

Epidemiological features and control of an outbreak of scarlet fever in a Perth primary school

Kynan T Feeney,¹ Gary K Dowse,² Anthony D Keil,³ Christine Mackaay,⁴ Duncan McLellan⁵

Abstract

Scarlet fever was associated with feared outbreaks and mortality in the 19th Century. It occurs sporadically in modern society and infection is readily treated with antibiotics. We report on a scarlet fever outbreak in children attending a primary school in Perth, Western Australia, in late 2003. A total of 13 cases were identified over a five week period. Six of the cases were pre-primary children (ages 4 to 5) from the same class of 26 children (attack rate 23.1%). Three of the remaining seven cases were older siblings of pre-primary cases who developed scarlet fever after their younger siblings. Screening of the children and teachers from the two pre-primary classes at the school yielded 12 positive pharyngeal swabs for group A *Streptococcus*. *Emm*-typing of the screening isolates indicated that a common strain was circulating within the outbreak pre-primary class, with four of six isolates identified as *emm*-type 3. The overall group A *Streptococcus* carriage rate in screened students in this class was 31.6 per cent and the carriage rate for *emm*-type 3 was 21.1 per cent. Carriers were treated with oral penicillin V to eradicate carriage and control the outbreak. No further cases of scarlet fever were reported after the treatment of pharyngeal carriers. Outbreaks of scarlet fever still occur in young children and identification and treatment of carriers may still be valuable. *Commun Dis Intell* 2005;29:386–390.

Keywords: scarlet fever, *Streptococcus*, *emm*-typing

Introduction

Scarlet fever is caused by infection with group A *Streptococcus* (GAS), usually in the context of acute pharyngitis. It is characterised by a distinctive erythematous blanching rash that is composed of fine papules that cause the skin to feel like sandpaper and usually starts on the upper trunk, travelling distally and sparing the soles and hands.¹ Other characteristic clinical features of scarlet fever include: an initial white covering of the tongue, followed by enlargement of the papillae, giving a distinctive 'strawberry tongue' appearance; flushing of the cheeks; and circumoral pallor.¹ Although rarely seen in present times, scarlet fever can be complicated by localised extension of infection leading to mastoiditis, sinusitis and peritonsillar abscesses and can be followed by serious complications, such as acute rheumatic fever.²

During the 19th Century scarlet fever was a feared disease due to its infectivity and associated high mortality rate.^{3–5} Severe scarlet fever is now uncommon and usually causes only mild disease, with many developed countries removing it from their notifiable disease registers over the past few decades.^{2,3,6}

Group A *Streptococcus* is most commonly transmitted through direct contact or large droplet spread and the incidence of infection is highest in young children.⁷ Administration of antibiotics will prevent transmission of GAS within 24 hours of beginning treatment.¹

We describe the clinical and epidemiological features of a recent outbreak of scarlet fever in young children attending a Perth primary school, and the public health measures that were implemented to control the outbreak.

1. Public Health Registrar, Department of Health, Perth, Western Australia
2. Medical Epidemiologist, Communicable Disease Control Directorate, Department of Health, Perth, Western Australia
3. Head, Department of Microbiology, Women's and Children's Health Service, Subiaco, Western Australia
4. Communicable Diseases Coordinator, North Metropolitan Public Health Unit, Perth, Western Australia
5. Infectious Diseases Physician and Clinical Microbiologist, Fremantle Hospital and Western Diagnostic Pathology, Perth, Western Australia

Corresponding author: Dr Kynan Feeney, Communicable Disease Control Directorate, Department of Health, Western Australia, PO Box 8172, Perth Business Centre, WA 6849. Telephone: +61 8 9431 0217. Facsimile: +61 8 9431 0222. Email: Kynan.Feeney@health.wa.gov.au

Methods

Scarlet fever remains a notifiable disease in Western Australia. Following notification of several cases of scarlet fever in children from the same primary school in Perth, Western Australia, an epidemiological investigation was performed, with a view to initiating control measures.

Case definition

Scarlet fever cases in school students and staff of the school, or family members of a child from the school who met the case definition, were categorised as possible, probable or confirmed, if they developed symptoms between 30 October and 30 November 2003 and fulfilled the following criteria:

Confirmed case: Clinical symptoms consistent with streptococcal sore throat AND at least one characteristic sign of scarlet fever AND a positive laboratory isolate of GAS from a throat swab. The characteristic signs of scarlet fever, as documented from a medical examination or reported by the child's parents, included:

- skin rash – fine erythematous, punctate, blanching on pressure and with a sandpaper texture and predominantly truncal distribution;
- strawberry tongue;
- flushing of cheeks and circumoral pallor; and
- desquamation of the skin in convalescence.

