycin sensitivity testing was done by the agar incorporation method using a multipoint inoculator (Denley) onto DST (Oxoid) agar containing 5% horse blood and doubling dilutions of erythromycin from 16 mg/l to 0·012 mg/l. An inoculum of $10^3$ cfu/spot was used.

Data for numbers of vaginal swabs (age < 12 years), numbers of throat swabs from patients of all ages and positive isolates of *S. pyogenes* from January 1986 to December 1989 were collected from the computerized laboratory records. Serotyping information was available for 1988 and 1989.

## RESULTS

From January 1986 to December 1989 a total of 1378 vaginal swabs from children aged less than 12 years were received. A total of 1076 (78%) of these were from general practitioners and 302 (22%) from hospital patients. Overall, 159 swabs (11·5%) from 157 patients yielded *S. pyogenes*. Table 1 shows the yearly totals with an increase both in numbers received and proportion of positives for *S. pyogenes*. The numbers of throat swabs from all patients received during the same 4-year period remained relatively constant (Table 2); however the proportion of positives for *S. pyogenes* showed an increase from 10% in 1986 to 17% in 1989.

The monthly totals for positive isolates of *S. pyogenes* show some peaks and troughs throughout the year (Table 3). The numbers of isolates per month ranged from nil in some months in 1986/87 to a maximum of nine in October 1988. There are small winter peaks of isolates from November to February for throat swabs

Table 1. *Childrens' vaginal swabs*

| Year | Total received | *S. pyogenes* + ve | (%) |
|---|---|---|---|
| 1986 | 285 | 17 | (6) |
| 1987 | 304 | 28 | (9) |
| 1988 | 382 | 50 | (13) |
| 1989 | 407 | 64 | (16) |
| Total | 1378 | 159 | (11.5) |

Table 2. *Throat swabs*

| Year | Total received | *S. pyogenes* + ve | (%) |
|---|---|---|---|
| 1986 | 9434 | 974 | (10) |
| 1987 | 8531 | 959 | (11) |
| 1988 | 8465 | 1221 | (14) |
| 1989 | 8980 | 1519 | (17) |
| Total | 35410 | 4673 | (13) |

Table 3. *Number of* S. pyogenes *positives in throat and vagina*

| | Throat | | Vagina | | | Throat | | Vagina | |
|---|---|---|---|---|---|---|---|---|---|
| 1986 | No. positive | No. sent | No. positive | No. sent | 1987 | No. positive | No. sent | No. positive | No. sent |
| Jan. | 121 | 817 | — | 11 | | 96 | 716 | 4 | 18 |
| Feb. | 101 | 834 | — | 27 | | 79 | 834 | 2 | 27 |
| Mar. | 76 | 1138 | 2 | 24 | | 74 | 919 | 2 | 29 |
| Apr. | 77 | 797 | — | 29 | | 63 | 680 | 2 | 20 |
| May | 77 | 731 | — | 19 | | 76 | 516 | — | 23 |
| June | 89 | 858 | — | 32 | | 79 | 773 | — | 36 |
| July | 79 | 684 | 4 | 31 | | 91 | 686 | 7 | 25 |
| Aug. | 68 | 659 | — | 16 | | 55 | 518 | 1 | 23 |
| Sept. | 64 | 733 | 1 | 25 | | 61 | 675 | — | 20 |
| Oct. | 66 | 676 | 1 | 15 | | 79 | 781 | 2 | 31 |
| Nov. | 69 | 656 | 3 | 26 | | 105 | 701 | 4 | 24 |
| Dec. | 87 | 851 | 6 | 30 | | 101 | 732 | 4 | 28 |
| 1988 | | | | | 1989 | | | | |
| Jan. | 110 | 736 | 4 | 34 | | 111 | 763 | 1 | 22 |
| Feb. | 120 | 807 | 6 | 31 | | 133 | 754 | 6 | 37 |
| Mar. | 99 | 800 | 5 | 41 | | 125 | 785 | 2 | 27 |
| Apr. | 89 | 580 | 3 | 26 | | 97 | 581 | 8 | 47 |
| May | 127 | 737 | 4 | 30 | | 113 | 716 | 7 | 43 |
| June | 120 | 805 | 6 | 46 | | 136 | 707 | 4 | 28 |
| July | 116 | 708 | 4 | 30 | | 146 | 682 | 7 | 42 |
| Aug. | 63 | 568 | 2 | 26 | | 134 | 633 | 7 | 31 |
| Sept. | 86 | 656 | 4 | 28 | | 105 | 719 | 3 | 32 |
| Oct. | 87 | 662 | 6 | 33 | | 146 | 887 | 9 | 44 |
| Nov. | 103 | 757 | 4 | 28 | | 181 | 1046 | 5 | 31 |
| Dec. | 101 | 649 | 2 | 29 | | 92 | 707 | 5 | 23 |

