Invasive group A streptococcus carriage in a child care centre after a fatal case

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Abstract

After a fatal case of invasive group A streptococcal disease, serotype T-1, in a child care centre, group A streptococcal T-1 prevalence was measured and risk factors for carriage were determined. A total of 87% (224/258) had throat culture tests. Group A streptococcus was isolated from 57 (25%), and of the 50 isolates serotyped, 38 (76%) were T-1. Group A streptococcal T-1 prevalence was 18% (38/217) and six of nine rooms had children with group A streptococcal T-1 isolates. The risk of group A streptococcal T-1 carriage was increased for children who shared the index case's room (odds ratio (OR)=2.7; 95% confidence interval (CI)=0.8 to 9.4) and for each additional hour per week in child care (OR=1.03; 95% CI=1.001 to 1.061); and decreased in children taking antibiotics in the preceding four weeks (OR=0.2; 95% CI=0.1 to 0.9). Carriage of the invasive group A streptococcal strain could not be determined by identified risk factors alone.

(Arch Dis Child 1994; 71: 318-322)

During the 1980s, the occurrence of invasive group A streptococcal disease in children and adults increased in the United States and other countries.¹⁻⁷ A recent historical review of severe group A streptococcal diseases during premodern and modern times described periodic fluctuations in the incidence of severe disease.⁸ It has been speculated that the declining incidence and severity of group A streptococcal infections earlier this century was due to improved living conditions, a possible decrease in the organism's virulence, and the effect of antibiotics.9 The reasons for the recent re-emergence of severe group A streptococcal disease remain unclear; the increase may be associated with changes in the serotype distribution. A recent investigation of group A streptococcal disease in the United States between 1972 and 1988 reported that the proportion of the disease caused by serotypes M-1 and M-3 increased significantly and that these serotypes are more likely to cause invasive disease.10

The spread of group A streptococcal infection in child care,^{11 12} primarily through large aerosolised respiratory droplets and direct contact, occurs because of the high degree of shared secretions and close physical contact among young children in this setting. Currently, group A streptococcal infections among children in child care are not recognised as a serious problem, with the exception of strains associated with rheumatic fever or glomerulonephritis.¹³

Group A streptococcal infections may take on more importance than previously because of the increase in the occurrence of invasive group A streptococcal disease, the potential for transmission of the infection among children in child care, and the recent increase in the number of children attending child care services.¹⁴ In order to address new concerns about invasive group A streptococcal disease and develop interventions, an understanding of the epidemiology of group A streptococcal infections in child care settings is important, including the role of cultures and antimicrobial treatment¹² or prophylaxis.

After a single fatal case of invasive group A streptococcal disease in a child care attendee in Alabama, we initiated an investigation to determine the prevalence of group A streptococcus and identified risk factors for carriage of the organism among children attending the centre. We also empirically recommended antibiotic prophylaxis to all persons who had a group A streptococcal isolate identified during the investigation.

Methods

Medical records of the index case were reviewed. Attendance records and work schedules of all children and staff present at any time from approximately two weeks before until one and a half weeks after the index patient's onset of illness on 1 August (specifically 15 July until 10 August 1991, hereafter referred to as the study period) were obtained from the child care centre. A questionnaire was administered to parents to determine symptoms of recent illness and medical treatment and to supplement data from day care attendance records. Mean hours per week at the centre and room location were determined from questionnaire information or centre records for respondents and from centre records alone for non-respondents.

Between one and two weeks after illness onset in the index case (August 8–14), throat swabs for group A streptococcus were obtained from the staff and, after parental informed consent, children who attended the centre during the study period. Cultures were not taken for other organisms. We calculated group A streptococcal T-1 prevalence by dividing the number of children with group A streptococcal T-1 isolates by the total number excluding those with non-serotyped isolates.

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Accepted 13 July 1994

For all who had group A streptococcal isolates during the culture survey, an antimicrobial regimen of oral penicillin for 10 days with rifampicin added on the final four days was recommended.¹⁵ Antibiotic use information and repeat cultures were obtained between five and 10 days after completion of the antibiotic regimen. Reported antibiotic use was confirmed by a doctor by telephone or mail.

