A Family Cluster of Five Cases of Group A Streptococcal Pneumonia

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ABSTRACT. A cluster of 5 family members, a mother and 4 children, were hospitalized for severe group A *Streptococcus* (GAS) pneumonia. Three family members had complications: sepsis (1), empyema (2), and a sterile parapneumonic effusion (1). Two additional family members had symptoms of upper respiratory tract infection, and 1 was hospitalized for these symptoms. GAS was isolated from the blood of 1 patient, the pleural fluid of 2 patients, and the oropharynx of 6 patients. Pulsed field gel electrophoresis testing revealed an identical deoxyribonucleic acid pattern in all 7 isolates. Genotyping revealed the *spe*A gene and serotyping the T-1, M-1 serotype in all isolates. This family cluster of invasive GAS disease is the largest reported to date, with an attack rate of 41.7% (5 of 12 family members). This report provides further support for antibiotic prophylaxis of close contacts of individuals with invasive GAS disease. *Pediatrics* 2003;112:e61–e65. URL: http://www.pediatrics.org/cgi/content/full/112/1/e61; *group A Streptococcus, family, cluster, invasive, pneumonia.*

ABBREVIATIONS. GAS, group A *Streptococcus*, streptococcal; STSS, streptococcal toxic shock syndrome; RBCH, Rainbow Babies and Children's Hospital; PFGE, pulsed-field gel electrophoresis; HLA, human histocompatibility leukocyte antigen.

Life-threatening invasive group A streptococcal (GAS) infections have been reported with increasing frequency during the last 2 decades.[1–3] Family clusters of invasive GAS infection are relatively uncommon, and most individuals in these families present with necrotizing fasciitis or streptococcal toxic shock syndrome (STSS).[4–6] In this report, we describe an outbreak of GAS pneumonia involving a mother and four children from a 12-member family. The 5 documented cases of GAS pneumonia comprise the largest family cluster of invasive GAS infection reported to date and provide additional evidence to support surveillance cultures and antibiotic prophylaxis of family members and other close contacts of an index case in this situation.

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PATIENTS

Family Members With Pneumonia

During a 1-week period in spring, a mother and 4 of her 10 children developed fever and a cough, along with other symptoms that varied according to the patient. All 5 individuals were admitted or transferred to University Hospitals of Cleveland/Rainbow Babies and Children's Hospital (RBCH) and seen by the authors (S.R., B.R., J.R.S., R.A.S., and C.C.J.) on the same day. Pneumonia was documented in all 5 family members, and *Streptococcus pyogenes* was isolated from cultures of blood,[1] pleural fluid,[2] or the oropharynx.[2]

Case 1

A previously healthy 46-year-old female presented to a local hospital with a 5-day history of cough, fever, chills, and dyspnea. Her temperature was 38.2°C and physical examination revealed diminished breath sounds in the left lung field and crackles in the right mid-lung field. Chest roentgenography demonstrated bilateral lower lung infiltrates and a left pleural effusion. A thoracentesis yielded purulent pleural fluid that grew *S pyogenes*. She was transferred to University Hospitals of Cleveland because of persistent fever despite 4 days of treatment with levofloxacin, ceftriaxone, and azithromycin. At University Hospitals of Cleveland, a computed tomogram of the chest showed loculated fluid in the left hemithorax. Intravenous penicillin G and clindamycin were started and she was taken to the operating room, where thick pleural exudates were drained and a large pleural peel excised. She was discharged after 9 days of treatment in the hospital. She was well on follow-up after completing a 3-week course of intravenous antibiotic therapy.

Case 2

The 9-year-old daughter of the patient in case 1 presented to a local hospital with cough, a hoarse voice and fever, which had worsened over the preceding 8 days. She was treated with racemic epinephrine, dexamethasone, and ceftriaxone and transferred the next day to the RBCH pediatric intensive care unit. On admission to RBCH, she had a temperature of 38.8°C, pulse rate of 150 beats per minute, respiratory rate of 50 breaths per minute, and blood pressure 91/48 mm Hg. Physical examination revealed pharyngitis, inspiratory stridor, crackles, and wheezes at both lung bases, diffuse abdominal tenderness, and a scarlatiniform rash of the legs, back,

trunk, and groin area. Blood and pharyngeal cultures yielded *S pyogenes*. Chest roentgenography disclosed a left lower lobe infiltrate without effusion. Intravenous penicillin G and clindamycin were initiated. Blood pressure stabilized with intravenous fluid administration. Clindamycin was discontinued on day 6 because of diarrhea. On day 14, she had a recurrence of fever to 40°C. Repeat chest roentgenography demonstrated a left pleural effusion. Thoracentesis relieved her symptoms, but the pleural fluid obtained yielded no growth on bacterial culture. Penicillin was changed to ceftriaxone. She was completely recovered after 14 days of ceftriaxone treatment.