and vaginal swabs and a summer peak in June and July seen in the vaginal isolates.

Other potential pathogens isolated from the vaginal swabs over the 4 years were *Haemophilus influenzae* (73 isolates), *Candida* sp. (37) and *S. pneumoniae* (15).

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Table 4. *Typing results 1988–9*

| M/R Type* | 1988 | 1989 | Total number |
|---|---|---|---|
| 4 | 7 | 15 | 22 |
| 6 | 4 | 11 | 15 |
| R28 | 8 | 4 | 12 |
| 12 | 3 | 8 | 11 |
| 2 | 4 | 3 | 7 |
| 1 | 4 | 2 | 6 |
| 75 | 1 | 5 | 6 |
| 3 | 3 | 2 | 5 |
| 9 | 1 | 1 | 2 |
| 49 | 2 | — | 2 |
| 22 | 2 | — | 2 |
| 25 | — | 1 | 1 |
| Total | 39 | 52 | 91† |

* Six strains did not type with the M antisera but were T types 28, 8/25/IMP 19, 5/27/44, 3/13/B3264 and 4.

† One strain was M and T negative, anti OF 48 positive.

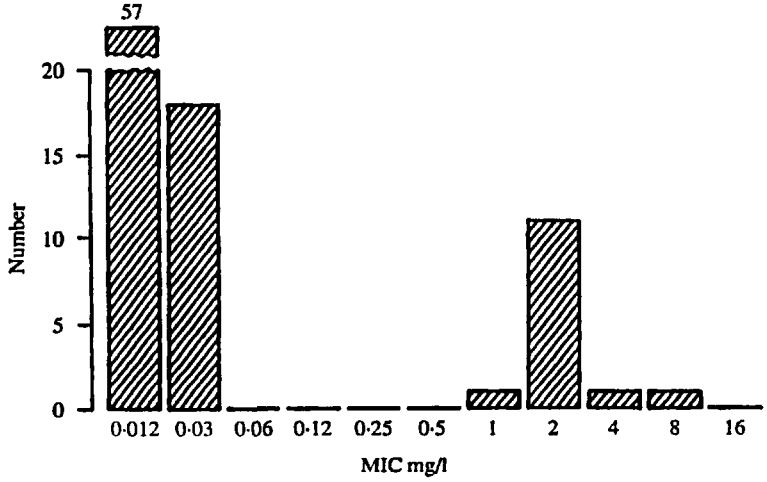


Fig. 1. Susceptibility to erythromycin of vaginal isolates of *S. pyogenes*.

Haemolytic streptococci not group A were isolated less frequently, namely group B (15 isolates), group G (13) and group C (9). Coliforms were commonly isolated but were generally considered not to be pathogens in this site. All swabs were cultured for *Neisseria gonorrhoeae*; during the years there were three isolates.

The serotyping results of 98 strains during 1988 and 1989 are shown in Table 4. The commonest M type was M4 which accounted for 22% of all strains. Four types, namely M4, M6, R28, and M12 accounted for 60% of all strains. These types are common serotypes currently circulating in the United Kingdom, and generally are associated with throat infections. They were evenly distributed throughout the year. There were smaller numbers of strains sometimes associated with skin infections such as T 8/25/IMP19, T 3/13/B3264, M49 and these were more often isolated in the summer months of June to August. The numbers of the

common types M4, M6, M12, have more than doubled from 1988 to 1989; that of R28 has halved. Altogether, 22 different serotypes were identified.