Throat swabs were transported to and then cultured by the Bureau of Clinical Laboratories, Alabama Department of Public Health, at the Montgomery Laboratory. Respiratory Bacterial Reference The Laboratory, Centers for Disease Control and Prevention (CDC), Atlanta, Georgia, performed T serotyping by the slide agglutination method and with antisera prepared at CDC; no M serotyping was performed because essentially all serotype T-1 strains are M-1 serotype.^{16 17} No further characterisation of the organism was performed. A recent report suggests that there is little variation in the genome within this serotype.¹⁸

Data were entered using Epi Info computer software (CDC, Atlanta, Georgia). Categorical variables were compared using the Mantel-Haenszel χ^2 test or two tailed Fisher's exact test as appropriate. SAS computer software (SAS Institute, Cary, North Carolina) was used for the multivariate logistic regression analysis. Variables examined in this analysis included age, race, sex, total attendees in classroom, entry time of day, hours per week in day care, index case classmate, and antibiotic use during the month before the culture survey.

Results

INVASIVE CASE

The index case was a 22 month old girl who attended the child care centre for several months and was present between 35 and 40 hours per week during the month before onset of the illness. She had experienced no previous serious illnesses or hospitalisations and had received all recommended vaccinations. She saw her physician three days before her death for a mild illness characterised by rash and fever. The rash, which was maculopapular with some vesicles, was distributed over the torso, face, the plantar aspect of one foot and was in her oral cavity. She was suspected to have varicella. Her temperature was 101.4°F. She was treated symptomatically and did not receive antibiotics. She developed vomiting and diarrhoea on the second day of illness. Relatively suddenly on the third day of illness she developed respiratory distress and returned for further medical evaluation. Upon reexamination she was found to have severe respiratory distress and shock. There were several haemorrhagic vesicular skin lesions on her trunk and extremities. A chest radiograph revealed a right middle and lower lobe cavity pneumonia. She went into cardiorespiratory arrest, resuscitation was unsuccessful, and the patient died within a

few hours of presentation. Group A streptococcus, subsequently determined to be serotype T-1, was isolated from the blood.

CHILD CARE CENTRE INVESTIGATION

The child care centre had nine rooms to which children were assigned with others consistent with their age and developmental level. Two rooms had children with a mean age <1 year in which limited child to child contact occurred; five rooms had children with a mean age between 1 and 4 years where extensive child to child contact occurred and play items were commonly shared; and two rooms had children with a mean age >4 years.

Throat swabs were obtained for culture from 224 (87%) of 258 attendees and 24 (96%) of 25 staff. More than two thirds of the parents or guardians of the participating attendees (152/224, 68%) and staff (20/24, 83%) completed questionnaires.

Fifty eight per cent of the attendees were white and 52% were boys. On average, attendees were present at the child care centre 23 hours per week (range 0.5-66). Fifty seven (25%) attendees had group A streptococcal isolates. Of the 50 (88%) isolates that were serotyped, 38 (76%) were T-1 and 12 (24%) were T-25. The overall group A streptococcal T-1 prevalence was 18% (38/217, seven unserotyped isolates were excluded). Children who had group A streptococcal T-1 isolates attended six of the nine rooms; T-1 isolates accounted for over half the total group A streptococcal isolates in four rooms and all the group A streptococcal isolates in two other rooms. The index case's room had the highest prevalence of group A streptococcal T-1 carriage (6/16, 38%). Attendees who shared the index case's room were 2.4 times more likely to have a group A streptococcal T-1 isolate than children in other rooms (6/16 v32/201; confidence interval (CI)=1.2 to 4.8). The risk of having a group A streptococcal T-1 isolate was also related to the number of hours per week spent at the centre (χ^2 for trend=4.8, p=0.03, table 1). The three rooms in which the group A streptococcal T-1 prevalence was zero included both rooms with the youngest children (mean age <1 vear).