Case 3

The 12-year-old son of the patient in case 1 was admitted to the RBCH pediatric intensive care unit with a 1-week history of worsening cough, fever, and dyspnea. His temperature was 38.5°C, and physical examination revealed pharyngitis, decreased breath sounds throughout his right lung, crackles at both lung bases, and a scarlatiniform rash on his legs, trunk, and groin. Chest roentgenography revealed a left basilar infiltrate and right basilar consolidation with a loculated effusion. Pleural fluid and pharyngeal cultures yielded *S pyogenes*. A respiratory antigen panel (Chemicon International, Temecula, CA) was positive for parainfluenza virus. Intravenous penicillin G and clindamycin were initiated, and a chest tube was inserted for empyema drainage. He was treated with penicillin and clindamycin for 6 days, then penicillin alone for 4 days, followed by ceftriaxone for 14 days. After discharge he experienced tissue herniation at his chest tube site and has recovered after repair of this complication.

Case 4

The 10-year-old son of the patient in case 1 presented to RBCH with a 3-day history of diarrhea, vomiting, cough, and fever. His temperature was 38.5°C, and his physical examination revealed pharyngitis, crusted lesions of the external nares, crackles at both lung bases, and a scarlatiniform rash of the arms, legs, trunk, and groin area. Chest roentgenography demonstrated right lower lobe and left upper lobe infiltrates without pleural effusion. Pharyngeal culture grew *S pyogenes*. Intravenous penicillin G and clindamycin treatment were initiated. He was discharged 2 days after admission to receive a 10-day oral course of clindamycin and recovered uneventfully.

Case 5

The 7-year-old son of the patient in case 1 presented to RBCH with a 4-day history of fever, cough, abdominal pain, sore throat, and mild shortness of breath. His temperature was 38.8°C and his physical examination revealed pharyngitis, wheezes, and crackles at both lung bases; diffuse abdominal tenderness; and a scarlatiniform rash of the back, trunk, and groin area. Chest roentgenography demonstrated a right upper lobe infiltrate without effusion. Pharyngeal culture grew *S pyogenes*. Intravenous penicillin G and clindamycin were initiated. He was discharged 2 days after admission with a 10-day oral course of clindamycin and recovered uneventfully.

Other Symptomatic Family Members

All 12 family members lived in the same house. A sixth family member (14 years old) with culture-proven GAS pharyngitis, severe cough, a scarlatiniform rash, and fever also required hospitalization but did not have evidence of pneumonia on physical examination or chest roentgenography. She received intravenous penicillin and clindamycin for 1 day and was discharged with 10 days of oral clindamycin. A seventh family member (3 years old) with culture-proven GAS pharyngitis was treated with erythromycin and did not require hospitalization.

Contacts of Symptomatic Family Members

Nine other individuals in close contact with the index case (the remaining 5 family members, an aunt and uncle, and 2 family friends) were empirically given amoxicillin prophylaxis for 10 days; 1 contact was penicillin-allergic and was given erythromycin. Pharyngeal cultures were not obtained from these individuals, and none became clinically ill.

METHODS

Bacterial cultures of blood (in 6 hospitalized patients), oropharynx (in 6 of 7 symptomatic family members), and pleural fluid (in 3 patients with pleural effusions) were obtained and tested at University Hospitals of Cleveland.

Laboratory Evaluation Methods for GAS Isolates

Isolates were confirmed as *S pyogenes* with the use of standard techniques. Minimal inhibitory concentration and minimal bactericidal concentration to penicillin were investigated by broth microdilution, according to the NCCLS standards.[7] The genetic relatedness of the *S pyogenes* strains was investigated by pulsed-field gel electrophoresis (PFGE) using *Sma* I digested fragments. The deoxyribonucleic acid plugs were prepared, lysed, and placed in a 1% agarose gel according to the instructions of the manufacturer (Gene Path; Bio-Rad, Hercules, CA). PFGE patterns were analyzed visually and interpreted according to standard guidelines.[8]

All isolates were serologically examined and were characterized by T-agglutination pattern and m-serotyping and/or opacity factor inhibition at the World Health Organization Collaborating Center for Reference and Research on *Streptococci* at the University of Minnesota.[9] The presence of *speA* and *speC* genes was assessed by the polymerase chain reaction as previously described.[10]

RESULTS

The clinical presentation and bacterial culture results of the 7 family members with symptomatic GAS infection are summarized in Table 1.