Throat swabs are not routinely sent with vaginal swabs from children, but eight pre-treatment throat swabs were received by request. These yielded *S. pyogenes* of the same type as the vaginal isolate; none of the children had symptoms or signs of pharyngitis.

Susceptibility to erythromycin was tested on 89 strains (Fig. 1). The majority (75; 84%) were fully sensitive with an MIC of 0·03 mg/l or less. Fourteen strains (16%) had an MIC of > 1 mg/l; 11 of these had an MIC of 2 mg/l, one of 4 mg/l and one of 8 mg/l. The latter strain was type M1, and apart from two non-typable strains these others were all type M4.

## DISCUSSION

Vulvovaginitis in prepubertal children is a common problem which may have several infective or non-infective causes. The anatomy of the area and the thin uncornified vaginal epithelium at this age makes it susceptible to infection, and there may be predisposing factors such as poor hygiene, use of irritant bubble baths, threadworm infestation, foreign bodies or sexual abuse. Bacterial infections may arise from gut organisms or may be transmitted from the respiratory tract by the hands.

*S. pyogenes* was first associated with pre-pubertal vulvovaginitis in 1948 [3] when 21 symptomatic patients with *S. pyogenes* in vaginal cultures were described: streptococcal infections in other sites were present in 10 of the patients, and 11 of the patients had the same strain in the throat. Other reports of vaginitis have shown throat carriage is also present: Ginsburg [2] reported nine children with symptoms of vaginitis and positive cultures, five of these had positive throat cultures. Hedlund [4] looked at all patients being treated for streptococcal infections and found 10–15% of them had vaginal infections each with the same strain as present in the throat. So one can surmise that the infection is transmitted from the throat to the vulva either by the fingers or by faecal carriage. Faecal carriage in healthy people is uncommon: Schoenknecht and co-workers [10] looked at 133 adults and 22 children and found only a single anal carrier. On the other hand, children with streptococcal throat infections or scarlet fever have been found to have an anal carriage rate of 6% [11]. Studies of vulval carriage in children are rare; one such study [12] looked at 100 healthy children and found no haemolytic streptococci; *Escherichia coli* was the commonest organism isolated. These findings would suggest that *S. pyogenes* may be a significant pathogen when cultured from this site, particularly in the presence of the characteristic signs of serosanguinous discharge and fiery red vulval mucosa. The response to penicillin treatment would also confirm this. A more recent study [13] looked at all haemolytic streptococci isolated from the genital tract and found that of the 41 patients with *S. pyogenes*, 16 of these were premenarchal, and all were more likely to have symptoms than patients with other streptococci, particularly group B streptococci.

Seasonal variation of streptococcal sore throats has been reported with winter peaks and a drop in the summer months [14]. A study looking specifically at

vulvovaginitis [6] found an increase in isolates in the winter, which would correspond to the winter peak of upper respiratory tract infections. Only 3% of the samples were from children, 20% of these were positive for *S. pyogenes*. Our study also shows a winter peak for these infections which would agree with previous reports.

No other report describes the number of cases we have seen in Nottingham, most other reports are of sporadic cases. The reasons for this increased incidence are unclear; there may be an increase in throat carriage of *S. pyogenes* with a concomitant increase in vulval infections; or there may be an increased awareness of vulvovaginitis in young girls, resulting in more swabs being sent.

A change in infecting serotypes could not account for the increase in infection: the common serotypes in this study correspond to the common types seen nationally. A report from the Division of Hospital Infection, Colindale [15], shows that M types 4 and 12 are common and have remained constant from 1980–7. Our other common types M6 and R28 are also seen, M6 having risen in incidence initially, then fallen and R28 having increased over the 7 years. Common serotypes from vaginal swabs of all ages are M1, M4, R28, which corresponds to our findings in children. The common skin types such as M49 are represented in our figures and were seen in the summer months when one could postulate that the infection was caused by skin organisms.

These common types are also reported in invasive infections; a series of bacteraemias from Cambridge [16] report M12 as the commonest type, and a series from Nottingham [17] where a range of types were seen, M1 being the commonest.