Among children whose parents responded to the questionnaire, those who were given

Table 1	Throat culture results for participating attendees,	
by mean	weekly duration in child care; figures are per cent	
(number	'total)	

Duration (hours/week)	Group A streptococcal*† isolates	Group A streptococcal T-1†‡ isolated
0-9	11.1 (5/45)	7.0 (3/43)
10-19	25.9 (7/27)	.15.4 (4/26)
20-29	25.6 (10/39)	18.4 (7/38)
30-39	39·1 (9/23)	34.8 (8/23)
40-49	36.6 (11/30)	30·0 (9/30)
50-59	0 (0/3)	0 (0/3)
60–69	0 (0/3)	0 (0/3)
Total	24.7 (42/170)	18.7 (31/166)

*Excludes 54 with unknown duration.

 $\uparrow \chi^2$ for linear trend: group A streptococcal isolates=3.7, p=0.05; group A streptococcal T-1 isolates=4.8, p=0.03. ‡Excludes 54 with unknown duration and four with unserotyped group A streptococcal isolate.

Table 2 Frequency (%) of signs and symptoms among the 111 attendees who did not take antibiotics for whom questionnaire responses are available, by group Astreptococcal T-1 status

	Culture status			
Sign/symptom	Group A streptococcal T-1 (n=26)	Negative* (n=85)	p Value	
Fever	23	17	0.56	
Sore throat	23	13	0.22	
Cough	19	27	0.42	
Earache	4	6	0.59	
Rhinorrhoea	23	27	0.69	
Fever, sore throat	19	8	0.15	
Fever, sore throat, cough Fever, sore throat, cough,	12	7	0.43	
rhinorrhoea	12	7	0.43	

*Includes seven who had group A streptococcal T-25 isolates.

antibiotics (for any illness) at any time during the four weeks preceding the culture survey were less likely to have group A streptococcal T-1 isolates than those who did not take antibiotics (3/36 v 26/111; relative risk (RR)=0.4; 95% CI=0.1 to 1.1). Among the 26 attendees who had group A streptococcal T-1 isolates and the 85 attendees who had negative cultures and did not use antibiotics, the proportions reporting fever or respiratory signs and symptoms were similar (table 2). No sign or symptom alone or in combination was statistically associated with group A streptococcal T-1 positivity. Seventeen (63%) of the 26 attendees who had T-1 isolates who did not use antibiotics reported no respiratory symptoms or fever during the study period. Four (15%) of these 26 had a parent reported rash some of which parents suspected were due to varicella.

Data for 138 attendees for whom centre records and questionnaire data were available were analysed by multivariate logistic regression. Because both the entry time of day and the total hours per week at the centre were highly correlated, only hours per week at the centre was included in the analysis. In addition, age, the total number of attendees in the classroom, and classroom assignment were also highly correlated. Of these factors, only sharing the index case's room was included in the analysis. Race and sex were excluded from the final analysis because neither factor had a significant effect and inclusion did not change the effect of other factors. Therefore, the hours per week at the centre, sharing the index case's room, and antibiotic use the month before the culture survey, were included in the final analysis by multivariate logistic regression. Increased time in child care was a significant risk factor (odds ratio (OR)=1.03; 95% CI=1.001 to 1.061) and antibiotic use was protective (OR=0.22; 95% CI=0.06 to 0.87). While the risk of group A streptococcal T-1 carriage was greater among children sharing the index case's room, this was not statistically significant (OR=2.7; 95% CI=0.8 to 9.4).

Over half (14/24, 58%) of the participating staff members were between 20 and 30 years old (range 19 to 54) and almost all were female (23/24, 96%). Two (8%) of 24 had group A streptococcal isolates; both isolates were serotype T-1. One staff person who had a T-1

isolate worked in a classroom where no T-1 isolates were identified in children, and the other worked in a room where 28% carried T-1 isolates.

After the 38 attendees who had group A streptococcal T-1 isolates were notified of their culture result and antibiotics were recommended, 34 (89%) received some treatment (varying agents and durations). One of the two staff members who had a group A streptococcal T-1 isolate took antibiotics after receiving the positive culture report. Eighteen (45%, 17 attendees and one adult) of the 40 who had T-1 isolates were recultured; 16 had taken penicillin and rifampicin, while two took only penicillin for 10 days. Among the 16 who took penicillin and rifampicin, two (13%) repeat cultures were positive for group A streptococcus T-1. Of the two who took only penicillin, one was repeat culture positive for group A streptococcus T-1.