Probable case: Clinical symptoms consistent with streptococcal sore throat AND at least one characteristic scarlet fever sign AND no throat swab performed or throat swab performed and had no significant growth.

Possible case: Clinical symptoms consistent with a streptococcal sore throat AND no characteristic signs of scarlet fever AND no throat swab performed or throat swab performed and had no significant growth.

Cases were identified following notification by a doctor, or through telephone interviews of parents of children, or staff members, who were reported to have been absent from school during the period 30 October to 30 November 2003.

Screening of students and teachers in class 1 (pre-primary class, ages 4 to 5 years) and class 2 (split pre-primary/year 1 class, ages 4 to 6 years) was performed at the school on 2 December and 4 December 2003. As most of the cases were in the pre-primary age group and class 1 and 2 were sometimes joined together for group activities, screening of children in both class 1 and 2 was performed to identify possible carriers.

Screening consisted of swabbing the posterior pharynx and tonsils using a dacron swab (Transtube© Medical Wire and Equipment, UK) by two of the authors (KF and CM). Swabs were stored at room temperature and transported to the microbiology laboratory on the day of screening.

Informed consent was obtained during the outbreak to obtain samples and administer antibiotics. Other data were obtained through the notifiable diseases register and school absentee records.

Laboratory methods

Diagnosis of GAS carriage was made by culturing swabs within four hours of collection onto Columbia agar (Oxoid, Australia) with 5 per cent horse blood containing colistin 0.75 mg/L and nalidixic acid 0.5 mg/L (CNA) plates. CNA plates were incubated at 37° C for 24 hours in 5 per cent CO₂. After primary plating, swabs were then placed into Todd Hewitt broth (Oxoid, Australia) with colistin 1 per cent mg/L and nalidixic acid 1 mg/L for enrichment culture for 24 hours at 37° C. Following incubation, an aliquot was subbed onto CNA plates for a further 24 hours incubation at 37° C in 5 per cent CO₂. Beta haemolytic colonies were serogrouped using a Streptococcal Grouping Kit (Oxoid, Australia).

Epidemiological surveillance of GAS infection has traditionally depended on the Lancefield M protein serotyping system. There are a number of limitations with this approach, in particular the high proportion of isolates in Australia which are M-non-typeable.^{8,9} To overcome these limitations a non-serologic typing system that involves sequencing the 5' end of the *emm* gene (*emm*-typing) was utilised in this study. This molecular typing system is based on the established relationship between published *emm* sequences and M serology and sensitively reflects the M specificity of the isolate.^{10,11}

The *emm*-typing of isolates was performed by one author (DM) who was blinded to the school class of origin and clinical status of the source of each of the isolates.

Results

Cases

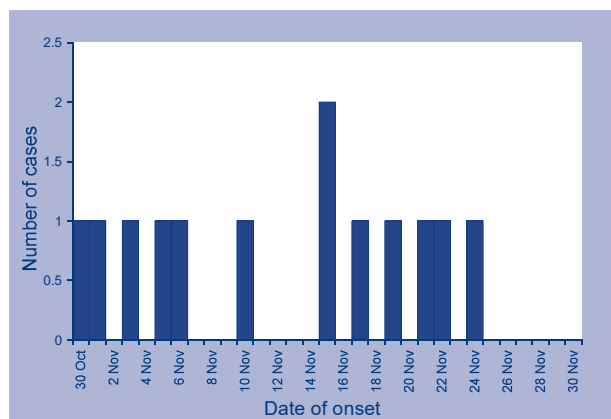
Based on notification data and interviews, there were three confirmed cases, 10 probable cases (of which eight had no throat swabs performed and two had negative throat swabs) and one possible case (Table 1). The low number of confirmed cases reflected the low rate of collection of microbiological samples to confirm GAS infection by general practitioners. Both confirmed and probable clinical cases (13 in total) are included together as cases for all subsequent analyses.

Table 1. Summary of epidemiological features of an outbreak of scarlet fever in a primary school, Perth, 2003

Variable	Number
Total number of cases:	
Confirmed	3
Probable	10
Possible	1
Background of confirmed and probable cases:	
Class 1 pre-primary students	6
Siblings of cases from class 1	3
Sibling of non-case child in class 1 (neither confirmed or probable case)	1
Student cases with no relationship to any students in class 1	2
Teacher case with no relationship to any students in class 1	1

As can be seen from the epidemic curve (Figure), the first case became symptomatic on 31 October, followed by a series of cases of scarlet fever spreading in a propagated outbreak pattern over the subsequent four weeks. The predominant clinical features experienced by the cases were sore throat (100%), fever (93%), rash (85%), headache (61%), abdominal pain (38%), strawberry tongue (31%), vomiting (31%) and skin desquamation (8%).