In all GAS isolates obtained from the 7 individuals, the minimal inhibitory concentration and minimal bactericidal concentration for penicillin were ≤ 0.015 μg/ml; serotype was characterized as T-1, M-1, and the *spe*A gene was present and the *spe*C gene absent. The PFGE patterns of the 7 isolates were identical, indicating genetic relatedness of the isolates (Fig 1).

DISCUSSION

This outbreak of pneumonia caused by the same GAS isolate in 5 members of a single family is the largest family cluster of invasive GAS infection reported to date. GAS was isolated from a sterile site in

TABLE 1. Clinical Features and Bacterial Culture Results of 7 Family Members with Symptomatic Group A *Streptococcus* Infection

Patient	Age (Years)	Sex	Clinical Manifestations	Time of Symptom Onset (Days)	Culture Growing *S pyogenes*
1	46	F	Pneumonia, empyema	T + 1	Pleural fluid
2	9	F	Pneumonia, sepsis, pleural effusion, pharyngitis	T = 0	Blood, pharynx
3	12	M	Pneumonia, empyema, pharyngitis	T + 3	Pleural fluid, pharynx
4	10	M	Pneumonia, pharyngitis	T + 7	Pharynx
5	7	M	Pneumonia, pharyngitis	T + 6	Pharynx
6	14	F	Pharyngitis, cough, fever	T + 6	Pharynx
7	3	M	Pharyngitis	T + 6	Pharynx

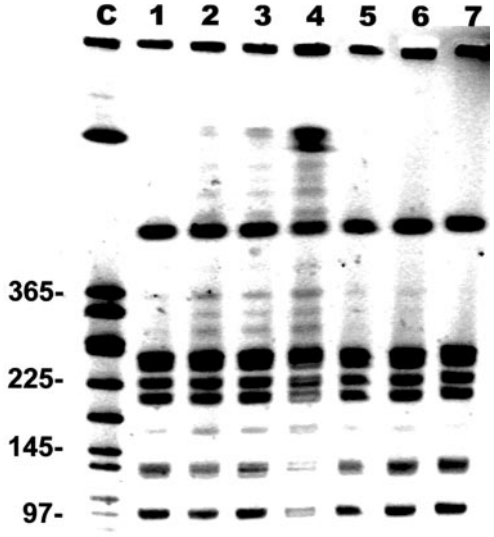

Fig 1. PFGE patterns of chromosomal deoxyribonucleic acid restriction fragments of the GAS isolates digested with *Sma*I. A similar pattern was obtained in all 7 GAS isolates (lanes 1–7, representing patients 1–7 in Table 1). *S aureus* control was used as a size standard control (lane C). The numbers on the side represent molecular weight standards in kilobases.

3 of the 5 family members with clinical and roentgenographic evidence of pneumonia and from pharyngeal cultures in the remaining 2 family members. The clinical evidence (pneumonia, scarlatiniform rash, GAS on pharyngeal culture, family members with concurrent GAS pneumonia) strongly supports a diagnosis of GAS pneumonia in these latter 2 members. The attack rate in this family was 41.7% (5 of 12 members) for invasive GAS infection and 58.3% (7 of 12 members) for symptomatic GAS infection.

Family clusters of invasive GAS infection are unusual and when described have typically involved only 2 individuals, most often adults.[4,6] However, clusters involving children have been reported, including a recent cluster of STSS involving 3 children in Taiwan.[5] GAS pneumonia has rarely been reported in family clusters.[11]

Potential reasons for family clustering of invasive GAS infection include prolonged close contact between the index case and susceptible family members, GAS strain virulence, and host susceptibility to GAS or certain strains of GAS. Prolonged close contact of individuals is a well-established risk factor for the spread of GAS infection including invasive infection,[12] and a greater length of contact time (>24 hours) and younger age have been reported as independent risk factors for infection.[13] The large number of children infected in the present family cluster likely reflects both the predominance of children as close contacts of the index case and the increased risk of GAS infection in exposed children as compared with adults.[13,14]

Infection with more virulent GAS strains has been proposed as a further reason for family clustering of invasive GAS infections. GAS isolates of the M1T1 serotype have been identified in many outbreaks of invasive GAS infection,[15] but numerous other M- and T-serotypes have also been associated with invasive GAS infection.[2] A recent study documented that prevalence of specific virulence-associated M-serotype GAS clones was similar in individuals with and without invasive infection, suggesting that M-serotype is likely not the major risk factor for invasive infection.[16] Similarly, frequency of the *spe*A gene and its encoding toxin SpeA have been found to be higher in individuals with invasive infection in some studies[15,17] but not others.[10,18]