No further invasive cases occurred among people associated with the child care centre and none were reported in other community members during the several months after this case.

Discussion

This investigation showed that carriage of the invasive organism was widespread, suggesting transmission may have occurred readily at the centre. The organism was recovered from children in two thirds of the nine rooms, with the highest prevalence in the index case's room. The absence of group A streptococcal carriage among children <1 year old may reflect their decreased mobility (nearly all were confined to cribs while at the centre) and less child-to-child contact and use of common play items in this age group compared with older children. The staff had a low group A streptococcal T-1 prevalence and apparently did not contribute to the spread of infection, although the role of staff in other institutional outbreaks remains unclear.^{3 11}

The risk of group A streptococcal carriage among children increased as time spent in the centre increased. A similar association has been noted between the occurrence of *Haemophilus influenzae* type b invasive disease and day care exposure.¹⁹ As might be expected, antibiotic use before the culture survey was protective against group A streptococcal T-1 carriage.

Fever and other respiratory signs and symptoms in those who did not receive antibiotics during the study period were uncommon for both those who had group A streptococcal T-1 isolates and negative cultures. Excluding persons who took antibiotics (some of whom possibly had group A streptococcal T-1 related illness) for this evaluation would tend to minimise our ability to detect an association between reported signs and symptoms and culture positivity. However, because the primary focus of this investigation was to detect potential predictors of group A streptococcal T-1 carriage among children in child care and antibiotic use was

shown to affect carriage, this group was excluded for this analysis.

An important question is whether those who harbour group A streptococcus after a case of invasive group A streptococcal disease in a day care centre can be identified by means other than an institution-wide culture survey. Of the risk factors identified, sharing the room with the index case identified only a fraction (16%)of the carriers. Antibiotic use during the study period occurred in approximately 25% of attendees and was associated with a protective effect from the carrier state, although nearly 10% (3/36) of antibiotic users had T-1 isolates. Reported clinical signs and symptoms were relatively uncommon in carriers and were not predictive of carriage status. The risk of carriage increased with increasing time spent at the centre, although some who attended fewer than 10 hours per week were carriers. In summary, none of these factors alone or in combination predicted all who were carriers. The effect of other infectious agents or environmental cofactors on carriage were not specifically addressed in this study.

Another important issue is the potential role of antibiotic treatment after a case of invasive disease to prevent secondary invasive disease. Because T serotype status was initially unknown, antibiotic treatment was recommended to all who had group A streptococcal isolates; most attendees and staff who had isolates received treatment. After this intervention, no further cases of invasive disease occurred, although it is unclear whether this was due to antibiotic administration. Although antibiotic treatment of those culture positive in a child care centre¹² and mass prophylaxis with penicillin in military recruits have curtailed epidemic non-invasive group A streptococcal disease,²⁰ any untreated individual can serve as a reservoir and transmit infection to those previously treated. Several logistic problems may hinder effective implementation of prophylactic regimens.²¹ Parents may not contact their child's doctor as advised, doctors may not be aware of or may disagree with the need for prophylaxis, and parents may have difficulty obtaining and or administering the medication. Further understanding of the actual risk of secondary invasive group A streptococcal disease after an index case is needed before the effectiveness of antibiotic intervention can be assessed.

Three of 18 who received antibiotics after their initial positive culture result had group A streptococcal T-1 isolated on repeat culture despite the effectiveness of penicillin alone or penicillin plus rifampicin for bacteriological cure of group A streptococcal disease.^{15 22} The bacteriological failures observed in children who received these antibiotic treatments, in part, may reflect poor compliance with antibiotic treatment or the length of time between antibiotic completion and reculturing allowing reinfection.¹⁵ Because repeat cultures were obtained for less than half of the treated carriers, culture results and bacteriological failures should be interpreted with caution.

Limitations of our study included the length of time required to conduct the culture survey, the grouping of children's exposure by single rooms when some actually had exposure to other rooms, and the lack of complete data both on the serotype of some children positive for group A streptococcal infection and the amount of time spent in the centre.