Figure. Epidemic curve showing number of scarlet fever cases, Perth, 2003, by date of onset of symptoms



The cases were composed of 12 children and one teacher. Six of the child cases were from class 1, giving an overall attack rate of 20 per cent (6 cases out of 26 students and 4 teachers). Among students only, the attack rate was 23.1 per cent. There were no cases in class 2.

Three of the remaining six child cases were siblings of the pre-primary cases from class 1. Two of these cases were in year 3 (aged 8 and 9 years) and one case was in year 2 (aged 7 years). All three of the older sibling cases developed symptoms of infection after their younger pre-primary sibling developed symptoms, with intervals between onset of 4, 5 and 20 days.

Of the three remaining child cases, one was in year 2 and had a younger pre-primary sibling in class 1. The latter child was the single 'possible' case who did have an upper respiratory tract illness but lacked specific signs of scarlet fever. The older sibling case developed scarlet fever after her pre-primary 'possible' sibling case was reported to be ill, suggesting the 'possible' pre-primary case from class 1 may have had GAS pharyngitis. The two remaining children, who were in years 3 (aged 9) and 5 (aged 11), did not have siblings in class 1.

The teacher with scarlet fever was not a pre-primary teacher. Her only contact with pre-primary children was during lunchtime. She had two young children, neither of whom attended the outbreak primary school or had experienced a recent illness consistent with scarlet fever or GAS pharyngitis.

Carriage studies

Table 2 summarises the epidemiological findings from classes 1 and 2. Although class 1 and 2 are in separate rooms, the two classes have intermittent combined teaching time in one room.

A total of 48 out of a possible 57 students and staff from the two classes were screened, consisting of 41 (of 50) children and all 7 teachers. Eleven of 41 students (26.8%) and one of seven (14.3%) teachers had positive throat swabs. All 12 positive throat swabs grew GAS on primary culture.

Class 1 had six children with positive swabs (carriage rate 23.1%), two of which were from previous cases of scarlet fever who had been treated with oral penicillin. The positive screening tests from class 2 were from five children (carriage rate 20.1%) and one teacher (carriage rate 33%). A course of oral penicillin V for 10 days was taken by all GAS carriers in an attempt to eradicate carriage of GAS within this population and terminate the outbreak.

Eleven of the 12 isolates were *emm* typeable: five from class 1 and six from class 2. As summarised in Table 2, four of six isolates from pre-primary class 1 were *emm*-type 3, giving a carriage rate in screened students of 21.1 per cent. Among the six isolates from class 2, two were *emm*-type 3, two were *emm*-type 2 and there was one each of *emm*-types 28 and 75.

Table 2. Epidemiological features of class 1 and 2 from affected primary school

Variable	Class 1	Class 2
Age of students	4-5 years	4-6 years
Number in each class:		
Students	26	24
Teachers	4	3
Number of cases:		
Confirmed	2	0
Probable	4	0
Attack rates:		
Students and teachers	20.0%	0%
Students only	23.1%	0%
Number screened:		
Students		
Total	19	22
Percentage positive	31.6%	22.7%
Number positive	6	5
Teachers		
Number	4	3
Percentage positive	0%	33.3%
Number positive	0	
<i>emm</i> type results from throat swab screening:		1
Not able to be typed	1	0
<i>emm</i> type:		
2	0	2
3	4	2
12	1	0
28	0	1
75	0	1

Discussion

This article reports on a school-based outbreak of scarlet fever. There were 13 confirmed or probable cases identified over a five week period. Six of these cases were from class 1 and the epidemiological and microbiological evidence suggests that this class was the epicentre for this outbreak, with most of the additional cases occurring in older siblings of children from this class.

Our investigation was limited to staff and students who attended the one primary school. As telephone questionnaires were undertaken with staff and parents of children with sick absences over the study period, we do not believe a significant number of cases were missed during the investigation.

The finding of a common *emm*-type (type 3) in four of five typeable isolates from children in the affected class suggests this was the circulating outbreak strain. Further evidence to support this theory is that of the two previously treated cases who subsequently tested positive when screened, one had an untypable isolate and the other was *emm*-type 3. This case had a short 5-day course of oral penicillin V, which is not considered as reliable as a ten day course of penicillin to reliably eradicate GAS from the pharynx.¹²

The other *emm*-types detected from screening of students and teachers were likely due to a carriage state of non-epidemic GAS. The prevalence of pharyngeal carriage of GAS can be up to 15 per cent outside of the outbreak setting.⁶ Asymptomatic pharyngeal carriage can be higher during institutional outbreaks, with rates of 20–30 per cent reported.^{6,13,14} Although asymptomatic pharyngeal carriers are not efficient transmitters of infection, screening and treatment of carriers is generally recommended in the outbreak setting of invasive GAS infection.^{1,6,13} In this instance, 21.1 per cent (4 of 19) of students in class 1 were carrying *emm*-type 3 at the time of screening, even after five of the 19 screened students had been treated for scarlet fever.