The lack of uniform virulence factors and the finding that particular GAS clones cause invasive infection in some individuals but not others suggest that host genetic susceptibility plays a role in the development of invasive GAS infection.[16] In vitro studies have documented interactions between class II MHC alleles and SpeA production[19] and human histocompatibility leukocyte antigen (HLA) type and response to other streptococcal antigens.[20] Other host-dependent factors associated with invasive GAS infection, such as lack of anti-SpeA antibodies[21] and inability to neutralize SpeA lymphocyte mitogenicity,[15] may also have a genetic component. Strong evidence for genetic susceptibility was provided by a recent study which documented that specific HLA class II haplotypes conferred protection from severe systemic GAS disease in humans while others increased the risk of disease.[22] This study also documented that individuals with protective haplotypes had significantly lower cytokine and proliferative responses to streptococcal superantigens than individuals neutral or risk-associated haplotypes. The study findings suggest that HLA class II haplotypes affect disease presentation by mediating the host immune response to streptococcal superantigens.[22] Epidemiologic evidence for increased genetic susceptibility to invasive GAS infection includes a study

from Pima County, Arizona, where annual incidence of invasive GAS infections in native Americans was found to be 10 times that of nonnative American individuals.[23] However, both genetic and lifestyle factors may have contributed to this increased incidence.

Antecedent viral infection in the family members may also have contributed to susceptibility to disease. All patients had upper respiratory infection symptoms 3 to 7 days before developing invasive GAS disease. Although these symptoms may have been attributable to upper respiratory tract GAS infection, the presence of croup symptoms in 1 child and the detection of parainfluenza virus detected on the respiratory antigen panel of another suggest that viral infection was present in at least some family members. Antecedent viral infection has not been documented in most outbreaks of invasive GAS infection, with the exception of varicella in children preceding STSS. However, in epidemics of GAS pharyngitis in military recruits, prior viral upper respiratory infection was documented in one third of the cases, suggesting that this may be a risk factor for development of GAS disease.[24]

The issue of antibiotic prophylaxis for contacts of individuals with invasive GAS infection continues to be debated.[25] Close contacts of patients with invasive GAS infection are at increased risk of invasive GAS infection: in a large prospective study, the risk for close contacts was estimated to be 2.9 per 1000 individuals, a risk almost 200 times that of the general population.[26] However, prophylaxis of all close contacts necessitates treatment of many individuals who would not develop invasive infection. In addition, the efficacy and optimal regimen of antibiotic prophylaxis against invasive GAS infection have not yet been established. For these and other reasons, the Prevention of Invasive group A Streptococcal Infections Workshop convened by the Centers for Disease Control and Prevention recently concluded that "for household contacts of index patients, routine screening for and chemoprophylaxis against GAS are not recommended" but that health care providers "may choose to offer chemoprophylaxis to household contacts who are at an increased risk of sporadic disease or mortality due to GAS."[27] In the present report, none of the affected individuals or contacts had any of the risk factors cited by the workshop. We believe that the high attack rate of invasive GAS infection in the family in the present report adds to the growing body of evidence that close contacts of individuals with invasive GAS infection, particularly those in contact for >24 hours, should receive antibiotic prophylaxis. Clinicians might elect to obtain throat cultures on contacts and treat only those with cultures positive for GAS or to treat all close contacts; in the absence of prospective data, either approach seems reasonable. Clindamycin and the combination of benzathine pencillin G and rifampin have been most effective at eradication of upper respiratory tract GAS and would probably be the regimens of choice for chemoprophylaxis of contacts.[27] Azithromycin has also been used successfully to eradicate upper respiratory GAS and is a reasonable alternative.[27] However, none of these regimens have been evaluated as prophylaxis against invasive GAS disease, so their efficacy for this purpose is unknown.

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ERRATUM

An error occurred with regard to the order of the authors' names and the authors' respective affiliations in the article by Roy et al titled "A Family Cluster of Five Cases of Group A Streptococcal Pneumonia," which appeared in the July 2003 issue *Pediatrics electronics pages* (*Pediatrics*. 2003;112:e61–e65).

The order of the authors' names (which appear right after the title of the article on page e61) should now read as follows:

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This has been corrected in the electronic version of the article that appears on the AAP's Web site.

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