During the one week culture survey the group A streptococcal carriage rate for those who were cultured during the last half of the week was similar to the rate for those cultured during the first half of the week (25% v 26%, respectively). The room assignment of each child was used to represent the location of exposure at the centre during the study. Because children were consolidated into rooms during low attendance periods a misclassification of room exposure was probably random and likely resulted in creating a relatively more uniform exposure for all children at the centre regardless of assigned rooms.

Children positive for group A streptococcal infection with no serotype information were in three different rooms and their number of hours per week in child care (available for only four of the seven), on average, was less than that for children who had complete serotype information (12 v 30, respectively). The 54 children for whom the duration in child care was not available attended eight of the nine classrooms and the group A streptococcal carriage rate among them was similar to that among children for whom attendance information was complete (15/54; 28% v42/170; 25%).

In conclusion, we discovered that group A streptococcal T-1 (the invasive strain) carriage was common among attendees. The use of risk factors for carriage during this investigation did not identify all carriers. Therefore, attempts to eradicate an invasive organism in this setting would require at least a culture survey (with 100% sensitivity) of the entire centre and effective treatment of all identified carriers or mass treatment of all associated with the centre. This approach does not take into account the potential environmental and family group A streptococcal reservoirs. Further studies of invasive group A streptococcal disease in child care are needed to better define risk factors for carriage and secondary invasive disease and to assess the effectiveness of interventions aimed at reducing or eliminating the risk of secondary disease. With the increasing incidence of group A streptococcal invasive disease, doctors should continue to diagnose and treat group A streptococcal infections aggressively.

We would especially like to thank Moye Davis, RN, Pat Williams, RN, Sharon Thompson, RN, Najmul Chowdhury, MBBS, MPH, and others from the Alabama Department of Public Health, along with Nathan Ruben, MD, for their invaluable assistance in conducting this study. We also would like to thank Robert Facklam, PhD, CDC, for performing the T serotyping tests and Nancy Barker, MS, CDC, for statistical support and assistance with the multivariate analysis.

Givner LB, Abramson JS, Wasilauskas B. Apparent increase in the incidence of invasive group A beta-hemolytic streptococcal disease in children. J Pediatr 1991; 118: 341-6.

- Wheeler MC, Roe MH, Kaplan EL, Schlievert PM, Todd JK. Outbreak of group A streptococcus septicemia in children. Clinical, epidemiologic, and microbiologic correlates. *JAMA* 1991; 266: 533-7.
 Centers for Disease Control and Prevention. Nursing home