Because of ongoing transmission of GAS causing scarlet fever within the school and family setting, the concern of parents and staff at the school, and the possible serious complications of GAS infection, it was decided to screen students and staff of the affected pre-primary school classes and treat asymptomatic carriers with the aim of interrupting transmission and preventing further cases.

No further cases of scarlet fever were notified from the primary school after screening and treatment of carriers was undertaken. Although we cannot be certain that the outbreak would not have terminated naturally at that time, the abrupt cessation suggests that the intervention of screening and administration of antibiotics to carriers of GAS was effective in halting the epidemic.

Scarlet fever is an uncommon infectious disease in Australia. There was a mean of 17.7 cases of scarlet fever notified in Western Australia per year from 1995–2004 (range 6–27). This outbreak was brought to the attention of the Department of Health Western Australia due to a small clustering of notified cases in children attending the same primary school. It is debatable whether scarlet fever should still be listed as a notifiable disease in Australia, however this outbreak and the subsequent control measures demonstrate there is some utility in continuing to monitor this infection.

Acknowledgments

We gratefully acknowledge the staff, students and families for their support during the investigation and management of the outbreak. We wish to thank the nursing staff, Ms Leanne Brown of the Department of Microbiology, Women's and Children's Health Service, Perth, Western Australia, for GAS culture and serogrouping, and the Molecular Diagnostics Unit at the PathCentre, Perth, Western Australia for performing the *emm*-genotyping.

References

1. Heymann DL, editor. *Control of Communicable Diseases Manual* 18th edn. Washington: American Public Health Association, 2004.
2. Stevens DL. Life-threatening streptococcal infections: scarlet fever, necrotizing fasciitis, myositis, bacteremia, and streptococcal toxic shock syndrome. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis*. New York: Oxford University Press; 2000. p. 163–179.
3. Efstratiou A. Group A streptococci in the 1990s [Review]. *J Antimicrob Chemother* 2000;45:3–12.
4. Denny FJ. History of Hemolytic Streptococci and Associated Diseases. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis*. New York: Oxford University Press; 2000. p. 1–18.
5. Duncan CJ, Duncan SR, Scott S. The dynamics of scarlet fever epidemics in England and Wales in the 19th century. *Epidemiol Infect* 1996;117:493–499.
6. Pickering L, Baker C, Overturf G, Prober C, eds. *Red Book*, 2003. Report of the Committee on Infectious Diseases. 26th edn. Elk Grove Village: American Academy of Pediatrics; 2003.
7. Stevens DL. Group A Beta-Hemolytic Streptococci: virulence factors, and spectrum of clinical infections. In: Stevens DL, Kaplan EL, editors. *Streptococcal Infections: clinical aspects, microbiology, and molecular pathogenesis*. New York: Oxford University Press; 2000. p. 19–36.
8. Relf WA, Martin DR, Sriprakash KS. Identification of sequence types among the M-nontypeable group A streptococci. *J Clin Microbiol* 1992;30:3190–3194.
9. Maxted W. Disease association and geographical distribution of the M-types of group A *Streptococcus*. In: Read, SE, Zabriskie, JB, eds. *Streptococcal diseases and the immune response*. New York: Academic Press; 1980. p. 763–777.
10. Beall B, Facklam R, Thompson T. Sequencing *emm*-specific PCR products for routine and accurate typing of group A streptococci. *J Clin Microbiol* 1996;34:953–958.
11. Beall B, Facklam R, Hoenes T, Schwartz B. Survey of *emm* gene sequences and T-antigen types from systemic *Streptococcus pyogenes* infection isolates collected in San Francisco, California; Atlanta, Georgia; and Connecticut in 1994 and 1995. *J Clin Microbiol* 1997;35:1231–1235.
12. Schwartz B, Marcy SM, Phillips WR, Gerber MA, Dowell SF. Pharyngitis – principles of judicious use of antimicrobial agents. *Paediatric* 1998;101:171–174.
13. Humes H, ed. *Kelley's Textbook of Internal Medicine*. 4th edn. Philadelphia: Lippincott Williams and Wilkins; 2000.
14. Falck G, Kjellander J. Outbreak of group A streptococcal infection in a day-care center. *Pediatr Infect Dis J* 1992;11:914–919.