Erythromycin resistance of *S. pyogenes* in the United Kingdom has been rare; there are reports of outbreaks with resistant strains from different areas [18–20] and transferable resistance has been described [7]. These resistant strains have typed as M4 or M12. A 2-year national survey showed a resistance rate of 3% for *S. pyogenes* [21]. Data from the Division of Hospital Infection, Central Public Health Laboratory, Colindale, suggests that 13% of their referred strains are erythromycin resistant; however this is probably biased because of the pattern of referrals to the Division of Hospital Infection.

The few highly resistant strains in our survey were not unexpected; it is perhaps worrying that there were altogether 16% of strains with moderate resistance to erythromycin as this is the usual alternative to penicillin in this age group.

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## REFERENCES

1. Forfar JO, Arneil GC, eds. Textbook of paediatrics. 3rd ed. Edinburgh: Churchill Livingstone 1984; 1095–6.
2. Ginsburg CM. Group A streptococcal vaginitis in children. Ped Infect Dis 1982; 1: 36–7.
3. Boisvert PL, Walcher DN. Hemolytic streptococcal vaginitis in children. Pediatrics 1948; 2: 24–9.

4. Hedlund P. Acute vulvovaginitis in streptococcal infections. Acta Paediat 1953; **42**: 388–9.
5. O'Connor PA, Oliver WJ. Group A $\beta$-haemolytic streptococcal vulvovaginitis: A recurring problem. Ped Emerg Care 1985; **1**: 94–5.
6. Morris CA. Seasonal variation of streptococcal vulvovaginitis in an urban community. J Clin Path 1971; **24**: 805–7.
7. Scott RJD, Naidoo J, Lightfoot NF, George RC. A community outbreak of group A beta haemolytic streptococci with transferable resistance to erythromycin. Epidemiol Infect 1989; **102**: 85–91.
8. Figueroa-Colon R, Grunow JE, Torres-Pinedo R, Rettig PJ. Group A streptococcal proctitis and vulvovaginitis in a prepubertal girl. Ped Infect Dis 1984; **3**: 439–42.
9. Cowan ST. Cowan and Steel's manual for the identification of medical bacteria, 2nd ed. London: Cambridge University Press, 1974.
10. Schoenknecht FD, Batjer JD, Sherris JC. Anal streptococci. New Eng J Med 1969; **281**: 220.
11. Asnes RS, Vail D, Grebin B, Sprunt K. Anal carrier rate of group A beta hemolytic streptococci in children with streptococcal pharyngitis. Pediatrics 1973; **52**: 438–41.
12. Hammerschlag MR, Alpert S, Rosner I, et al. Microbiology of the vagina in children: Normal and potentially pathogenic organisms. Pediatrics 1978; **62**: 57–62.
13. Lewis RFM. Beta-haemolytic streptococci from the female genital tract: clinical correlates and outcome of treatment. Epidemiol Infect 1989; **102**: 391–400.
14. Boycott JA. Seasonal variations in streptococcal infections. Lancet 1966; i: 706–7.
15. Gaworzewska E, Colman G. Changes in the pattern of infection caused by *Streptococcus pyogenes*. Epidemiol Infect 1988; **100**: 257–69.
16. Francis J, Warren RE. *Streptococcus pyogenes* bacteraemia in Cambridge – A review of 67 episodes. Quart J Med 1988; **68**: 603–13.
17. Ispahani P, Donald FE, Aveline AJD. *Streptococcus pyogenes* bacteraemia: an old enemy subdued, but not defeated. J Infect 1988; **16**: 37–46.
18. Barnham M, Cole G. Erythromycin-resistant beta-haemolytic streptococci in North Yorkshire. J Infect 1986; **13**: 200–2.
19. Youngs ER. Erythromycin resistant *Streptococcus pyogenes* in Merseyside. J Infect 1984; **8**: 86–7.
20. Walker M, Whetstone RJ, Whipp J. Erythromycin resistant *Streptococcus pyogenes* in Cambridge. J Infect 1984; **8**: 88–9.
21. Spencer RC, Wheat PF, Magee JT, Brown EH. Erythromycin resistance in streptococci. Lancet 1989; i: 168.