- A streptococcal bacteremia in Sweden: an epidemiological and clinical study. J Infect Dis 1991; 164: 595-8.
- 6 Stevens DL. Invasive group A streptococcus infections. Clin Infect Dis 1992; 14: 2-13.
- 7 Benjamin EM, Gershman M, Goldberg BW. Communityacquired invasive group a beta-hemolytic streptococcal infections in Zuni indians. Arch Intern Med 1992; 152: 1881-4.
- 8 Katz AR, Morens DM. Severe streptococcal infections in bitorical perspective. *Clin Infect Dis* 1992; 12: 298-307.
 Bisno AL. Group A streptococcal infections and acute rheumatic fever. *N Engl J Med* 1991; 325: 783-93.
 Schwartz B, Facklam R, Breiman RF. Changing epidemi-
- ology of group A streptococcal infections in the USA. Lancet 1190; **336**: 1167-71.
- Smith TD, Wilkinson V, Kaplan EL. Group A strepto-coccus-associated respiratory tract infections in a day-care center. *Pediatrics* 1989 83: 380-4.
- 12 Falck G, Kjellander J. Outbreak of group A streptococcal infection in a day-care center. Pediatr Infect Dis J 1992; 11: 914-9
- 13 American Academy of Pediatrics. In: Peter G, ed. Report of the committee on infectious disease. 22nd Ed. Chicago, Illinois: AAP, 1991.
- 14 National Commission on Children. Beyond rhetoric: a new
- A National Commission on Children and families. Bayona interioric a network American agenda for children and families. Washington DC: National Commission on Children, 1991.
 Chaudhary S, Bilinsky SA, Hennessy JL, et al. Penicillin V and rifampin for treatment for group A streptococcal pharyngitis: a randomized trial of 10 days penicillin vs 10 days penicillin with rifampin during the final 4 days of therapy 7 Perior 1985: 106: 481-66 therapy. J Pediatr 1985; 106: 481-6 Vilson E, Zimmerman RA. Mo
- therapy. J Pediatr 1985; 106: 481-6.
 16 Wilson E, Zimmerman RA, Moody MD. Value of T-agglutination typing of group A streptococci in epidemiologic investigations. Health and Laboratory Science 1968; 5: 199-207.
 17 Moody MD, Padula J, Lizana D, Hall CT. Epidemiologic characterization of group A streptococci by T-agglutination and M-precipitation tests in the public health laboratory. Health and Laboratory Science 1965; 2: 149-62. 149-62
- 18 Seppala H, Vuopio-Varkila J, Osterblad M, Jahkola Rummukainen M, Holm SE, Huovinen P, Evaluation of methods for epidemiologic typing of group A streptococci. J Infect Dis 1994; 169: 519-25.
- 19 Fleming DW, Leibenhaut MH, Albanes D, et al. Secondary
- Prenning Dw, Debenhaut Writ, Roanes D, et al. Secondary haemophilus influenzae type b in day-care facilities. Risk factors and prevention. JAMA 1985; 254: 509-14.
 Gray GC, Escamilla J, Hyams KC, et al. Hyperendemic streptococcus pylogenes infections despite prophylaxis with penicillin G benzathine. N Engl J Med 1991; 325: 92-7 a2_'
- 21 Li KI, Dashefsiy B, Wald ER. Haemophilus influenzae type b colonization in household contacts for infected and colonized children enrolled in day care. Paediatrics 1986; 78:
- 22 Reed BD, Huck W, Zazove P. Treatment of beta-hemolytic streptococcal pharyngitis with cefaclor or penicillin. Efficacy and interaction with beta-lactamase-producing organisms in the pharynx. J Fam Pract 1991; 32: 138-44.

Commentary

Streptococcus the Α pyogenes, group streptococcus, β-haemolytic remains а troublesome micro-organism. Good hygiene, antibiotics, and other attributes of a high standard of living have lessened the frequency of severe infection, but the bacterium has kept its virulence factors. Given a chance to spread among close contacts and to invade through broken skin, outbreaks with severe or fatal cases are possible. The paper by Engelgau and colleagues reminds us of this and shows how investigations should be done. Three aspects merit a comment: the organism, the route of infection, and the outbreak management.

The M type protein of S pyogenes is an important determinant of its pathogenesis and epidemiology. This is clearest in glomerulonephritis, which is the sequel of infections by a few M types (for example, M types 12, 49, 55, and 60). T type 1 is equivalent to M type 1 in this incident, but T typing is less specific for most other types. M type 1 is the type most often reported in overwhelming infection, but it is a common strain and typing is most likely to be done for the bacteraemic and other severe infections. The M type is associated with specific protective immunity, which should not be confused with the antibodies against exotoxins used in diagnostic tests, of which streptolysin O is the best known. This type specific immunity partly explains why streptococcal diseases fluctuate under the influence of herd immunity to common strains.

Chickenpox is one of several skin conditions that may become secondarily infected by S pyogenes. Burns, insect bites, and minor injuries are commoner portals of streptococcal skin infection, but seem to have less risk bacteraemia with consequent shock, of osteomyelitis, or arthritis. Children are the population in whom S pyogenes circulate most as they have least type specific immunity and closest contacts with other infected individuals.

Streptococcal infection is endemic, and virulent bacteria in susceptible children will cause the occasional death. Should we investigate the contacts of people who have severe streptococcal disease, when little is done in the numerous mild cases? Is this not shutting the stable door after the horse has bolted? The death of a child creates a feeling that 'something must be done'. The paper by Engelgau et al shows that it is difficult to test everyone in a large group, and the time taken for laboratory tests diminishes the benefit of prophylactic treatment. The finding of lower carriage rates in children who had had antibiotics recently, suggests that good access to primary care is one reason why streptococcal infections have become less threatening in modern times.

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